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Original Article

An outbreak of *Salmonella* Muenchen after consuming sea turtle, Northern Territory, Australia, 2017.

Anthony D. K. Draper, Christian L. James, Joy E. Pascall, Kathryn J. Shield, Jennifer Langrell, Adrian Hogg

Abstract

An outbreak of *Salmonella* Muenchen gastroenteritis occurred in a remote coastal Aboriginal community in the Northern Territory (NT) of Australia. There were 22 people sick (attack rate 55%); 7 had laboratory confirmed *S.* Muenchen infection; 2 required medical evacuation and admission to the intensive care unit (ICU). We conducted a descriptive case series to investigate the outbreak. All cases ate meat from a single green turtle (*Chelonia mydas*). The animal's pre-death stress, improper butchering, insufficient cooking and the unsatisfactory storage of meat all likely contributed to the outbreak. Turtle meat requires safe preparation which includes thorough cooking and appropriate storage to avoid *Salmonella* infection.

Keywords: outbreak, *Salmonella*, *Salmonella* Muenchen, gastroenteritis, foodborne disease, turtle, *Chelonia mydas*.

Background and methods

On Monday 19 June 2017, the Northern Territory (NT) Centre for Disease Control (CDC) was alerted to a group of people who presented to a remote health centre in the previous 24 hours with gastrointestinal symptoms. All reported eating turtle meat. We conducted a descriptive case series to investigate the outbreak and to determine factors that led to the outbreak occurring.

We used information recorded by the treating clinicians, regional Aboriginal land and sea rangers, and from two of the persons who caught, prepared and ate the turtle to formulate the case series. Data were analysed using Microsoft Excel 2010. Ethics approval was not sought for this investigation as it was conducted under the auspices of public health legislation.[1]

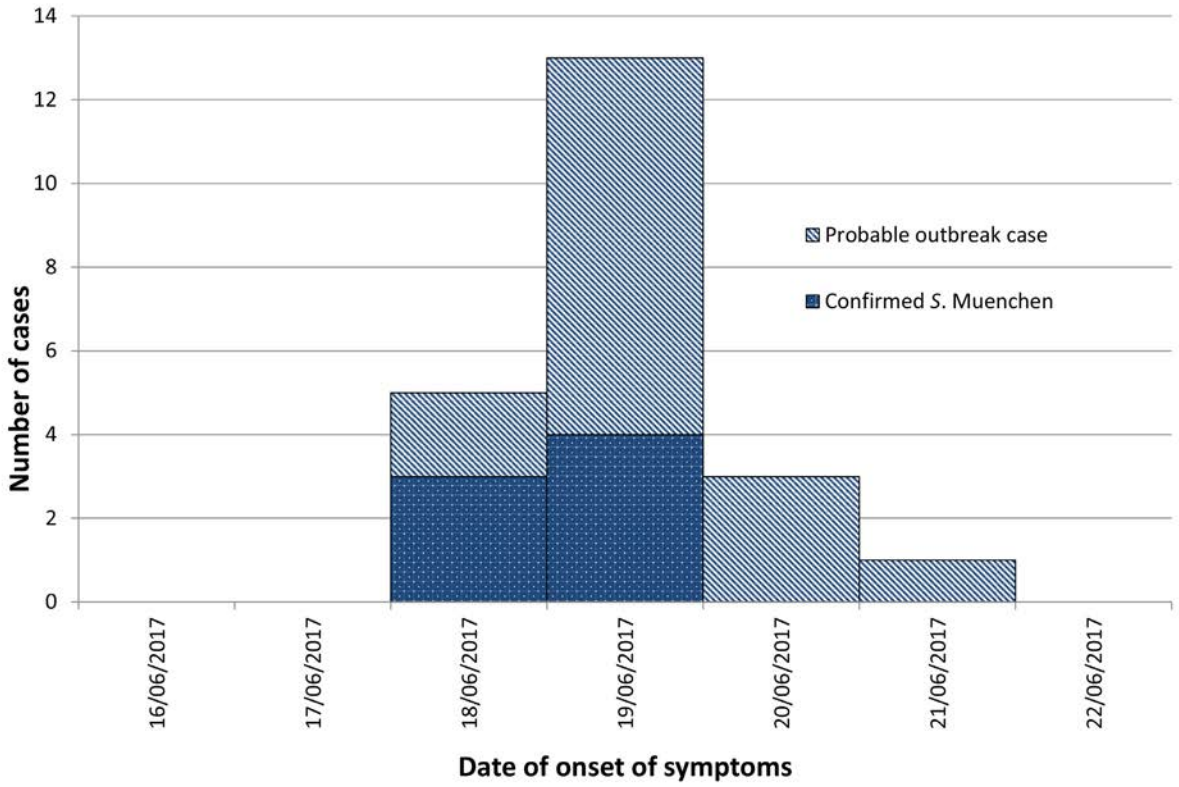
Description of outbreak

An outbreak case was defined as anyone from a specified coastal NT community with laboratory confirmed *Salmonella* Muenchen or a clinically compatible illness (diarrhoea and fever) with onset after 17 June 2017. There were 22 cases in total and 7 had laboratory confirmed *S.* Muenchen infection (Figure 1). All 22 cases reported symptoms of diarrhoea, abdominal pain, fever and vomiting. All cases sought medical attention at the local clinic and 2 were evacuated to Darwin where they were admitted to the intensive care unit (ICU) with sepsis.

The median incubation period was 15.5 hours (range 12-68 hours) after consuming meat from a single green turtle (*Chelonia mydas*). The median age of cases was 29 years (range 3-63 years), 12/22 (55%) were female and everyone fully recovered.

A large green turtle (Figure 2) was caught on a beach while laying eggs, at approximately

Figure 1: Epidemiological curve of outbreak cases by onset day after eating turtle meat (n=22).



4:00pm on 16 June 2017. The turtle was transported live on the back of a vehicle for approximately 4 hours and then rested upside-down on its shell. It was killed at approximately 4:00pm on 17 June 2017 and at approximately 8:00pm the turtle was cooked upside down on open fire for 1 hour, with the carapace acting as a natural cooking vessel. It was reported that the fat and liver in the animal were black rather than the typical green colour prior to cooking. Hot stones were placed inside the carapace to aid with cooking. The meat and organs inside the animal were eaten when half cooked and resembled a stew of meat, fat and blood. Sixteen people were recorded as being sick from eating this meal. Meat that was not eaten was given to other family members who 're-cooked' the meat the following day by boiling it at their home. There were 6 people sick as a result of this second meal from the turtle. At no point was refrigeration or ice used to preserve the meat. It is estimated that 40 people in total ate the turtle meat over 2 days (attack rate 55%).

In response to the outbreak, the health clinic at the community issued an alert to community members to take extreme caution when preparing and consuming turtle meat.

Discussion

Turtles are well known reservoirs of *Salmonella* spp. and are renowned sources of human infection.[3,4,5] Green sea turtles have also been shown to carry *Salmonella* spp.[6] Consumption of turtle meat is popular in Top End Aboriginal communities[7] and the rights of Aboriginal people to hunt turtle are protected by legislation.[8] In 1998 an outbreak of *S.* Chester gastroenteritis was reported after consumption of a green turtle in a remote coastal Aboriginal community of the NT.[9]

Our outbreak investigation identified a number of factors that likely contributed to the outbreak occurring. Firstly, the turtle was caught on the beach while laying eggs. Studies have shown that turtles can progress from being healthy latent carriers of *Salmonella* to hyper-excretors or even bacteraemic during periods of stress.[10] The initial stress of oviposition followed by capture, trans-

Figure 2: Adult green turtle (*Chelonia mydas*).[2]

port and death 24 hours later likely increased excretion of *Salmonella* spp. The discolouration of internal organs may be evidence of illness in the animal.

Secondly, the preparation and cooking method of the turtle were likely factors contributing to this outbreak. Preparation generally involves removing the head and intestines, placing hot stones or coals into the cavity and then cooking on a fire until the tissue inside the carapace resembles a stew.[11,12] Incomplete 'gutting' combined with insufficient cooking time would permit *Salmonella* spp. to survive within the turtle's carapace. Thirdly, lack of refrigeration would assist *Salmonella* spp. to continue to contaminate the meat consumed by those who ate a day after the initial meal.

There are several limitations to our outbreak investigation. Firstly, it is often difficult to investigate outbreaks of disease in remote communities due to public health staff being unable to attend the outbreak site because of the associated cost and distance. There was also a lack of telephone reception which restricted the ability to contact people as well as the inherent difficulty in trying to contact what is typically a highly mobile population. Our estimate that 40 people ate the turtle as well as the details surrounding its capture, preparation and consumption, was provided by the only two cases we could contact by telephone. As a result there is uncertainty that the cohort who consumed the turtle meat was indeed 40 persons. Our outbreak case definition was limited to only those who sought medical attention which means that less serious cases and non-symptomatic cases were not included in the outbreak case definition.

Raw turtle meat should be regarded by the public in the same light as raw chicken meat which can be assumed to be contaminated with *Salmonella* until cooked.[13] It should be handled safely and cooked thoroughly to ensure that *Salmonella* spp. and other bacteria are killed prior to consump-

tion. The potential for outbreaks to occur is high and it is likely that they are occurring and being under-reported. This outbreak was detected because the seriousness of disease resulted in multiple cases seeking medical attention.

Targeted health promotion messages should be directed towards Australian Aboriginal communities to highlight the increased risks of food poisoning from eating undercooked turtle meat and provide the measures that can be taken to mitigate these risks.

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Original Article

Probable epidemic Mycoplasma pneumoniae disease activity in metropolitan Sydney, 2015: combining surveillance data to cross-validate signal detection

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Abstract

Introduction

Mycoplasma pneumoniae is a leading cause of community acquired pneumonia and well-recognised cause of encephalitis in children. Sentinel hospital surveillance identified a temporal cluster of children with suspected encephalitis associated with *M.pneumoniae* in NSW. We aimed to determine whether this cluster was associated with epidemic *M.pneumoniae* activity.

Methods

A multi-faceted investigation was undertaken using existing data sources including:

- Active clinical surveillance of suspected encephalitis (ACE study) associated with *M.pneumoniae* at the Children's Hospital at Westmead (CHW).
- Syndromic surveillance of Emergency Department presentations for pneumonia in children 0-16 years to 86 NSW hospitals.
- Laboratory sentinel surveillance of *M.pneumoniae* testing performed at CHW and the Institute for Clinical Pathology and Medical Research (ICPMR), Westmead.

Results

We detected an increased number of cases of hospitalised suspected encephalitis associated with *M. pneumoniae* in 2015 at the Sydney Childrens Hospital Network (SCHN), with a peak in July, that were predominantly in Western Sydney. Concurrently, monthly pneumonia presentations to 86 NSW emergency departments in children aged 0-16 years increased in 2015. This increase was greater in children aged 5-16 years compared to those aged 0-4 years and in metropolitan sites compared with rural hospitals. Laboratory data from sentinel laboratories showed increased frequency of testing, and of tests returned positive for *M.pneumoniae* in 2015 compared to preceding years. In aggregate, this information was considered suggestive of epidemic activity of *M.pneumoniae* in metropolitan Sydney in 2015.

Conclusions

Active surveillance for childhood encephalitis has the potential to provide sentinel surveillance data to identify epidemic infectious disease activity. Combining multiple sources of surveillance data affords opportunities to cross-validate epidemic signals. *M.pneumoniae* disease activity is challenging to measure, and may be a cause of significant disease burden in Australian children during epidemic years.

Keywords: *Mycoplasma pneumoniae*, surveillance, encephalitis, pneumonia, children, Australia

Introduction

Mycoplasma pneumoniae infection has well-recognised neurological complications that occur most commonly in children, the most severe form being encephalitis.[1, 2] *M.pneumoniae* most often causes respiratory tract infection and disease including atypical pneumonia, acute otitis media and coryzal illness.[3, 4] The importance of *M.pneumoniae* as a cause of childhood pneumonia was recently emphasised in a large study of hospitalised childhood community-acquired pneumonia (CAP) from the United States. In this study, *M.pneumoniae*, confirmed by polymerase chain reaction assay (PCR), was the most frequent bacterial cause of childhood pneumonia (20-35%) amongst children aged 5-17 years.[5] The epidemiology of *M.pneumoniae* disease is not well characterised, but it is considered to be a worldwide endemic infectious disease with superimposed epidemics every four to seven years.[3, 4] Major epidemics were reported from several European countries, and Israel between 2010 and 2013. [6-10]

M.pneumoniae has been reported to be one of the most frequently identified pathogens associated with encephalitis in children.[11, 12] However, the strength of its causal assocation remains controversial, because it is infrequently identified in cerebro-spinal fluid.[1, 13] A further challenge is the lack of available laboratory tests with high sensitivity and specificity.[14]

Here we report results from a collaborative investigation undertaken following identification of an increased frequency of suspected encephalitis associated with *M.pneumoniae* by childhood encephalitis surveillance (the Australian Childhood Encephalitis -ACE- study). This increase in cases suggested epidemic *M.pneumoniae* disease activity to ACE investigators and prompted reporting to Western Sydney Local Health District (WSLHD) Public Health Unit (PHU) followed by a subsequent joint investigation with the PHU and NSW Ministry of Health (NSW MoH) Rapid Surveillance team.

Methods

We sought to determine whether the temporal cluster of *M.pneumoniae* encephalitis was associated with evidence of epidemic *M.pneumoniae* activity in NSW by aggregating existing data sources in order to determine if further public health action was required.

1. Data sources

The Australian Childhood Encephalitis (ACE) study:

The ACE study utilises the Paediatric Active Enhanced Disease Surveillance (PAEDS) network to undertake active, sentinel site surveillance for childhood encephalitis at five tertiary paediatric hospitals across Australia. Surveillance commenced in 2013 at the Children's Hospital at Westmead (CHW), the NSW sentinel site and PAEDS coordinating centre. From 2014, surveillance extended to involve the national PAEDS network.[15] The ACE study methodology has been previously published.[16] Suspected encephalitis is defined as: a child aged 0 to 14 years AND hospitalised with acute encephalopathy AND has *one or more* of the following: fever, seizures, focal neurological findings, at least one abnormality of cerebrospinal fluid (CSF): age determined pleocytosis, or elevated protein ≥ 40mg/dl, or EEG/ neuroimaging findings consistent with encephalitis. An association with *M.pneumoniae* is defined as the presence of specific IgM antibodies in acute sera. All suspected encephalitis cases were reviewed by an expert panel of clinicians and designated as confirmed encephalitis or not encephalitis using published case definitions with higher specificity than the surveillance definition.[16-18]

Public Health Rapid Emergency Disease and Syndromic Surveillance (PHREDSS) system:

In 2003, NSW MoH implemented a syndromic surveillance system including administrative Emergency Department (ED) data.[19] The system, now called the Public Health Rapid Emergency Disease and Syndromic Surveillance (PHREDSS) system, combines near real-time

data directly from participating ED patient information management systems with cleaned, more complete Emergency Department Records for Epidemiology (EDRE) data sourced from the NSW Emergency Department Data Collection. The PHREDSS system automatically groups primary provisional ED diagnosis codes, assigned by treating clinicians, into acute illness and injury syndromes for monitoring. The diagnosis codes used include any of the Australian clinical implementations of the International Classification of Diseases (ICD) 9th revision, ICD-10th revision (ICD-10AM) or the Systematized Nomenclature of Medicine - Clinical Terminology (SNOMED-CT).[20, 21] Syndromes are monitored daily to detect unusual patterns that may signify an emerging outbreak or issue in the population. The PHREDSS 'pneumonia' syndrome includes provisional diagnoses of viral, bacterial, atypical or unspecified pneumonia, SARS and legionnaires disease, but excludes pneumonia with influenza, which is included in the 'influenza-like illness' syndrome.

Sentinel laboratory surveillance of *M. pneumoniae*:

Laboratatory testing for *M.pneumoniae* is usually undertaken with serology. At the Children's Hospital at Westmead (CHW), testing is performed for *M.pneumoniae* IgM using a commercial enzyme linked immunosorbent assay (ELISA:Diesse™ Chorus IgM); a positive is based on the product specified cut-off. At the Institute for Clinical Pathology and Medical Research (ICPMR), Westmead, *M.pneumoniae* serology is performed using complement fixation assay (CF: Virion\Serion™ reagents) and a commercial Immunofluoresence assay (IFA: Vircell™ IgM). Tests are reported positive where a CF titre of 64 or higher is measured, or IgM is identified based on the product specified cut-off. In addition at ICPMR, PCR for *M.pneumoniae* nucleic acid is performed as an 'in house' assay. CHW refers specimens to ICPMR for this test.

2. Investigation and analyses

ACE study:

ACE study surveillance continued at CHW with enhanced real-time review of cases. In addition, we contacted the Sydney Children's Hospital, Randwick infectious diseases department to identify additional cases.

Pneumonia syndromic surveillance:

A retrospective analysis of 'pneumonia' ED presentations in children aged 0-16 years was conducted. Data from EDRE were used to provide greater coverage of NSW EDs and included 86 NSW facilities that participated continuously from 2010-2015 and had diagnosis complete in 75% or more records. These data represented 85% of all NSW ED presentations in 2015. 'Pneumonia' ED presentations in children aged 0-16 years in 2015 were compared to the mean annual count for 2010-2014. The PHREDSS system uses an automated threshold for signalling of five standard deviations (SD) above the expected count to indicate a significant increase. The expected count is the mean count for the same period over the previous five years. We compared 'pneumonia' presentations by age group (0-4 years and 5-16 years), and geographical location (metropolitan and rural). The number of admissions and the proportion of presentations that were admitted were analysed. In addition, we sought to sub-categorise the 'pneumonia' syndrome to include a subset of ICD-9, ICD-10 and SNOMED-CT diagnosis codes more specific to atypical pneumonia (CS, PB & SB agreed the included codes).

Sentinel Laboratory surveillance:

We requested records of *M. pneumoniae* serology and PCR nucleic acid detection tests from ICPMR and CHW diagnostic laboratories. We examined number of tests ordered and the proportion that returned positive for 2014 and 2015.

Data were collated in Microsoft (WA, USA) Excel™ v14 (2010) , and statistical testing

performed using CDC Epi Info™ (GA, USA). Proportions were compared using two-tailed chi-square test with Yates correction.

This was a public health investigation conducted using provisions in the NSW *Public Health Act*, 2010 therefore ethical approval was not required. De-identified ED data were released for the purposes of this investigation by the Executive Director, Centre for Epidemiology and Evidence using provisions in the NSW *Health Administration Regulation*, 2015. The ACE study was approved by the Sydney Children's Hospitals Network human research ethics committee.

Results

Suspected encephalitis surveillance

Between January and October 2015 (10 months), *M.pneumoniae* was associated with 29% (13/45) of suspected encephalitis cases identified by the ACE study, including five in July alone (Table 1). Between May 2013 and December 2014 (20 months), the ACE study identified four cases of suspected encephalitis associated with *M. pneumoniae* at CHW, of 79 total cases (5%; two tailed chi square p-value <0.001 comparing 2015 to 2013/14). Of the 13 cases in 2015, median age was 10.1 years (range 3-11.7); ten were male. Seven cases resided in outer western Sydney. All cases presented with an altered level of consciousness. Other symptoms/signs included: fever (8/13), headache (9/13), focal neurological signs (7/13), seizure(s) (6/13), cerebrospinal fluid pleocytosis (7/11), and abnormal neuroimaging (6/13). Four of the thirteen children were admitted to intensive care. The median length of stay in hospital was 7 days (inter-quartile range 5.5, 15.5). Following review of these patients by the expert panel, two of the thirteen were deemed not to have encephalitis but other neurological syndromes; one with cerebral venous sinus thrombosis, and the other a non-specific seizure episode.

SCH-Randwick reported two cases of *M. pneumoniae* associated suspected encephalitis hospitalised in 2015; one in February and one in July (Table 1). This was considered to be in keeping with expected case numbers (A Bartlett, personal correspondence).

Table 1: Cases of suspected encephalitis* associated with Mycoplasma pneumoniae infection#^.

Sex	DOA	Age (yrs)	Geographic Statistical Sub-division (SSD)
Cases identified by PAEDS surveillance at the Children's Hospital at Westmead			
M	29/12/2014	5.7	Sydney-Inner Western SSD
M	15/03/2015	11.7	Sydney-Blacktown SSD
M	17/04/2015	11.5	Sydney -St George/Sutherland SSD
M	22/04/2015	10.8	Sydney-Central Western SSD
F	4/07/2015	5.7	Sydney-Central Western SSD
F	10/07/2015	3.0	Sydney-Blacktown SSD
M	16/07/2015	10.8	Sydney-Blacktown SSD
M	25/07/2015	10.1	Sydney-Blacktown SSD
F	25/07/2015	10.7	Sydney-Blacktown SSD
M	6/08/2015	8.2	Sydney-Blacktown SSD
M	23/09/2015	10.7	Central West-Lachlan SSD
M	30/09/2015	8.5	Sydney-Central Northern SSD
M	25/10/15	8.4	Sydney-Blacktown SSD
Additional cases identified by infectious diseases at Sydney Children's Hospital, Randwick			
M	03/02/2015	9.9	Sydney-St George-Sutherland SSD
M	22/07/2015	10.9	Sydney-Northern Beaches SSD

*Suspected encephalitis: Age 0 to 14 years AND Hospitalised with acute encephalopathy (defined by altered level of consciousness, lethargy and/or personality change lasting ≥24 hours) AND Has one or more of the following: fever, seizures, focal neurological findings, at least one abnormality of cerebrospinal fluid (CSF; age determined pleocytosis, or elevated protein ≥ 40mg/dl), or EEG/ neuroimaging findings consistent with infection-related encephalitis.
#Defined as the presence of positive *M.pneumoniae* specific IgM antibodies on acute sera.
^In three cases an additional possible infectious cause was identified (one enterovirus PCR positive on stool; one respiratory syncytial virus (RSV) PCR positive on naso-pharyngeal specimen; one RSV and human meta pneumovirus (hMPV) PCR positive on naso-pharyngeal specimen).
Abbreviations: DOA=date of admission; PAEDS=Paediatric Active Enhanced Disease Surveillance network; SSD=statistical sub-division

Pneumonia syndromic surveillance:

In 2015, among children aged 0-16 years there were 5,337 ED presentations to 86 NSW hos-

pitals, which was significantly greater (> 5 SD) than the mean number of annual presentations (3,824) during the previous five years (2010-2014). Monthly counts of "pneumonia" presentations were increased from March to September 2015, peaking in August with 663 "pneumonia" presentations (Figure 1).

In 2015 the increase in "pneumonia" ED presentations, compared to the mean number of annual presentations of the previous 5 years, was greater in children aged 5-16 years than in children aged 0-4 years (Figure 1) and in metropolitan hospitals compared to the rural hospitals (Figure 2).

Of the "pneumonia" presentations in 2015, 3,032 were admitted from ED to a ward (not critical care), which was significantly greater than the mean number of annual admissions (2,483) in the previous 5 years. However, the proportion of presentations that were admitted from ED in 2015 (57%) was lower than the mean proportion admitted between 2010 and 2014 (64%).

Of the pneumonia ED presentations in children aged 0-16 years in 2015, 81% had non-specific diagnosis codes assigned (Table 2).

Laboratory sentinel surveillance:

Analysis of diagnostic laboratory data obtained from CHW showed both an increased number of *M.pneumoniae* tests ordered and an increase in positive results in 2015 compared to 2014 (Figure 3a). From March to September, there was a significantly higher proportion of positive tests in 2015 compared to 2014 (35% vs 9%; two tailed chi square p-value <0.001). The proportion was similarly higher comparing full years (34% vs 12%; p-value <0.001).

Data from ICPMR showed that a greater number of *M.pneumoniae* serology tests were ordered in 2015 compared to 2014, and there was a peak in positive serology results (CFT or IgM) in August (Figure 3b). The proportion of positive tests (CFT and IgM) was not significantly higher in 2015 compared to 2014 for the whole year (10% vs 7%; p-value <0.2), but showed statistical significance when stratified to a 5-14 years age group (11% vs 3%; p-value <0.001) and approached significance for March to September (5% vs 2%; p-value 0.07). Additionally, a significantly higher proportion of *M.pneumoniae* PCR tests returned positive in 2015 relative to the preceding two years (Table 3).

Discussion

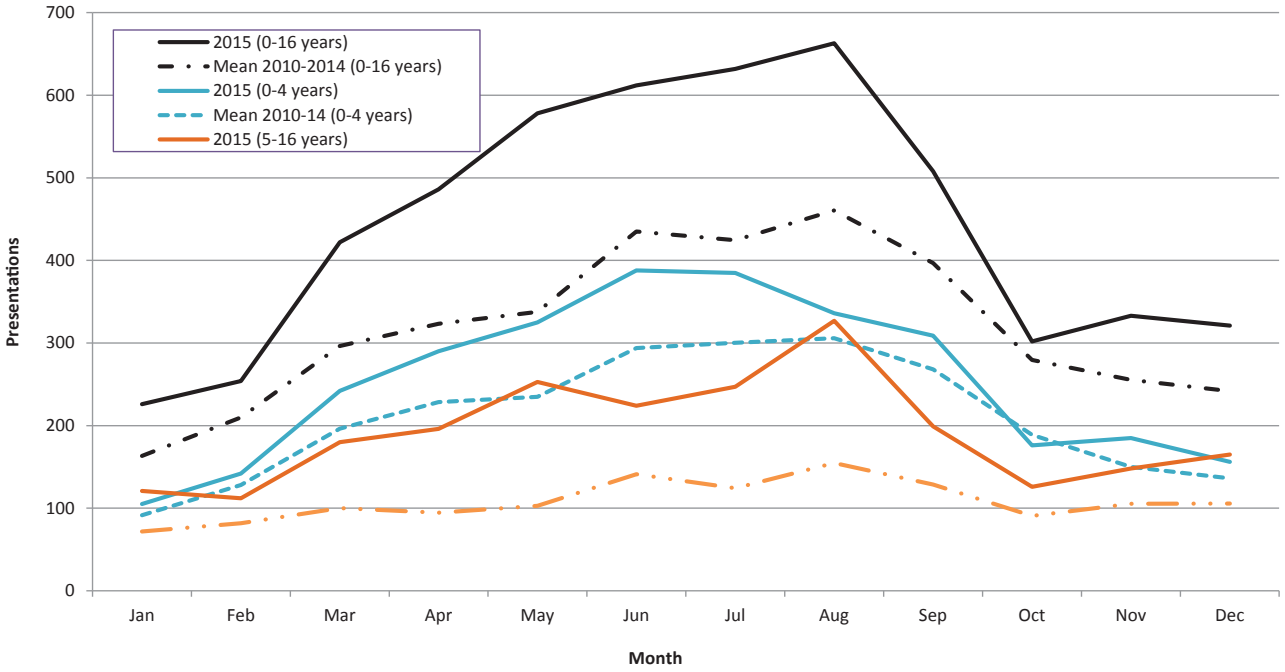
In 2015, active clinical surveillance for childhood encephalitis showed an increase in suspected encephalitis cases in NSW associated with *M.pneumoniae*. The cluster peaked in July in one geographic location, the Blacktown area. A similar increase in encephalitis cases was not observed, albeit passively, in Eastern Sydney (at SCHN-Randwick). In 2015, "pneumonia" ED presentations significantly increased in NSW in the age group in which *M.pneumoniae* is most common [5] (i.e. children aged 5-16 years) and in metropolitan locations. Laboratory surveillance showed increased frequency of testing, and tests returned positive for *M.pneumoniae* in 2015 compared with previous years at two sentinel referral laboratories. Together, this information was suggestive of epidemic *M.pneumoniae* activity in metropolitan Sydney in 2015. As this

Table 2: Frequency of top 5 diagnosis codes included in the PHREDSS "pneumonia" syndrome in 2015.

Diagnosis Code	Coding System*	Description	Frequency	Percentage
233604007	SNOMED-CT	Pneumonia (disorder)	2421	45%
J18.9	ICD-10AM	Diagnosis: Pneumonia, unspecified (Ed.1-Ed.9)	1135	21%
385093006	SNOMED-CT	Community acquired pneumonia (disorder)	540	10%
233606009	SNOMED-CT	Atypical pneumonia (disorder)	273	5%
53084003	SNOMED-CT	Bacterial pneumonia (disorder)	218	4%

*Abbreviations: SNOMED-CT = the Systematized Nomenclature of Medicine - Clinical Terminology; ICD-10AM = the Australian clinical implementations of the International Classification of Diseases ICD-10th revision.

Figure 1: Monthly counts of pneumonia Emergency Department presentations in children for 2015 (black solid lines), compared with each of the mean of the 5 previous 5 years (coloured dashed lines lines), persons aged 0-16 years, to 86 NSW hospitals in NSW, by age group



information was gathered, it was forwarded on to the WSLHD PHU and the Communicable Diseases Branch, Health Potection NSW. Clinicians (neurology, infectious diseases) at CHW and SCH-Randwick were made aware by email of the increased identification of *M. pneumoniae* associated encephalitis cases and to consider early testing and treatment. No further public health actions were undertaken.

We reported this cluster of *M.pneumoniae* associated encephalitis in Western Sydney and probable epidemic *M.pneumoniae* activity to demonstrate that active surveillance for childhood encephalitis has the potential to provide sentinel surveillance data to identify epidemic infectious disease activity. We have shown that combining surveillance data affords opportunities to cross-validate epidemic signals, and facilitate hypothesis testing. We have also shown that *M.pneumoniae* is likely an underappreciated cause of hospitalisation and acute morbidity in children in epidemic years.

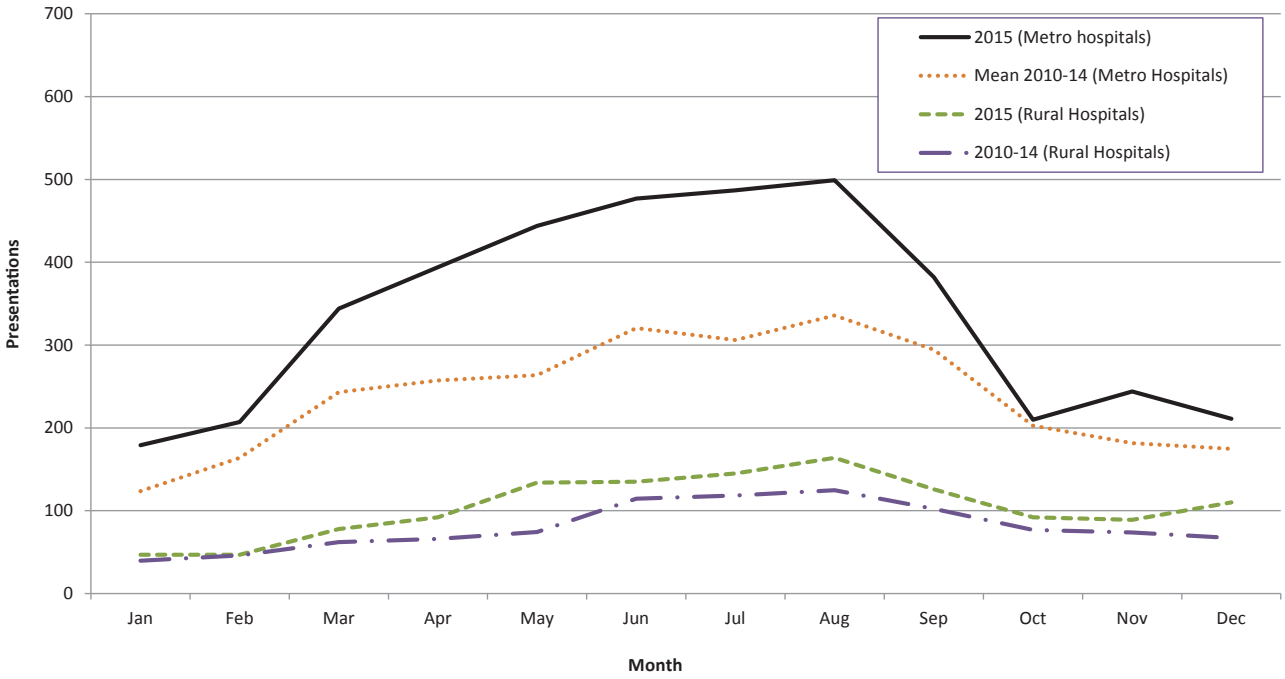
There are scarce published data describing the epidemiology of *M.pneumoniae* infection and disease from Australia. A recent molecular epidemiology study showed the circulation of diverse genotypes of *M.pneumoniae* in Sydney with a low prevalence of genetic markers of antibiotic resistance.[22] Furthermore, a clinical study from the Children's Hospital at Westmead showed *M.pneumoniae* infection to be most often associated with respiratory tract infection

Table 3: *Mycoplasma pneumoniae* polymerase chain reaction nucleic acid (PCR) testing performed at the Institute for Clinical Pathology and Medical Research (ICPMR) at Westmead, 2011 to 2015.

	2015	2014	2013	2012	2011
Total samples tested	617	501	409	401	211
Samples positive (%)	40 (6.5)	9 (1.8)	8 (2.0)	15 (3.7)	12 (5.7)
Two tailed Chi square p-value*		<0.001	<0.01	0.08	0.7

*compared with 2015. p-value <0.05 considered as statistically significant

Figure 2: Monthly counts of pneumonia Emergency Department presentations in children for for 2015 (solid lines), compared with the mean of the previous 5 years (dashed lines lines), to 86 NSW hospitals, by location



*Abbreviation: Metro = Metropolitan

Source (Figures 1-2): Emergency Department Records for Epidemiology (EDRE), held by the NSW Ministry of Health Secure Analyics for Population Health Research and Intelligence (SAPHaRI).

and children aged 5-9 years.[23] In this five year study, half of the cases occurred in a single 'epidemic' year (2001).[23] We could not identify any other contemporary published studies.

Epidemics of *M.pneumoniae* infection can result in a significant burden of disease. In 2015 there was a considerable increase in 'pneumonia' ED presentations amongst school aged children and an increase in admissions. A similar pattern was observed during an epidemic of *M.pneumoniae* in Scotland in 2010-11 where infection resulted in a high hospitalisation rate (59%) of cases with acute respiratory illness.[8] Furthermore, *M.pneumoniae* encephalitis results in one third to a half of cases suffering neurological morbidity (e.g. motor or cognitive dysfunction or seizures).[24-26] Given the significant increase in *M.pneumoniae* associated encephalitis that we observed in 2015, we are concerned about potential long-term morbidity arising from this epidemic year. We are undertaking a follow-up study that includes many of the cases described here that will further clarify neuro-psycological outcomes.

Monitoring *M.pneumoniae* disease activity is challenging for several reasons. Firstly, *M.pneumoniae* causes a variety of clinical syndromes, most commonly respiratory tract infections. Respiratory tract infections, including pneumonia, are among the most common reasons for children to present to primary care practitioners and emergency departments and are caused by a variety of pathogens.[5, 27] Furthermore, a Cochrane systematic review showed that *M. pneumoniae* cannot be reliably distinguished from other causes of pneumonia by clinicians on clinical grounds alone.[28] Despite these limitations, given the likely high proportion of pneumonia in school aged children caused by *M.pneumoniae,* we suggest that pneumonia when restricted to this age group is a potentially useful proxy for *M.pneumoniae* disease activity. The limitations of the laboratory diagnosis of *M. pneumoniae* infection preclude its use as a sole mechanism for disease surveillance. Serological diagnosis is hampered by cross-reactivity of IgM assays, limited sensitivity and specificity of single elevated titres of total antibody, and

Figure 3a: Mycoplasma pneumoniae IgM testing at the Children's Hospital at Westmead (CHW) 2014 – 2015.

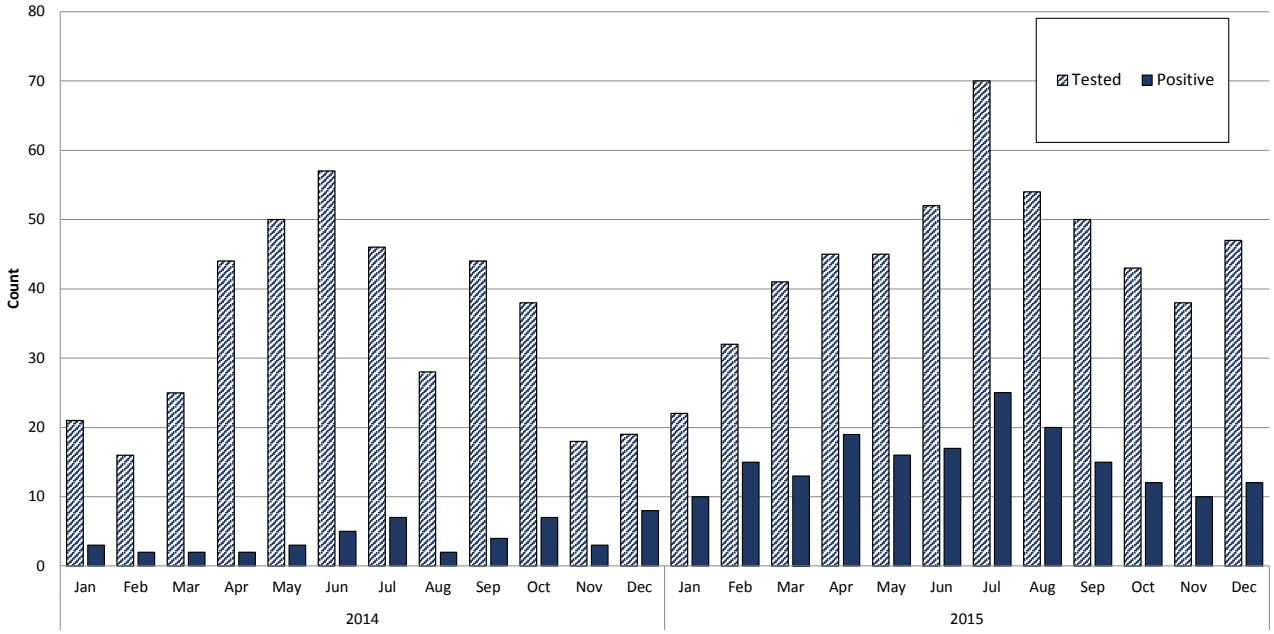
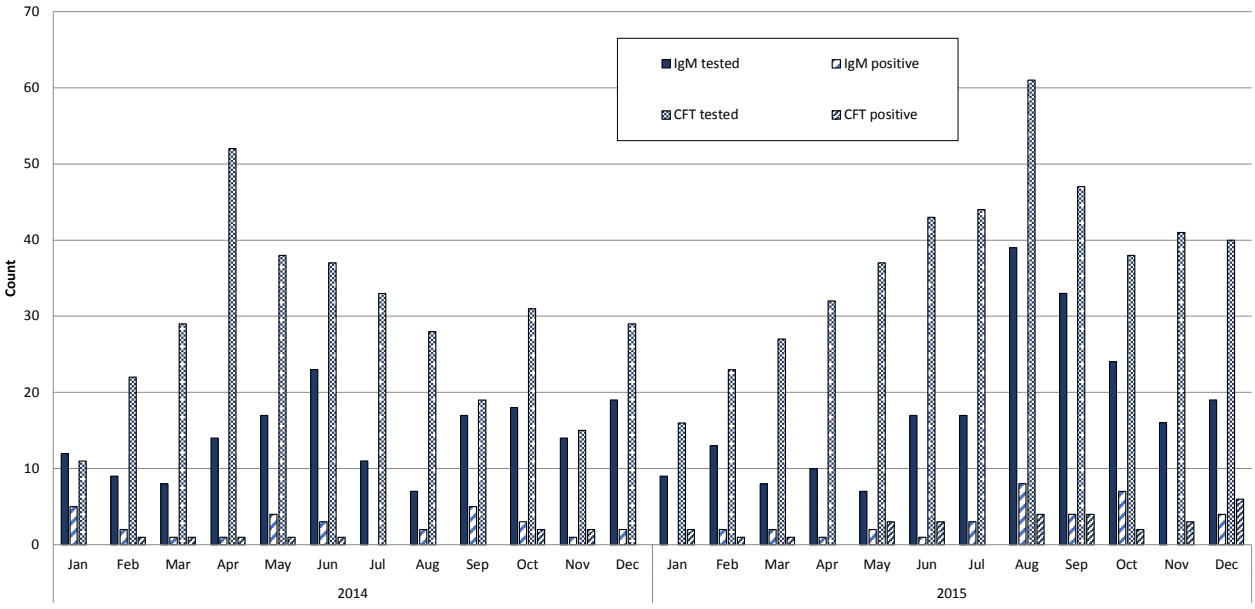


Figure 3b: Mycoplasma pneumoniae serology testing performed at the Institute for Clinical Pathology and Medical Research (ICPMR) at Westmead 2014 – 2015.



infrequent convalescent sampling required to demonstrate a fourfold rise in titres.[14, 29, 30] PCR is also limited by high rates of asymptomatic carriage in children.[4] As a result, cross validation of syndromic surveillance with laboratory surveillance, as performed in this outbreak investigation, is important in monitoring *M. pneumoniae* disease activity. This may still provide conflicting results, as shown in this investigation. In Norway, investigators have studied the possible monitoring of macrolide prescriptions at a population level, cross referenced with laboratory reports.[31] Further work is required to identify the best combination of tools for ongoing *M.pneumoniae* surveillance.

The Public Health response to infectious diseases outbreaks can be categorised into three core activities (i) monitoring and surveillance, (ii) communication and (iii) control of transmission. In this outbreak investigation, the PHU and Rapid Surveillance team undertook additional monitoring and surveillance of available data (PHREDSS) to identify the pattern of *M. pneumoniae* disease. Communication of the the evolving findings to children's hospital clinicians was facilitated through SCHN in August, 2015, to alert them to the possible epidemic period. Such alerting of clinicians supports earlier diagnosis and treatment, potentially reducing complications of severe illness, or exclusion of a diagnosis in critically ill patients. No specific community or healthcare transmission control measures were implemented in this situation. Here, our main goal was to cross validate surveillance mechanisms which proved complex and resulted in a delay in confidently identifying the epidemic. Community outbreaks remain largely undetected as *M.pneumoniae* surveillance is not routinely conducted.[32] As a result, this outbreak was a novel consideration for the PHU. These factors and the absence of clear nosocomial or institutional transmission resulted in the level of public health action being minimal. However, we note that *M. pneumoniae* outbreaks within healthcare facilities have been reported, with high attack rates and significant morbidity despite control measures.[14] The optimal public health response to community outbreaks of *M. pneumoniae* infection is unclear.[32] Given the likely community transmission, during future epidemics, additional public health actions for consideration include active communication with primary care practitioners to optimise testing and antimicrobial use, and with schools and child care facilities to emphasise preventive health behaviors. The possible need for direct communication with the public is emphasised by evidence that households may be central to amplifying transmission.[32]

We acknowledge a number of limitations with this analysis. Firstly, the use of a single positive IgM in the case definition for *M.pneumoniae* encephalitis lacks specificity; however this definition was unchanged across the surveillance period so should not affect trends. Secondly, we do not have an explanation for why the encephalitis cases were clustered in Western Sydney, despite evidence of an increase in pneumonia cases across metropolitan NSW. It is possible that cases at Sydney Children's Hospital, Randwick may have been missed given that case ascertainment at this site was based on clinician identification retrospectively rather than on established active surveillance. Furthermore, possible shared exposures were not explored directly with cases. Thirdly, ED provisional diagnoses are allocated at the end of the ED episode of care by a clinician (nurse or doctor) from a drop down list and there may be differences in ED provisional diagnosis coding practices across time and facility. Although we attempted to develop a more specific "atypical pneumonia" syndrome sub-category, the non-specific nature of ED diagnosis coding precluded this being useful. Fourthly, the assumption that up to a third of community acquired pneumonia in children aged 5-19 years is caused by *M.pneumoniae* is unproven in this study.[5] Finally, given the variability in the absolute number of laboratory tests performed at CHW and ICPMR for *M.pneumoniae* it is likely that testing is selective, and we do not know what additional factors apart from disease activity may have influenced this across the time period from which data were generated.

In conclusion, active, sentinel site surveillance for childhood encephalitis has the capacity to identify epidemic infectious diseases in children. Combining, and cross validating syndromic surveillance signals requires a collaborative approach and the combination of clinical, public health, and biostatistical expertise. We suggest that *M.pneumoniae* disease burden in Australia is currently inadequately measured, but that it may be considerable and should be the focus of future research.

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Original Article

Diagnostic testing in influenza and pertussis-related paediatric intensive care unit admissions, Queensland, Australia, 1997-2013

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Abstract

Severe respiratory infections make up a large proportion of Australian paediatric intensive care unit (ICU) admissions each year. Identification of the causative pathogen is important and informs clinical management.

Methods

We investigated the use of polymerase chain reaction (PCR) in the ICU-setting using data collated by the Australian and New Zealand Paediatric Intensive Care (ANZPIC) Registry from five ICUs in Queensland, Australia. We reviewed diagnostic testing among all pertussis and influenza-related paediatric ICU admissions between 01 January 1997 and 31 December 2013.

Results

There were 177 influenza-related and 78 pertussis-related ICU admissions. Overall, 157 (89%) influenza-related admissions had an influenza-specific diagnostic test conducted, of which 129 (82%) had a PCR test requested. Patients that were tested for influenza using non-PCR tests all occurred prior to 2007. An influenza-positive result was recorded for 130 (82%) of the tested influenza-related ICU admissions – 73% of all ICU admitted influenza-related cases. Among pertussis-related admissions, 63 (81%) had a pertussis-specific diagnostic test ordered, of which 60 (95%) were tested using PCR. A pertussis-positive result was recorded for 53 (86%) of those tested, and 68% of all ICU admitted pertussis-related admissions.

Conclusions

PCR has become the preferred diagnostic method to test influenza and pertussis-related ICU admissions, largely replacing other methods. This finding mirrors trends observed across other health care settings, but appears to have occurred earlier among ICU admissions. The move to PCR testing, has allowed more sensitive and rapid diagnosis of severe pertussis and influenza infections among children.

Keywords: pertussis, influenza, intensive care, paediatric, polymerase chain reaction, diagnostic testing

Introduction

Severe respiratory infections among infants and young children make up approximately one-third of all Australian paediatric intensive care unit (ICU) admissions each year.[1, 2] Respiratory infections can be caused by a range of viral and bacterial pathogens, and given that infants and children can manifest a broad array of non-specific symptoms, identifying the aetiology based on clinical presentation alone is difficult.[3-5] Identification of the causative pathogen is important, particularly in severe cases, in order to inform clinical and infection control management.[5] As such, infants and children that are hospitalised or admitted to ICU are likely to be tested for a broad range of respiratory pathogens.[6]

In the last decade, polymerase chain reaction (PCR) has largely replaced traditional diagnostic methods, such as culture and immunofluorescence, for routine testing of respiratory samples.[7, 8] In Australia, influenza and pertussis are notifiable conditions under public health legislation, and all cases that meet the case definitions must be reported to state and territory health departments.[9, 10] Influenza and pertussis are the two most common vaccine preventable notifiable diseases in Australia,[8, 11] and the increasing use of PCR has, in part, been linked to better case recognition and magnification of the number of notifications.[8, 12]

The incidence of paediatric influenza and pertussis-related ICU admissions has increased in recent years.[13, 14] It is unclear whether there has been a true increase in severe disease over this time period, or whether a shift in diagnostic methods (to PCR) has led to improved case detection and therefore more accurate coding of admissions. To date, only two Australian studies have reported diagnostic testing use among pertussis and influenza hospitalisations, however these studies focused on overall laboratory findings and not changes to laboratory methods over time.[3, 15] The aim of this study was to investigate the use of PCR in the ICU-setting by reviewing the diagnostic testing of pertussis and influenza-related paediatric ICU admissions in Queensland, Australia, over a 17-year period.

Methods

We conducted a retrospective cohort study using data collated by the Australian and New Zealand Paediatric Intensive Care (ANZPIC) Registry on ICU admissions between 01 January 1997 and 31 December 2013. Data relating to the admission of paediatric patients aged 0 to 16 years was extracted. The ANZPIC Registry collects paediatric intensive care patient episode information from contributing specialist paediatric ICUs (PICUs) as well as general ICUs (which admit mainly adult and some paediatric patients) across Australia and New Zealand.[2] Ethics approval for this study was obtained from the Children's Health Services Queensland Human Research Ethics Committee.

For this study, only admissions to contributing ICUs located in the state of Queensland were extracted. Data from two hospitals with PICUs located in Brisbane ('PICU A' and 'PICU B'), and three hospitals with general ICUs located in regional areas ('General ICU A', 'General ICU B', and 'General ICU C') were used. The two Brisbane-based PICUs, PICU A and PICU B, contributed data for the full study period (1997-2013), during which time they treated 10,264 and 13,158 all-cause admissions, respectively. The other three ICUs began contributing admission data for 0 to 16 year old patients to the ANZPIC Registry later: General ICU A from 2002 (1,028 all-cause paediatric ICU admissions 2002-2013), General ICU B from 2006 (331 all-cause paediatric ICU admissions 2006-2013), and General ICU C from 2009 (97 all-cause paediatric ICU admissions 2009-2013). Participating ICUs collect data in real-time and a single record is created for each ICU admission.[2]

Admissions are defined using ANZPIC Registry-specific standardised diagnosis codes,[16] and include 'principal diagnosis' (the diagnosis most directly responsible for the ICU admission), 'underlying diagnosis' (the principal underlying diagnosis contributing to the need for ICU

admission), and up to seven 'associated diagnoses'. Associated diagnoses are conditions additional to the principal and underlying reasons that contributed to the ICU admission, and can include other syndromes, diseases, abnormalities, or diagnoses identified on or during ICU admission. For this study, we extracted ANZPIC Registry data for all Queensland paediatric ICU admissions with diagnosis codes of 470 – Pertussis Syndrome, 720 – Pertussis, or 715 – Influenza Virus occurring in any of the diagnosis fields. The line-listed data extract included patient demographic variables and ICU admission details, as well as a hospital identification number. Immunisation history, medications/treatments prescribed during admission, and laboratory data used to support the diagnosis coding are not collected in the ANZPIC Registry.

Using the hospital identification number for each ICU admission, we searched for any respiratory diagnostic tests occurring from 14 days prior to ICU admission to seven days after ICU discharge (based on specimen collection date) in appropriate pathology databases (the Pathology Queensland Laboratory Information System, Auslab, and/or the Mater Pathology Laboratory Information System, Kestral). Where tests were found, we recorded the sample date, sample method (e.g. nasopharyngeal swab or aspirate), diagnostic method (e.g. PCR, serology, culture, antigen detection), and result for each test. Diagnostic method was coded as PCR (≥1 test done, where at least one PCR-based), non-PCR (≥1 test done, none PCR-based), or no tests found. The test results were coded as influenza positive only, pertussis positive only, other respiratory pathogen positive (influenza and pertussis negative), influenza and other respiratory pathogen positive, pertussis and other respiratory pathogen positive, or negative (no respiratory pathogen identified). Co-detection was defined as a respiratory test positive for another respiratory pathogen, in addition to a positive influenza/pertussis result. Where another respiratory pathogen was detected, the organism was recorded.

Descriptive statistics are presented as frequency (percentage) or median (range) as appropriate. Comparisons between groups were calculated using the non-parametric Mann-Whitney U-test. We calculated the proportion of ICU admissions tested using any testing modality, as well as the proportion of admissions with a matching positive diagnostic test result. All analyses were conducted using Stata statistical software v.12 (StataCorp, College Station, TX, USA).

Results

From 1997 to 2013, there were 177 influenza-related paediatric ICU admissions and 78 pertussis-related paediatric ICU admissions in the five participating hospitals (Table 1). Admissions predominantly occurred in Brisbane (PICU A, 52%; n=133 and PICU B, 40%; n=103). Children with influenza-related admissions were older than children with pertussis-related admissions (median age: 2.0 years vs. 51 days, respectively, $p<0.001$), and had a shorter median length of ICU stay (2.6 days vs. 4.3 days, respectively, $p=0.049$). A total of 16 deaths were recorded, 8 (5%) influenza-related admissions and 8 (10%) pertussis-related admissions.

Influenza-related admissions were highest in 2011 (n=26 admissions), and 119 (67%) occurred between 2007 and 2013 (Figure 1). Overall, 157 (89%) influenza-related admissions had an influenza-specific diagnostic test conducted. An influenza positive result was recorded for 130 (83%) of those tested and 73% of all influenza coded admissions (Table 2). Prior to 2007, 28 influenza-related admissions were tested using non-PCR diagnostic tests, which included one or a combination of: immunofluorescence, antigen detection, culture, or serology. From 2008 onwards, all tested influenza-related admissions included a PCR test (10 in combination with antigen detection, serology, or immunofluorescence).

Pertussis-related admissions between 2009 and 2012 accounted for 62% (n=48) of total pertussis-related admissions during the study period (Figure 2). Among pertussis-related admissions, 62 (80%) had a pertussis-specific diagnostic test ordered. A pertussis-positive result was

recorded for 53 (86%) of those tested and for 68.0% of all pertussis coded admissions. Only three pertussis-related ICU admissions, all prior to 2002, were tested using a non-PCR diagnostic method (immunofluorescence) and all three had negative results. Serology, antigen detection, culture, or immunofluorescence were used, in combination with PCR, for 17 of the remaining 59 tested pertussis-related admissions.

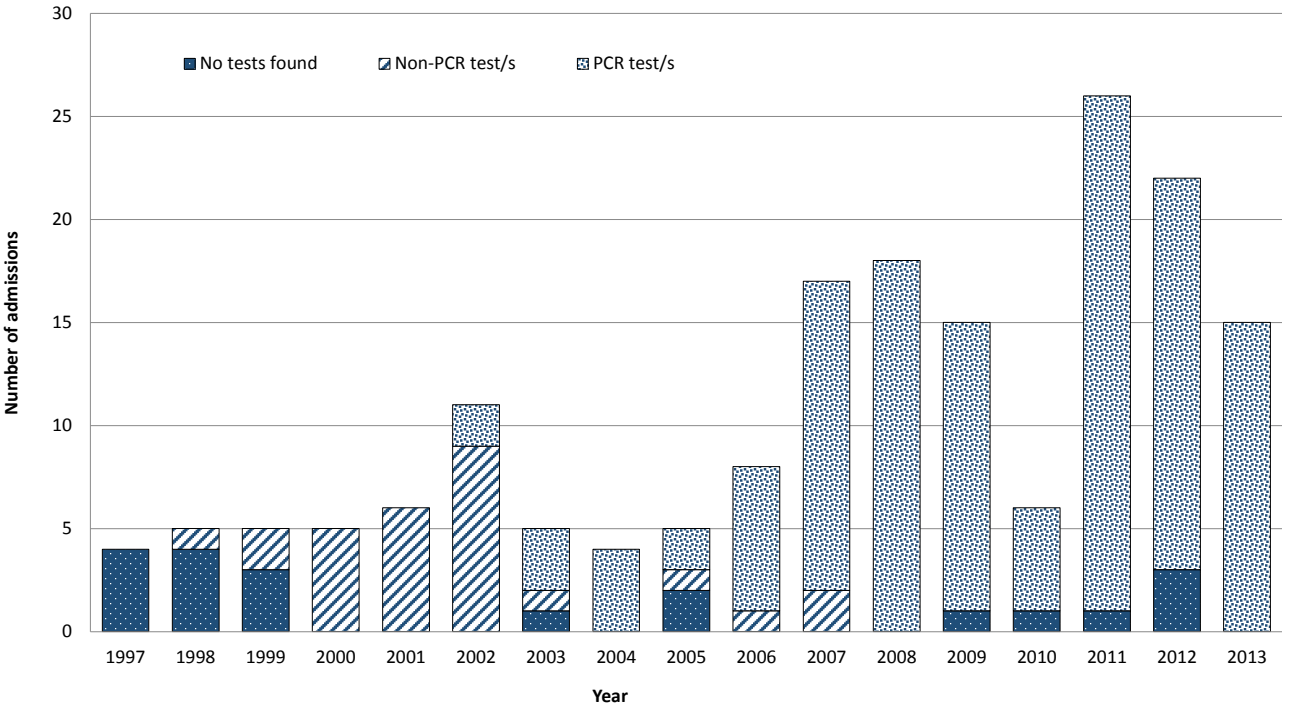
Co-detection of another respiratory pathogen was identified in 14% (n=24) influenza-related admissions and 19% (n=15) pertussis-related admissions (Table 1). Additionally, there were 18 ICU admissions coded as influenza-related, that tested negative for influenza but positive for one or more respiratory pathogens, including: parainfluenza (type 2 n=1, type 3 n=9), rhinovirus (n=5), *Haemophilus influenzae* type

Table 1: Influenza and pertussis admissions, 1997-2013, Queensland Australia

	Influenza	Pertussis
Total admissions	**177**	**78**
Admitting ICU [n (%)]		
PICU A	105 (59%)	28 (36%)
PICU B	63 (36%)	40 (51%)
General ICU A	8 (5%)	2 (3%)
General ICU B	1 (1%)	4 (5%)
General ICU C	0 (0%)	4 (5%)
Sex - Male [n (%)]	97 (55%)	42 (54%)
Age [median (range)]	2.0 years (16 days – 14.6 years)	51 days (16 days – 8.4 years)
Length of stay [median (range)]	2.6 days (0.2 - 57.7)	4.3 days (0.3 - 120.3)
Deaths [n (%)]	8 (5%)	8 (10%)
Tested for coded illness [n (%)]	157 (89%)	62 (80%)
Number of coded pathogen specific tests performed per admission [a] [median (range)]	2 (1 - 10)	3 (1 - 21)
Admissions with relevant positive laboratory test [n (%)]	130 (73%)	53 (68%)
Co-detection of other respiratory pathogen [n (%)]	24 (14%)	15 (19%)
RSV-positive	12 [b]	4
adenovirus-positive	6 [b]	3
rhinovirus-positive	3 [c]	6 [d]
parainfluenza type 3-positive	2	2 [d]
human metapneumovirus-positive	3 [c]	1

a. where one or more diagnostic tests conducted. Cases with no test identified were excluded.
b. one case positive for influenza, RSV, and adenovirus – counted in both rows.
c. two cases positive of influenza, rhinovirus, and human metapneumovirus – counted in both rows.
d. one case positive for pertussis, rhinovirus, and parainfluenza type 3 – counted in both rows.

Figure 1: Influenza admissions by test method and year, 1997-2013, Queensland Australia



B (HiB, n=2), and respiratory syncytial virus (RSV, n=2). Among these 18 admissions, only one HiB-positive admission and both RSV-positive admissions had relevant HiB or RSV-specific ICU diagnosis codes in addition to the influenza ICU diagnosis code. There were no influenza/pertussis co-detections.

Discussion

The vast majority of paediatric ICU admissions for influenza and pertussis in Queensland, Australia, between 1997 and 2013 had at least one pathogen-specific diagnostic test conducted. This aligns with the clinical imperative to identify the aetiology of severe respiratory illness. For both illnesses, PCR largely replaced other methods over time and became the predominant diagnostic test, mirroring what has previously been observed among national influenza and pertussis notifications.[8] Interestingly, the move to PCR testing appears to have occurred earlier among ICU admissions (from approximately 2001-2002 onwards) than among notifications (from approximately 2007 onwards).[8] As PCR testing was still relatively novel in 2001-2002, its use at that time will have been expensive, limited to larger laboratories, and run as one-off diagnostic tests rather than mass routine testing.[17] However the benefits of PCR compared to culture, such as much higher sensitivity (94% vs. ~15%, respectively) and faster results,[18] likely still made it a preferable choice for clinicians treating infants with severe respiratory infections. It was only in 2005 that public funding commenced, under the Australian Government-funded Medicare Benefits Schedule, for laboratories to test clinical specimens using PCR.[19] Additionally, during the 2009 H1N1 influenza pandemic, public funding was allocated to laboratories to purchase equipment (notably PCR suites) to enhance capacity.[20] While funding facilitated the expansion of PCR availability, the development and use of large-scale, multiplex PCR assays has allowed testing and identification of a broader range of respiratory pathogens, including the detection of co-infections.[21-25]

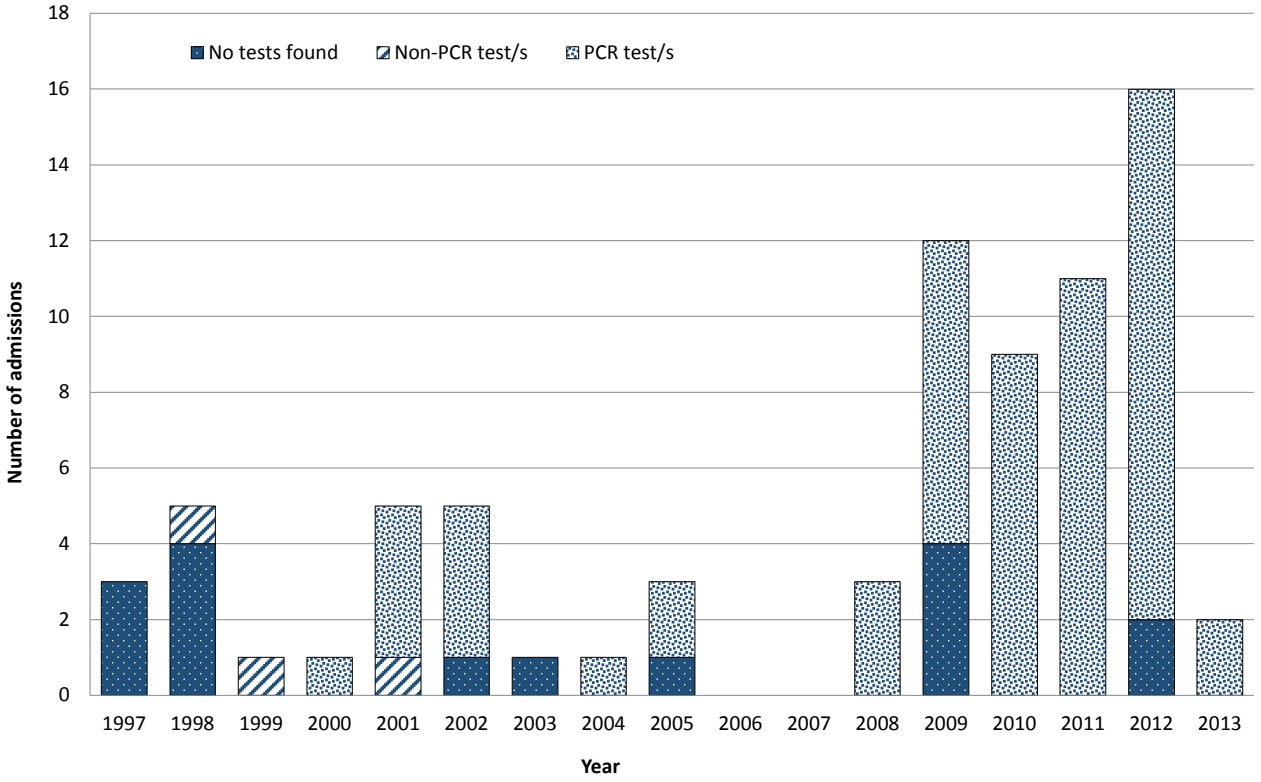
The ANZPIC Registry is a well-established dataset, and has been collating data from participating ICUs since 1997. A particular strength of the Registry is that it captures data from the two large dedicated PICUs in Queensland, as well as from three smaller general ICUs in more regional areas, therefore capturing the vast majority of paediatric ICU admissions across the state

Table 2: Influenza and pertussis paediatric ICU admissions by test type and result, 1997-2013, Queensland Australia

	Influenza						Pertussis					
Year	Total admissions n	Non-PCR test/s n (% positive)[a]	PCR test/s n (% positive)[a]	Not tested n	Tested % [b]	Test positive % [a]	Total admissions n	Non-PCR test/s n (% positive)[a]	PCR test/s n (% positive)[a]	Not tested n	Tested % [b]	Test positive % [a]
1997	4	0 (-)	0 (-)	4	0	0	3	0 (-)	0 (-)	3	0	0
1998	5	1 (100)	0 (-)	4	20	20	5	1 (0)	0 (-)	4	20	0
1999	5	2 (50)	0 (-)	3	40	20	1	1 (0)	0 (-)	0	100	0
2000	5	5 (100)	0 (-)	0	100	100	1	0 (-)	1 (100)	0	100	100
2001	6	6 (100)	0 (-)	0	100	100	5	1 (0)	4 (75)	0	100	60
2002	11	9 (100)	2 (100)	0	100	100	5	0 (-)	4 (100)	1	80	80
2003	5	1 (0)	3 (100)	1	80	60	1	0 (-)	0 (-)	1	100	0
2004	4	0 (-)	4 (100)	0	100	100	1	0 (-)	1 (100)	0	100	100
2005	5	1 (100)	2 (100)	2	60	60	3	0 (-)	2 (100)	1	67	67
2006	8	1 (100)	7 (86)	0	100	88	0	-	-	-	-	-
2007	17	2 (100)	15 (100)	0	100	100	0	-	-	-	-	-
2008	18	0 (-)	18 (83)	0	100	83	3	0 (-)	3 (100)	0	100	100
2009	15	0 (-)	14 (79)	1	93	73	12	0 (-)	8 (88)	4	67	58
2010	6	0 (-)	5 (80)	1	83	67	9	0 (-)	9 (100)	0	100	100
2011	26	0 (-)	25 (72)	1	96	70	11	0 (-)	11 (89)	0	100	89
2012	22	0 (-)	19 (84)	3	86	73	16	0 (-)	14 (88)	2	88	75
2013	15	0 (-)	15 (53)	0	100	53	2	0 (-)	2 (100)	0	100	100
Total	177	28 (93)	129 (81)	20	89	73	78	3 (0.0)	59 (90)	16	80	68

a. percentage of admissions positive for specific pathogen
b. percentage of admissions tested for specific pathogen using any testing modality

Figure 2: Pertussis admissions by test method and year, 1997-2013, Queensland Australia



and allowing generalisability of the findings. Additionally, by keeping data definitions largely consistent over a 17-year period, it provides the opportunity to analyse trends over a long time frame. A limitation of the ANZPIC Registry is that laboratory results are not captured, and the diagnosis codes have not previously been validated. We found that for approximately 70% of influenza and pertussis-related ICU admissions there was a corresponding relevant positive influenza or pertussis laboratory test result. When limited to only those admissions that had a diagnostic test conducted (89% of influenza-related admissions, 80% of pertussis-related admissions), approximately 85% had a matching positive result. We were unable to locate any previous studies, specifically validating coding for ICU admissions, with which to compare our results. Our findings however, were consistent with a previous study which validated the ICD-10 coding of influenza and pertussis hospitalisations in Western Australia.[15]

Our results likely represent minimum values as we were reliant on the accuracy and completeness of the databases that we used. We included all ICU admissions coded as influenza and pertussis, however any admissions that were due to, but not coded as, 'influenza' or 'pertussis' would have been missed. Similarly, as we did not include a medical chart review, we will have included any admissions incorrectly coded as influenza or pertussis-related. For example, we identified 18 influenza-coded ICU admissions that were negative for influenza, but positive for other respiratory pathogens. While these ICU admissions may have been diagnosed as influenza-related based on clinical symptoms, it is possible that there was some misclassification with the ANZPIC Registry coding, particularly influenza coded admissions where parainfluenza or *Haemophilus influenzae* type B were laboratory diagnosed. Additionally, although we conducted an extensive search of the pathology datasets, any diagnostic tests not contained within the database, for example, tests conducted by a private pathology provider or outside of our search criteria (between 14 days before ICU admission through to 7 days after ICU discharge), will have been missed. However, it is reassuring that the majority of ICU admissions had at least one diagnostic test identified.

The move to PCR testing in the ICU setting, allowing more sensitive and rapid diagnosis of respiratory pathogens compared to earlier diagnostic methods, may have improved clinical management of severe paediatric pertussis and influenza infections and led to a reduction in overall health care costs. Compared to bacterial pathogens, viruses are more commonly responsible for acute respiratory infections in children.[7, 26] Therefore rapid detection of a bacterial pathogen may aid a clinician's decision to prescribe and/or continue appropriate antibiotics, thus likely improving clinical management.[26] Although detection of a viral pathogen in a general practice setting would allow a GP to withhold antibiotic treatment, in the ICU setting, viral detection may not alter antibiotic use due to concerns of secondary bacterial infection.[7, 26] However additional (often unnecessary) laboratory tests or diagnostic imaging, pursued where the illness aetiology is not yet established, may be avoided due to the fast turn-around of PCR results, contributing to an overall reduction in health care costs.[7, 27] Furthermore, rapid diagnosis in a hospital/ICU setting would allow appropriate infection control measures to be enforced, limiting the likelihood of nosocomial infections.[7]

In conclusion, PCR has become the preferred diagnostic method in influenza and pertussis-related ICU admissions. This finding mirrors the trends observed more broadly across other health care settings.

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Conflicts of interest

SBL reports not having shares, paid employment, or consultancies with any influenza vaccine manufacturer; he has been an investigator on vaccine and epidemiological studies sponsored by bioCSL, Merck, GSK, Novartis, and Sanofi; his institute has received honoraria from Merck for talks he has given on rotavirus epidemiology and vaccines. Authors MCK, SS, RSW, JAM, and MGC declare that they have no competing interests.

Author contributions

All authors (MCK, SS, RSW, JAM, MGC and SBL) contributed to the study design. MCK and SS obtained the data. MCK conducted the data analysis and drafted the manuscript. All authors (MCK, SS, RSW, JAM, MGC and SBL) contributed to the interpretation of the results and revision of the manuscript. All authors (MCK, SS, RSW, JAM, MGC and SBL) read and approved the final manuscript.

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Short report

Children with melioidosis in Far North Queensland are commonly bacteraemic and have a high case fatality rate

Simon Smith, James D. Stewart, Catherine Tacon, Neil Archer and Josh Hanson

Abstract:

Paediatric melioidosis is uncommon in Northern Australia. In the Northern Territory, children with melioidosis often report an inoculation event and localised skin and soft tissue infections predominate. However, in Far North Queensland, children with melioidosis are frequently bacteraemic and have a high case fatality rate. To confirm this observation, all culture-confirmed cases of *Burkholderia pseudomallei* processed at Cairns Hospital between 1998 and March 2017 were reviewed. During the study period, *B. pseudomallei* was isolated from 223 people; ten (4%) were children (aged from three days to 14 years). Bacteraemia occurred in 6/10 (60%) children compared with 161/213 (76%) adults (p=0.24). The primary diagnosis was localised, cutaneous disease in three children, meningoencephalitis in two and pneumonia in two. Three had bacteraemia with no primary source evident. No child had a parotid abscess or liver abscess. Five children (50%) died, and all of whom were bacteraemic.

Keywords: Tropical medicine, melioidosis, paediatrics

Background and methods

Melioidosis, a disease caused by the environmental bacterium *Burkholderia pseudomallei*, has a diverse range of clinical presentations. Some patients have skin and soft tissue infections (SSTI) that resolve without antibacterial therapy, while others present in septic shock and have a high case fatality rate, even with optimal supportive care. In adults, clinical presentation is strongly linked to the presence of comorbidities, particularly diabetes mellitus, renal disease, chronic lung disease and hazardous alcohol use. Indeed, the disease is uncommon in adults without these conditions.[1] Conversely, children with melioidosis usually lack comorbidities[2] and uncommonly develop symptomatic melioidosis.[3] The reason that only some children develop symptomatic infection may relate to the route of transmission, the size of the inoculum, the presence of bacterial virulence factors or host susceptibility.[4]

Adults have similar presentations in different countries, but in children, the clinical phenotype varies significantly by geographic location. In Southeast Asia, children with melioidosis commonly have suppurative parotitis and liver abscesses, possibly due to the ingestion of *B. pseudomallei* contaminated water.[5, 6] Bacteraemia is reported in over a third of hospitalised cases in Thailand.[7] In contrast, in the Northern Territory (NT) of Australia, bacteraemia occurs in only 16%, much less frequently than in adults. Children with melioidosis usually report an inoculation event; SSTIs predominate while parotid involvement is unusual.[2]

The paediatric case fatality rate is over 20% in Asia compared to 7% in the NT. [2, 5, 7] This is at least partly explained by access to healthcare;[2] however, the higher rate of bacteraemia in Asian case series also contributes. [5, 7] High case fatality rates are also seen with neonatal melioidosis and neurological melioidosis.[8, 9]

In Australia, adults with melioidosis have a similar prognosis wherever they are managed,[1, 10] however, anecdotally, children with melioidosis in Far North Queensland (FNQ) have a less benign clinical course than that reported in the NT. To confirm this observation, all culture-confirmed cases of *B. pseudomallei* processed at Cairns Hospital between 1998 and March 2017 were reviewed. Cairns Hospital provides microbiological laboratory services for the Cairns region, Cape York Peninsula (CYP) and Torres Strait Islands (TSI). The study was approved by the Far North Queensland Human Research Ethics Committee.

Results

During the study period, *B. pseudomallei* was isolated from 223 people; ten (4%) were children (aged from three days to 14 years); six (60%) of whom were male (Table 1). Four children identified as Aboriginal or Torres Strait Islanders, three were Caucasian and three were from Papua New Guinea (PNG). Three children acquired their infection in PNG, three in the Cairns region, two in the Torres Strait and two on the CYP. Only two children recorded an inoculation event; one child injuring his head swimming in a flooded river in Cairns and one child from PNG having mud applied to an open head wound by a traditional healer. Only two children had classical risk factors for melioidosis – one with diabetes mellitus and another receiving high dose corticosteroids for systemic lupus erythematosus (SLE). Both cases survived. There was one neonate in our case series who died within two days of hospitalisation.

Bacteraemia occurred in 6/10 (60%) children compared with 161/213 (76%) adults (p=0.24). The primary diagnosis was localised, cutaneous disease in three children, meningoencephalitis in two and pneumonia in two. Three had bacteraemia with no primary source evident. No child had a parotid abscess or liver abscess. Five children (50%) died compared with 26/213 (12%) adults (p=0.001). Every child that died was bacteraemic. Three children died within two days of hospitalisation, none of whom received antibacterial therapy with *B. pseudomallei* cover. Two children died despite appropriate antimicrobial therapy and intensive care unit (ICU) support; one child with hydrocephalus requiring an external ventricular drain died 14 days after admission and one child with multi-organ failure requiring extracorporeal membrane oxygenation, died four days after hospitalisation.

Table 1. Demographics, risk factors, clinical presentation and outcomes of paediatric melioidosis cases in Far North Queensland (n=10)

Age(yrs) /Sex	Location	Inoculation event	Comorbidities	Weight in kg/ (Percentile)	Primary site of infection	Bacteraemic	ICU Admission	Died
0M	TSI	No	Neonate	3 (21)	Bacteraemia	Yes	Yes	Yes
4M	PNG	No	Malnourished	13 (<3)	CNS	Yes	No*	Yes
6F	PNG	No	Malnourished	14 (<3)	Pneumonia	Yes	No†	Yes
6M	CYP	No	Nil	19 (15)	Pneumonia	Yes	Yes	Yes
10M	PNG	Yes	Nil	30 (35)	Bacteraemia	Yes	Yes	Yes
11M	Cairns	No	Diabetes mellitus	35 (42)	SSTI	No	No	No
11F	TSI	No	Nil	N/A	SSTI	No	No	No
12F	Cairns	No	Nil	50 (79)	CNS	No	Yes	No
13M	Cairns	Yes	Nil	58 (76)	SSTI	No	No	No
14F	CYP	No	SLE, immunosuppressed	75 (95)	Bacteraemia	Yes	No	No

TSI = Torres Strait Islands; PNG = Papua New Guinea; CNS = Central nervous system; CYP = Cape York Peninsula; SSTI = Skin and soft tissue infection; N/A = Not available; SLE = Systemic lupus erythematosus
* Intubated and awaiting ICU bed, however died in Emergency Department
† Intubated in remote hospital and awaiting ICU bed in Cairns, however died prior to transfer

Discussion

There were only 10 cases over the study period, demonstrating that paediatric melioidosis is uncommon in FNQ. Nonetheless, the high case fatality rate and common finding of bacteraemia contrasts starkly with NT findings. This may be partly due to the small sample and reporting bias. Three of the children that died were PNG nationals, all were bacteraemic and two had significant comorbidity. Delayed ICU admission and poor physiological reserve resulting from socioeconomic disadvantage almost certainly contributed to their poor outcomes.

The higher proportion of bacteraemic cases might result from less aggressive case finding of SSTI, which was much less common than in the NT. In remote communities, patients commonly receive co-trimoxazole for mild SSTI (to treat community-acquired methicillin-resistant *Staphylococcus aureus*) without collection of samples for culture. Furthermore, *B. pseudomallei* infection may resolve in the absence of antimicrobial therapy.[11]

However, acknowledging these potentially confounding factors, the rate of bacteraemic melioidosis in adults and children in FNQ continues to be amongst the highest ever reported.[1] This is particularly relevant in the paediatric population given their extremely poor prognosis if bacteraemic. The case fatality rate of bacteraemic children in a Thai case series[7] was 60% and was even higher in a Cambodian case series (72%).[5] In our case series all but one (83%) of the bacteraemic children died. In the NT, there were only three children that died over the 24 years of one study however, two of the three cases were bacteraemic, while the third did not have blood cultures collected.

B. pseudomallei has multiple potential virulence factors and a highly variable genome, which partly explain the disease's protean clinical manifestations. It is possible that this might result in a greater propensity for patients to develop bacteraemia and its associated complications when infected with particular strains. Notably, non-bacteraemic skin infections have been associated with strains that lack the virulence factor filamentous hemagglutinin gene, fhaB3 – a gene that may be absent in FNQ.[12] In our study, a minority of patients had comorbidities or reported inoculation events which would support the hypothesis that patients were infected with more virulent strains, however this contention is limited by the retrospective nature of the study.

These findings are provocative, but they require prospective validation. It should also be noted that paediatric melioidosis remains uncommon in FNQ. Clinicians should only prescribe empirical regimens covering *B. pseudomallei* in children if they have a high clinical suspicion.

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Policy

Position statement on interferon-γ release assays for the detection of latent tuberculosis infection

Ivan Bastian, Chris Coulter and the National Tuberculosis Advisory Committee (NTAC)

Summary

Interferon-*γ* release assays (IGRAs), such as the Quantiferon (QIFN) TB-Gold Plus assay (Qiagen, Hilden, Germany) and the T-SPOT.*TB* test (Oxford Immunotec Limited, Abingdon, United Kingdom), are marketed as a substitute for the tuberculin skin test (TST) for the detection of latent tuberculosis infection (LTBI). The relative merits of IGRAs and TST have been hotly debated over the last decade. The specificity of IGRAs has been optimised by using *Mycobacterium tuberculosis*-specific antigens. However, IGRAs are functional *in vitro* T-cell-based assays that may lack reproducibility due to specimen collection, transport, processing and kit manufacturing issues.

Longitudinal studies comparing the ability of IGRAs and TST to predict the future development of active tuberculosis disease (TB) are the ultimate arbiters on the respective utility of these assays. Three meta-analyses addressing this comparison have now been published and clinical experience with IGRAs is accumulating. The systematic reviews show that IGRAs and TST have similar (but poor) ability to identify patients with LTBI at risk of developing active TB disease. The improved specificity of IGRAs however may reduce the number of patients requiring preventative therapy.

Based on these meta-analyses, The National Tuberculosis Advisory Committee (NTAC) now recommends either TST or an IGRA for the investigation of LTBI in most circumstances. Both tests may be used in patients where the risk of progression to active TB disease is high and the disease sequelae potentially severe (eg. LTBI testing in immunocompromised patients or those commencing anti-tumour necrosis factor-α (TNF) therapy). Neither test should be used in the investigation of active TB disease (though TST and/or IGRA may be used as supplementary tests in paediatric cases). The choice of test for serial testing in healthcare workers (HCWs) remains controversial. A preference remains for TST in this circumstance because IGRAs have been bedevilled by higher rates of reversions and conversions when used for serial testing. These recommendations supersede all previous NTAC IGRA statements.

Background

Detection and treatment of latent tuberculosis infection (LTBI) is an increasingly important element of tuberculosis (TB) control efforts in Australia and other low-incidence countries.[1,2] *In vitro* T-cell based interferon-*γ* release assays (IGRAs) are marketed as a substitute for the tuberculin skin test (TST) for the detection of LTBI.

The National Tuberculosis Advisory Committee (NTAC) has released position statements on the use of these assays (the last statement being in early 2012) and has undertaken to revise the recommendations on a regular basis.[3] As for the 2012 statement, the Committee has followed a template recommended in a survey of international IGRA guidelines by Denkinger et al.[4] Each Committee member reviewed one of the following sub-sections. The Committee then

discussed each member's literature review and proposed recommendation for each sub-section before reaching a consensus position.

The Committee has not formally graded the quality of the evidence supporting each recommendation but has cited meta-analyses where possible and has provided a few key references for each sub-section.

Summary of available commercial interferon-*y* release assays

The methodology for TST and IGRAs has been described in detail elsewhere.[5,6] Briefly, tuberculin (or purified protein derivative-PPD) has been used as an *in vivo* test for LTBI for over 50 years.[5] Tuberculin is injected intradermally on the volar aspect of the forearm; the diameter of induration is read 48 hours later. Disadvantages of the TST include that the patient must return to the clinic for the result to be read (leading to large drop-out rates) and that the TST lacks specificity because the tuberculin preparation contains antigens that cross-react with BCG and non-tuberculous mycobacteria (NTM).[5,7] However, TST's long history of use has provided valuable research data and experience, particularly longitudinal data that provides important predictive information, that is slowly becoming available for IGRAs.[3]

The Quantiferon (QIFN) TB-Gold Plus assay (Qiagen, Hilden, Germany) is the most-commonly used IGRA in Australia. The specificity of this assay has been optimised by utilising pooled synthetic antigens, such as early secretory protein 6 (ESAT-6) and culture filtrate protein 10 (CFP-10), from the *M. tuberculosis*-specific region of difference 1 (RD1).[8] Four tubes - two test tubes containing TB antigens, a positive control tube (containing mitogen), and a negative control tube - are inoculated with the patient's blood; incubated for 16–24 hours; the plasma is separated; and the IFN-γ concentration released from lymphocytes measured by an ELISA. Unlike previous versions of the Quantiferon assay, QIFN TB-Gold Plus has a second antigen tube with peptide configuration designed to assess CD8 T lymphocyte response as well as a CD4 response.

An alternative commercial assay, the T-SPOT.*TB* test (Oxford Immunotec Ltd, Abingdon, Oxfordshire, UK), is available but has not been marketed widely in Australia. An enzyme-linked immunospot (ELISPOT) methodology is used to enumerate activated effector T-cells that react to TB-specific peptides from ESAT-6 & CFP-10.[9] The assay is technically demanding requiring separation and counting of peripheral blood mononuclear cells, and subjective reading by a technician. However, some studies suggest that the T-SPOT.*TB* test is more sensitive than the QIFN tests, particularly in immunocompromised individuals.[10]

The antigens employed in both IGRA formats are absent from BCG and most NTM, but present in *M marinum*, *M. kansasii*, and *M. szulgai*.[7,8] The antigens may also be present in other unrecognised un-sequenced NTM. A small potential for cross-reaction with NTM therefore remains even with the IGRAs.

The following NTAC guidelines consider the QIFN and T-SPOT.*TB* tests as comparable assays and, unless specified, refer to these tests by the generic term "interferon-*y* release assays". The choice of commercial IGRA that may be used is left with Australian laboratories and other healthcare professionals.

Review of recent literature and other national guidelines

Successive NTAC statements have noted the need for longitudinal studies estimating the performance of IGRAs in predicting the long-term progression to active TB disease in untreated individuals. The utility and interpretation of IGRAs depend on such studies. The last NTAC statement summarised two relevant meta-analyses that found that both IGRAs and TST were poor predictors of subsequent development of active TB disease.[3,11,12] For example, Rangaka et al analysed nine studies that reported incidence rates per person time of follow-up and

found TB rates in IGRA-positive patients were only 4-48 cases per 1,000 person-years and were even lower (2-24 cases per 1,000 person-years) in TST-positive patients.[12] The median follow-up in these nine studies was four years (IQR 2-6 years). A subsequent review paper by Pai et al extended the meta-analysis of Rangaka et al to include five additional papers.[10] Tuberculosis incidence rates varied between 3.7-84.5 cases per 1,000 person-years in IGRA-positive patients and between 2.0-32.0 cases per 1,000 person-years in IGRA-negative patients.

More recently, as part of the development of LTBI guidelines,[13] the World Health Organization (WHO) undertook a novel systematic review and meta-analysis of individual risk of progression to active TB following LTBI diagnosis with either TST or IGRA. The primary effect measure was the risk ratio (TB incidence in those with positive tests versus negative tests, in those who did not receive chemopreventative therapy). The overall pooled risk ratio (from 29 studies) for TST was 2.64 (95% CI 2.04-3.43) and 8.45 (95% CI 4.13-17.31) for IGRA. In a subgroup analysis including only studies where TST and IGRA were compared head-to-head (8 studies), the risk ratio for TST was 2.58 (95% CI 1.72-3.88) and IGRA was 4.94 (95% CI 1.79-13.65). While the overall risk ratio was significantly higher for IGRA, the difference was not statistically significant when limited to head-to-head comparison studies, and accordingly the WHO guidelines recommend that either TST or IGRA is appropriate for contact investigation.

These four important meta-analyses show that the ability of IGRAs to predict future active TB disease is poor but marginally better than TST (probably due to the improved specificity of IGRAs). Better biomarkers and an improved understanding of the spectrum of immune reactions that portends progression to active TB disease are required for targeting LTBI treatment programs.[10]

Some countries, such as the United States (US) and Japan, have been "early adopters" of IGRAs.[4,14] Other national guidelines have been updated to recommend both IGRAs and TST as acceptable tests for LTBI based on the accumulating evidence described above. For example, the European Centre for Disease Prevention and Control (ECDC) supervised a meta-analysis[11] and had an expert scientific committee review other literature.[15] They suggest that "IGRAs may be used as part of an overall risk assessment to identify individuals for preventive treatment", and provide detailed advice for specific patient groups and settings. Similarly, the Canadian Tuberculosis Standards were revised in 2014 based on a review of meta-analyses and other literature.[6] The revised Canadian guideline states, "Both the TST and IGRA are acceptable alternatives for LTBI diagnosis. Either test can be used for LTBI screening in any of the situations where testing is indicated…" with some exceptions listed.

With the increasing use of IGRAs, problems with test reproducibility have been recognised. Test variability of the QIFN assays has been studied more thoroughly than for the T-SPOT.*TB* test. QIFN results may vary due to pre-analytic factors (including faulty kit manufacturing, kit transport temperatures, blood volume inoculation, tube shaking, delayed tube incubation) and analytic factors (e.g. pipetting errors).[10,16] A systematic review found that under ideal conditions (i.e. repeat testing of an aliquot of the same sample) the QIFN interferon (IFN)- y result could vary ± 0.26 IU/ml (95% CI, 0.23-0.29) if the initial test result fell between 0.25-0.8 IU/ml (with the manufacturer's recommended cut-off being 0.35 IU/ml).[16] If the QIFN test was repeated 4 weeks later (introducing more variation in specimen collection, transport and processing), 95% of the repeat test results would fall within ± 0.70 IU/ml (95% CI, 0.66-0.75) if the initial test result was between 0.25-0.8 IU/ml. The same systematic review highlighted that the blood volume inoculated into the QIFN tubes (range 0.8-1.2 ml) and delay before tube incubation were the major causes of QIFN variability.[16] Clinicians interpreting IGRA results must consider the variability of IGRAs, particularly for the serial testing of HCWs.

Diagnosis of active tuberculosis in adults

The previous NTAC statement in 2012 recommended against the use of IGRAs for the diagnosis of active TB disease in adults citing a meta-analysis by Metcalfe et al.[3,17] For diagnosing active TB disease, this review found that IGRAs had a pooled sensitivity of 69 - 83% in HIV non-infected subjects and 60 - 76% in HIV co-infected patients (i.e. equivalent to prior results for TST).[17] Also, like TST, IGRAs cannot distinguish between LTBI, active TB or past infection. Hence specificity for active TB was low: 52 - 61% in HIV non-infected and 50 - 52% in HIV infected subjects. Further meta-analyses on the use of IGRAs to diagnose extrapulmonary TB,[18] and in immunocompetent and immunosuppressed patients using IGRAs to test blood and other body fluids (e.g. pleural fluid),[19] have reached similar conclusions. The limited sensitivity and specificity of IGRAs means that these tests cannot be used to rule-in or rule-out active TB disease in adults, and have no place in the investigation of active TB disease in adults. Sadly, anecdotal experience amongst TB physicians and limited published data suggest that IGRAs are (mis)used for this purpose in Australia.[20]

Recommendation unchanged

TST and IGRAs have no place in the initial investigation of active TB disease in adults.

IGRA (like TST) cannot and should not be used to exclude suspected TB disease in adults.

Contact investigation in adults

Contact tracing and identification of LTBI following an exposure to active, infectious TB is an important component of TB control, particularly in low-TB incidence settings.[1,21] Various studies have provided different estimations for the progression rate to active disease two years after TST/IGRA conversion but the overall lifetime risk is generally described as 10%–15%. Treatment of LTBI with isoniazid reduces risk of future disease by 75%–90%.[22] Early identification of infected contacts and appropriate preventive treatment therefore has the potential to minimise future incident cases and ongoing transmission of infection. Amongst key limitations for effective contact investigation is the lack of a gold standard test that can identify LTBI, differentiate between active and latent infection, or predict patients at highest risk of progressing to active disease. Both TST and IGRAs detect a cellular immune response to *M. tuberculosis* antigens as an imperfect surrogate marker for LTBI. Specificity of IGRA for diagnosis of LTBI is higher than TST, particularly in the setting of previous BCG vaccination.[23]

Experience with use of IGRA in programmatic contact tracing has expanded since the 2012 NTAC IGRA recommendations. Some jurisdictions, particularly in the US, have phased out the use of TST in favour of IGRA.[24] No Australian jurisdiction has replaced TST with IGRA, however experience with local use in contact tracing has been reported in at least one state TB program.[25] In this study, the negative predictive value for subsequent development of active TB was 99.5%.

The four meta-analyses of longitudinal studies summarised in the above section show that both TST and IGRA can (poorly) stratify risk of active TB following exposure in TB contacts.[10-13] While a number of studies have suggested a higher risk of progression to active TB after positive IGRA, this difference is not significant in meta-analysis of head-to-head studies to date.[13] Accordingly, either TST or IGRA may be used for investigation of contacts of active TB. In some populations, particularly those contacts with a history of BCG vaccination, the improved specificity of IGRA may allow better targeting of preventative therapy. The specificity of TST is minimally affected by BCG immunisation administered before the age of one year, especially if immunisation occurred ≥ 10 years ago.[26] However, TST specificity is adversely affected if immunisation occurs after infancy or if BCG is repeatedly administered.[26]

Revised recommendations

Either TST or IGRA can be used in adults exposed to patients with active TB disease (i.e. in contact tracing). IGRA may be preferred in contacts with a history of multiple BCG immunisations, or immunisation with BCG after the age of one year.

Diagnosis of active tuberculosis in children

The 2012 NTAC position recommended that IGRAs (like TST) should only be as an adjunctive test to standard microbiological and radiological investigations in the investigation of active TB disease in children, and that IGRAs (like TST) cannot and should not be used to exclude suspected TB disease in children.

Studies of children with bacteriologically confirmed tuberculosis, including studies in low TB endemic settings, suggest a similar sensitivity of IGRA and TST.[27-31] A recent systematic review and meta-analysis reported that in children with microbiologically confirmed TB, sensitivity of TST, QIFN-Gold In Tube and T-SPOT. TB was 79%, 81% and 81% respectively with similar findings when stratified to low income countries (74%, 66% and 80% respectively) and high income countries (86%, 86% and 79% respectively).[30] It was concluded that IGRAs did not perform better than TST.

IGRAs (like TST) cannot and should not be used to exclude TB disease. Given the difficulty of establishing an accurate diagnosis of active TB in children, an IGRA (and/or TST) may provide additional evidence of *M. tuberculosis* infection in a child with suspected TB. A positive IGRA or TST result does not, however, discriminate between TB disease and LTBI. Neither test should be used as a replacement for standard microbiological and radiological investigations.

Recommendation unchanged

In the diagnosis of active TB in children, IGRAs (like TST) should only be used as an adjunctive test in addition to standard microbiological and radiological investigations.

IGRA (like TST) cannot and should not be used to exclude suspected active TB disease in children.

Diagnosis of latent tuberculosis infection in children

Detection of LTBI is undertaken in children at risk for active TB for whom preventive therapy is indicated. These include recent contacts of active cases and migrants from high TB incidence settings. The 2012 NTAC position statement recommended that IGRA does not replace TST for detection of LTBI in children and (like TST) cannot be used to exclude LTBI.[3] It was noted that IGRA may have additional value over TST in children that received BCG vaccination after the first year of life.

IGRAs (like TST) can be used to diagnose LTBI but a negative IGRA or a negative TST does not exclude LTBI. A large number of studies have compared the performance of IGRAs with TST as a marker of LTBI in children.[30,32] The absence of a recognised gold standard makes it difficult to estimate the 'true' sensitivity and specificity of IGRA or TST for the detection of LTBI. Therefore, defined exposure to *M. tuberculosis* has become an accepted quasi 'gold standard' on which to base comparative evaluations between TST and IGRA in children.[32]

Discordance between IGRA and TST results are common in children, with TST-positive and IGRA-negative (TST+/IGRA-) being the most common discordant pattern in the low TB endemic setting.[33] This discordance may be partly due to false-negative IGRA results. It may also be partly due to false-positive TST results due to previous BCG or infection with non-tuberculous mycobacteria.[26,33] Indeterminate IGRA results are also commonly reported in young children [34](< 5 years). Further, as with TST,

the timing of the IGRA is likely to be important (e.g. may be false-negative if the exposure is very recent). Therefore, a negative IGRA should not be used to exclude LTBI in children.

In settings with low rates of BCG immunisation, such as Australia, IGRA adds little over TST in the context of TB testing or contact investigation. In BCG-immunised children (usually immigrants) IGRA may have an advantage, as TST can yield false positive results in BCG vaccinated children (especially during the first 2-5 years of life, if vaccinated at birth).[26,35] Studies of immigrant children from regions with routine BCG immunisation suggest that IGRA may be a better test than TST to guide the use of preventive therapy.[36-39] LTBI testing as part of pre-migration testing has recently been introduced for children older than 2 years immigrating to Australia and the USA.[36] It is always important to explore potential close contact with a TB source case. The infectiousness of the source case, the proximity and duration of contact, and risk of the child contact to progress to disease (greatest in young children <2-5 years of age with recent TB exposure) are the most important factors in deciding the need for preventive therapy, irrespective of the IGRA or TST result.

The Australian Immunisation Handbook recommends that all individuals (except infants < 6 months of age) should undergo a TST before BCG vaccination.[40] Only immunocompetent persons with a TST induration < 5 mm should receive BCG. The rationale for this TST is to detect individuals already infected with *M. tuberculosis* or an NTM, or who have an immediate cutaneous reaction to TST. An adverse reaction to BCG may occur in this latter group.[41] The evidence for this pre-BCG TST is limited. While there is no literature on using IGRAs for such pre-BCG testing, TST must remain the preferred test for this purpose.

Revised recommendations

IGRA and TST are acceptable options for LTBI diagnosis, but neither is 100% sensitive or specific.

IGRA may be the preferred test for testing for LTBI in children with prior BCG vaccination.

Testing of immigrants

The evolving epidemiology of TB in Australia is driven mostly by migration of individuals from countries with a high burden of disease. Following arrival in Australia, disease amongst immigrants occurs most commonly as a result of reactivation of latent TB. In 2014, overseas-born people contributed 89% of the total TB case-load.[42] The TB incidence rate in the overseas-born population was 19.1 cases per 100,000 population. This rate is more than 17 times the incidence rate experienced in the Australian born population.

Post-arrival testing and treatment of LTBI in newly-arrived refugees has been shown to be a cost-effective measure, due to the prevention of TB transmission in the community and number of cases and deaths from TB averted.[43] Among countries that test for LTBI, there is heterogeneity in which immigrant subgroups are tested. A survey of 31 member countries of the Organisation for Economic Cooperation and Development (OECD) found 16 (55.2%) of 29 respondent countries tested for LTBI; the TB incidence threshold from country of origin for testing ranged from >20 cases per 100,000 to >500 cases per 100,000.[44] This wide variation likely reflects uncertainty about the optimal threshold at which to test. Setting the incidence threshold too low results in large numbers of immigrants needing to be tested, increasing costs and potentially overwhelming TB testing services. The most cost-effective policy option is likely to be to target at an intermediate incidence that balances the numbers of immigrants being tested against prevalence of LTBI in the immigrant population.

In 2014, Australia introduced LTBI testing of children in immigration detention facilities as well as offshore testing for migrants aged 2-10 years. More extensive LTBI testing of migrants to prevent disease may become an increasingly-important component of TB control within

Australia. The extent of this LTBI testing will depend on the policies, priorities and resources of the state and territory TB control services.

As outlined in previous sections, there has been increasing evidence since the 2012 NTAC position statement that IGRA is as sensitive and more specific than TST as a test for LTBI.[3] The survey of LTBI testing practices in OECD countries found that 6 (37.5%) of 16 countries used IGRA as part of their testing algorithm. Furthermore, studies from the USA indicate that IGRA-based testing is potentially more cost-effective and safer for children.[39]

Revised recommendation

If LTBI testing is performed for immigrants from high-incidence population settings after arrival, either IGRA or TST may be used.

Immunocompromised individuals with HIV infection

HIV infection significantly increases the risk that LTBI will progress to clinical disease. In TB non-endemic areas, HIV-positive patients co-infected with TB have an annual risk of 5-8% per year of progressing to active TB disease compared with a 10% lifetime risk in the general population.[45] Hence, when the risk of TB infection is high for a HIV-positive person, such as being a close household contact of an infective TB case, treatment for LTBI should be considered irrespective of the results of TST or IGRA testing. Before commencing LTBI treatment, a careful assessment to exclude active TB disease must be undertaken in all HIV-infected subjects, including culture of sputum or induced sputum because the chest X-ray appearance may be atypical or normal in the presence of culture-positive sputum.

In the absence of a history suggesting recent infection, all HIV subjects of all ages should be tested for LTBI with a view to instituting preventative treatment. Numerous studies summarised in a systemic review by Catamanchi *et al* confirm that the sensitivity of IGRA tests are reduced in HIV-infected subjects with similar findings for the TST.[46] A lower CD4 count (<200 cells/μL) was associated with more negative and indeterminate results. The same meta-analysis suggested that the T-SPOT.*TB* test had greater sensitivity than the QIFN assay in HIV subjects when using active TB disease as a surrogate for LTBI to assess IGRA sensitivity.[46] Performance of IGRAs in detecting active TB cases however may not necessarily mirror their performance in detecting latently-infected subjects. Subsequent studies have produced discordant results reporting higher positivity rates and/or higher indeterminate rates with either the T-SPOT.*TB* test or the QIFN assay.[47-49] Two review articles have summarised these disparate studies by stating that neither IGRA test has been shown to be consistently more sensitive than TST in detecting LTBI in HIV-positive patients,[46] and that IGRA tests overall perform similarly to TST.[10]

Whether IGRA tests are useful in HIV-positive patients in predicting progression from latency to active disease is not well studied. Three studies found that IGRA-positive HIV-infected subjects were about three times more likely to develop TB than IGRA-negative patients.[10] A recent French study compared the results of QIFN, T spot and TST in 415 anti-retroviral-therapy (ART)-naïve HIV infected patients and followed their clinical progress for two years.[49] Of 47 patients with one or both IGRA tests positive, eight (14.5%) developed active disease, all within 4 months of enrolment. The eight cases of TB documented included two cases with a negative TST. No patient who had a negative result with both IGRA tests developed tuberculosis in the two-year follow up period. A systematic review and meta-analysis also found that a negative QIFN test implied a very-low short-to-medium risk of active TB.[50]

WHO recommends that either IGRA or TST can be used in a low-burden high-income country such as Australia.[13] Guidelines from some countries provide caveats to such a recommendation. Recognising that the number of false-negative and indeterminate tests increase when the CD4 count is low, UK national guidelines recommend that both IGRA and TST be performed concurrently when the CD4 count is (<200 cells/

μL), while Canadian and European guidelines suggest that concurrent testing may be helpful in immunocompromised subjects including HIV-positive individuals.[6,15,51]

A recent study from Taiwan suggested that an algorithm utilising HIV viral load, CD4 count and IGRA results improves the sensitivity and negative predictive value of testing, potentially reducing the number needing chemoprophylaxis. [52]

Revised recommendations

HIV infected subjects who have close household or other close prolonged exposure to an active infective TB case should be considered for treatment for latent TB without, or irrespective of, IGRA or TST testing on the assumption that transmission was likely, the risk of disease progression high and that existing diagnostic tests are imperfect for exclusion of latent infection.

HIV infected subjects with CD4 count (>200 cells/μL)

In the absence of recent significant close TB contact, all HIV infected subjects should be tested for LTBI. Where CD4 count is (>200 cells/μL), either TST or IGRA can be used.

HIV infected subjects with CD4 counts ≤200 cells/μL

All HIV infected subject presenting with advanced immuno-suppression (CD4 counts ≤200 cells/μL) should be assessed for active TB utilising chest X-ray and sputum examination (and other cultures depending on clinical findings).

Where there is no evidence of active disease and CD4 is less than 200 cells/μL, latent tuberculosis should be considered and both tests should be performed if the first test is negative or indeterminate.

Although the specificity of TST is lower than IGRA when there is a history of BCG vaccination, either a positive TST (>=5) or IGRA should be considered an indication for preventative therapy in the setting of HIV infection, regardless of BCG history.

In the absence of either test being positive prior to commencement of ART, subsequent testing should be considered following restoration of immune function.

In keeping with recommendations on serial LTBI testing (see below), in HIV subjects where repeat exposure to TB is likely, TST may be subject to less conversions/reversions than IGRA tests and is the preferred investigation for repeated evaluation.

Immunocompromised individuals receiving anti-tumour necrosis factor-α therapy

Patients with immune-mediated inflammatory diseases (IMID) - such as rheumatoid arthritis, ankylosing spondylitis, psoriatic arthritis, ulcerative colitis and Crohn's disease – are at increased risk of developing active TB disease due to their traditional immunosuppressive therapy (e.g. prednisolone) and particularly when receiving the newer immunomodulatory biological agents, such as tumour necrosis factor-α (TNF- α) inhibitors.[53] Jick et al reported that "low-dose" (< 15 mg/day) and "high dose" (≥15 mg/day) prednisolone was associated with active tuberculosis with odds ratios of 2.8 (95% CI 1.0–7.9) and 7.7 (95% CI 2.8–21.4), respectively.[54] Five TNF- α inhibitors are available in Australia: infliximab, adalimumab, etanercept, certolizumab and golimumab. The TNF- α inhibitors have been associated with 4-20 fold increases in active TB disease with infliximab and adalimumab carrying a greater TB risk than etanercept.[53] TB risk in those taking TNF- α inhibitors is higher if they are also on corticosteroids, methotrexate or azathioprine.[55,56]

The "standard of care" is therefore to test for LTBI before beginning treatment with TNF- α inhibitors.[55] LTBI testing in IMID patients is problematic because they are often already on prednisolone therapy (which can confound LTBI testing) and controversy surrounds the choice of

test (i.e. TST or IGRA). Smith et al summarised 14 studies comparing TST and IGRAs in a total of 1,630 patients with a variety of IMIDs.[57] The lack of a "gold standard" for LTBI again confounded these studies, which therefore relied upon correlating TST and IGRA results; five publications also studied the association of test results with TB risk factors by multivariate analysis. The summary of these 14 studies was that IGRAs could not be demonstrated to be superior to TST for LTBI testing in IMID patients.[57] Higher-level evidence of the efficacy of IGRAs in IMIDs is also lacking (such as a formal meta-analysis or longitudinal studies of the risk of active TB in IGRA-positive and -negative patients).

Several societies and organisations in high-income countries with a low incidence of TB have published guidelines for LTBI testing in IMID patients.[53,57] These guidelines generally recommend TST and/or IGRA. Emphasis is also placed upon the importance of an extensive clinical history looking for TB risk factors (eg. exposure to a TB patient; residence in a TB-endemic country; working or living in congregate settings such as hospitals, jails or homeless shelters) and on a chest X-ray (looking for fibronodular opacities suggestive of inactive TB). For example, the Australian Rheumatology Association recommends a case history risk assessment, chest x-ray within last three months, and either two step TST skin test or IGRA.[55]

Recommendation unchanged

Either TST or IGRA are acceptable for LTBI testing in IMID patients. IGRA may be preferred if there is a history of BCG immunisation after age one year. Both TST and IGRA may be performed if the risk of LTBI is considered high; a diagnosis of LTBI would be made by a positive result in either test.

The TB exposure history and chest X-ray are central in interpreting the TST/IGRA result and in determining the overall risk of LTBI in IMID patients.

Other immunocompromised individuals

Other immunocompromised populations (eg. pre-organ transplantation, patients with end-stage renal failure on dialysis) are also at increased risk of TB reactivation. For example, the incidence of post-transplant TB is 1.2%-6.4% in non-endemic countries, which is 20-74 fold higher than the general population.[58] Testing for LTBI is therefore indicated in these groups. Unfortunately, published comparisons of IGRAs and TST in these populations are limited and there is a high rate of indeterminate IGRA results in these groups.[58-60] There is also a lack of higher-level evidence of the efficacy of IGRAs in these "other immunocompromised patient groups". Hence, NTAC makes the same recommendations for LTBI testing in these "other immunocompromised" individuals as for IMID patients pre- anti-tumour necrosis factor-α therapy.

Recommendation unchanged

Either TST or IGRA are acceptable for LTBI testing in other immunocompromised patients. IGRA may be preferred if there is a history of BCG immunisation after age one year. Both TST and IGRA may be performed if the risk of LTBI is considered high; a diagnosis of LTBI would be made by a positive result in either test.

The TB exposure history and chest X-ray are central in interpreting the TST/IGRA result and in determining the overall risk of LTBI in immunocompromised patients.

Serial testing of healthcare workers

These new NTAC guidelines provide an overall recommendation that either TST or IGRA may be used for the detection of LTBI in most settings. The regular (annual) serial testing of HCWs is one situation where the choice of investigation remains controversial.[14] While IGRAs have advantages including convenience and specificity, high rates of conversions and reversions have been reported leading to more-costly follow-up of test-positive subjects.[10,61]

These conversions and reversions tend to occur more frequently when the initial QIFN result is close to the cut-off (0.35 IU/ml).[61,62] The manufacturer does not recommend a "grey zone" but the literature suggests that IFN-γ results of 0.25-1.0 IU/ml should be interpreted with caution.[62] The Committee therefore still prefers TST for the serial testing of HCWs. If an IGRA such as QIFN is used, NTAC recommends that the laboratory report the numeric IFN-γ result (IU/ml) as well as the "positive" or "negative" interpretation. Depending on the clinical circumstances of the HCW, the clinician may choose to repeat the IGRA test if the initial result falls within a pre-determined "grey zone".

Recommendation unchanged

The problem of defining an appropriate cut-off point has resulted in a trend towards more cautious use of IGRAs for HCW testing. For the present, TST remains the preferred test for serial HCW testing in Australia with IGRA's role limited to supplementary testing as a specificity tool.

Indeterminate results

IGRAs can produce un-interpretable (termed "indeterminate") results either due to inappropriately high or low IFN-γ response in the negative or positive controls, respectively. The rate of indeterminate results has varied between studies, between populations, and between assays.[47,61,63] Advice on the handling of indeterminate results is conflicting. Kobashi et al found that indeterminate IGRA results are more common among immunosuppressed patients, and subsequent IGRA testing one month later in this patient group is often indeterminate again.[63] Hence, when an initial IGRA result is indeterminate, a TST may be the preferred sequential test. In contrast, the Canadian guidelines recommend repeat testing of immunocompromised patients with an initial-indeterminate result.[3] There is insufficient evidence to favour an alternative IGRA test as a supplementary assay following an initial indeterminate IGRA result. Repeated indeterminate results are considered a marker of anergy. The clinician must then determine the patient's LTBI status based on TB exposure history and other results.

The handling of indeterminate results highlights an important principle. IGRAs should be performed in cooperation with clinicians experienced in the diagnosis and management of TB and LTBI. The investigation and management of such patients should occur in liaison with the relevant state or territory TB service. Problematic IGRA results, including indeterminate reactions, can then be assessed expertly in the patient's clinical setting.

Cost-effectiveness analyses

While international studies have attempted to define the performance and utility of IGRAs, NTAC notes a continuing absence of high-quality cost-effectiveness analyses (CEAs) of IGRAs internationally and more particularly under Australasian TB program conditions. Three meta-analyses of IGRA CEAs have all bemoaned the methodologic flaws and the variability in test parameters and cost estimates, and warned that any IGRA CEA results be viewed with caution.[64-66]

Both NTAC and the state-based TB services encourage further clinical and economic evaluation of IGRAs, particularly independent cost-benefit analyses on the use of IGRAs using states' and territories' preferred protocols of investigating LTBI in Australia. Such analyses are needed to determine the relative economic outcomes of changing from TST to IGRAs taking into account the structure of TB services and program delivery in Australia.

This NTAC position statement supersedes all previous NTAC IGRA recommendations. NTAC is committed to ongoing monitoring of new diagnostic tests that may be of value in TB control. This IGRA position statement will remain under ongoing review and will be revised when significant developments occur in this field.

Transparency declaration

The members of the Committee have no additional declarations beyond those made in the 2012 statement.[3]

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Annual report

Influenza epidemiology in patients admitted to sentinel Australian hospitals in 2016: the Influenza Complications Alert Network (FluCAN)

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Abstract

The Influenza Complications Alert Network (FluCAN) is a sentinel hospital-based surveillance program that operates at sites in all states and territories in Australia. This report summarises the epidemiology of hospitalisations with laboratory-confirmed influenza during the 2016 influenza season.

In this observational study, cases were defined as patients admitted to one of the sentinel hospitals with an acute respiratory illness with influenza confirmed by nucleic acid detection. Data are also collected on a frequency matched sample of influenza negative patients admitted with acute respiratory infection as a control group.

During the period 1 April to 30 October 2016 (the 2016 influenza season), there were 1,952 patients admitted with confirmed influenza to one of 17 FluCAN sentinel hospitals. Of these, 46% were elderly (≥65 years), 18% were children (<16 years), 5% were Aboriginal and Torres Strait Islander Peoples, 3% were pregnant and 76% had chronic co-morbidities. A small proportion were due to influenza B (7%). Estimated vaccine coverage was 73% in the elderly (≥65 years), 51% in non-elderly adults with medical comorbidities and 15% in children (<16 years) with medical comorbidities. The estimated vaccine effectiveness in the target population was 13% (95% confidence interval (CI): -5% to 27%).

There were a large number of hospital admissions detected with confirmed influenza in this national observational surveillance system in 2016 with case numbers similar to that reported in 2014 and 2015.

Introduction

Influenza affects up to 5-10% of the population each year[1]. Because infection with influenza virus is relatively widespread, the incidence of hospitalisation from influenza is of public health significance, although the risk of hospitalisation is low[2]. In this report, we describe the epidemiology of hospitalisation with laboratory-confirmed influenza in the 2016 season in Australia.

Methods

The Influenza Complications Alert Network (FluCAN) is a national hospital-based sentinel surveillance system[3]. Since 2011, the participating sites have been Canberra Hospital (ACT), Calvary Hospital (ACT), Westmead Hospital (NSW), John Hunter Hospital (NSW), Children's Hospital at Westmead (NSW), Alice Springs Hospital (NT), Royal Adelaide Hospital (SA), Mater Hospital (QLD), Princess Alexandra Hospital (QLD), Cairns Base Hospital (QLD), Royal Hobart Hospital (TAS), The Alfred

Hospital (VIC), Royal Melbourne Hospital (VIC), Monash Medical Centre (VIC), University Hospital Geelong (VIC), Royal Perth Hospital (WA), and Princess Margaret Hospital (WA). Ethical approval has been obtained at all participating sites and at Monash University. Hospital bed capacity statistics were obtained from each participating hospital, and national bed capacity was obtained from the last published Australian Institute of Health and Welfare report.[4]

An influenza case was defined as a patient admitted to hospital with influenza confirmed by nucleic acid testing (NAT). Surveillance is conducted from early April to end October (with follow up continuing to the end of November) each year. Admission or transfer to an intensive care unit (ICU) included patients managed in a high dependency unit (HDU). The onset date was defined as the date of admission except for patients where the date of the test was more than 7 days after admission, where the onset date was the date of the test. The presence of risk factors and comorbidities was ascertained from the patient's medical record. Restricted functional capacity was defined as those who were not fully active and not able to carry out all activities without restriction prior to the acute illness [5].

We examined factors associated with ICU admission using multivariable regression. Factors independently associated with ICU admission were determined using a logistic regression model with no variable selection process, as all factors were plausibly related to ICU admission.

Vaccine coverage was estimated from the proportion of vaccinated individuals in each age group, stratified by the presence of chronic comorbidities. Vaccine effectiveness was estimated from the odds ratio of vaccination in cases versus controls using the formula, with the odds ratio calculated from a conditional logistic regression, stratified by site and adjusted for age group, the presence of chronic comorbidities, pregnancy and Aboriginal or Torres Strait Islander ethnicity.

Results

During the period 1 April to 30 October 2016 (the 2016 influenza season), there were 1,952 patients admitted with laboratory-confirmed influenza to one of 17 FluCAN sentinel hospitals. The peak weekly number of admission was in mid-August (week 35) (Figure 1). The majority of cases were due to influenza A (93%). The proportion due to influenza B was higher in the West Australian hospitals (46/180, 26%; Princess Margaret Hospital 34/105, 32%; Royal Perth Hospital 12/75 16%) compared to all other jurisdictions (5.0%).

Of these 1,952 patients, 904 (46%) were >65 years of age, 359 (18%) were children (<16 years), 101 (5%) were Aboriginal and Torres Strait Islander peoples, and 1492 (76%) had chronic co-morbidities (table 1; table 2). There were 50 pregnant women which represented 21% of the 243 female patients aged 16-49, or 3% of the total. Of the 1,599 patients (82%) where influenza vaccination status was ascertained, 752 (47%) had been vaccinated.

Incidence of hospital admissions with influenza

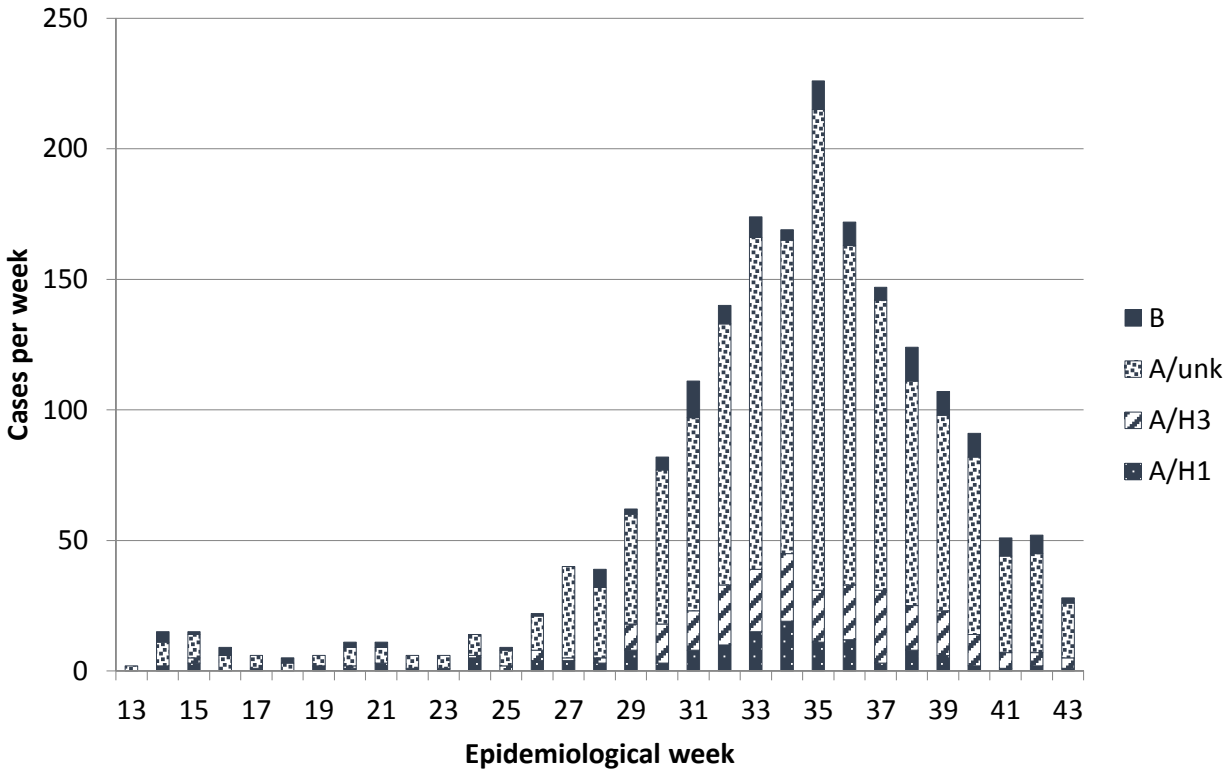
Overall, the peak incidence of admissions with confirmed influenza was 3.2 per 100 hospital beds (in epidemiological week 35), but varied from 0.54 per 100 hospital beds at Princess Alexandra Hospital (QLD) to a 9.7 per 100 hospital beds at Westmead Hospital (NSW).

Presentation and management

For 1,759 patients with laboratory-confirmed influenza where the duration of symptoms was known, the median duration of symptoms prior to admission was 2 days (interquartile range (IQR): 1, 4 days). Of all cases, 65 cases (3%) were diagnosed more than 7 days after admission and therefore were likely to be hospital-acquired. Radiological evidence of pneumonia was present in 363 patients (19%).

Of all cases, 214 patients were admitted to ICU, including 180 patients (9%) were initially admit-

Figure 1: Date of admission in patients hospitalized with confirmed influenza



By week beginning on listed date; representing date of admission (or date of influenza diagnosis if acquired >7 days in hospital)

ted to ICU and a further 34 (2%) subsequently transferred to ICU after initial admission to a general ward. The elderly (>65 years) and residents of nursing homes were less likely to be admitted to intensive care. There were no statistically significant differences in the risk of admission to ICU by influenza type in patients admitted to hospital with influenza.

Outcome

The mean length of hospital stay for all patients was 5.6 days. Admission to ICU was associated with a mean hospital length of stay of 11.1 days compared to those not admitted to ICU (4.9 days). Of the 1899 patients where hospital mortality status was documented, 65 patients died (3%), which included 25 patients in ICU. Case fatality was higher in the elderly (51/864; 6%) than in non-elderly adults and children (14/1,035; 1%). Of the 65 deaths, 62 (95%) occurred in patients with comorbidities. The case fatality of influenza-associated pneumonia was 8% (29/359).

Vaccine coverage and effectiveness

Vaccination status was ascertained in 1,599 of 1,952 cases (82%) and 1,386 of 1,715 test negative control patients (81%). Estimated vaccine coverage was 73% (467/636) in the elderly (≥65 years), 51% (212/414) in non-elderly adults with medical comorbidities and 15% (18/116) in children (<16 years) with medical comorbidities. In the target population, the crude odds ratio of vaccination in cases versus controls was 0.87 (95% confidence interval (CI): 0.73 to 1.02) and the adjusted odds ratio of vaccination was 0.87 (95% CI: 0.73 to 1.05). The estimated vaccine effectiveness in the target population was therefore 13% (95% CI: -5%to 27%). In the elderly (>65 years), there was no evidence of vaccine effectiveness (estimated VE -19%, 95% CI: -52% to 8.0%)

Discussion

In the 2016 season, we have documented more than 1,900 cases of influenza, which represents a similar number of admissions as 2014 (n=2,097)

Table 1: Demographic characteristics of hospitalized patients with confirmed influenza

	Influenza type/subtype				Total
	A/H1	A/H3	A/unknown	B	
Number of cases	139	256	1422	135	**1952**
Age group					
<16 years	32 (23%)	1 (0%)	258 (18%)	60 (44%)	**351 (18%)**
16-49 years	44 (32%)	47 (18%)	279 (20%)	27 (20%)	**397 (20%)**
50-64 years	32 (23%)	40 (16%)	211 (15%)	17 (13%)	**300 (15%)**
65-79 years	25 (18%)	78 (31%)	337 (24%)	18 (13%)	**458 (24%)**
80+ years	6 (4%)	90 (35%)	337 (24%)	13 (7%)	**446 (23%)**
Female*	67 (48%)	144 (56%)	726 (51%)	54 (40%)	**991 (51%)**
Pregnant	5 (4%)	3 (1%)	42 (3.0%)	0 (0.0%)	**50 (3%)**
Aboriginal or Torres Strait Islander peoples	6 (4%)	9 (4%)	76 (5%)	10 (7%)	**101 (5%)**
State					
ACT	22 (16%)	78 (31%)	152 (11%)	16 (12%)	**268 (14%)**
NSW	14 (10%)	14 (6%)	450 (32%)	24 (18%)	**502 (26%)**
NT	0 (0.0%)	0 (0.0%)	43 (3%)	3 (2%)	**46 (2%)**
QLD	7 (5.0%)	21 (8%)	125 (9%)	12 (9%)	**165 (9%)**
SA	0 (0.0%)	6 (2%)	165 (12%)	7 (5%)	**178 (9%)**
TAS	65 (47%)	31 (12%)	29 (2%)	4 (3%)	**129 (7%)**
VIC	14 (10%)	50 (20%)	397 (28%)	23 (17%)	**484 (25%)**
WA	17 (12%)	56 (22%)	61 (4%)	46 (34%)	**180 (10%)**

*Sex missing for 2 patients; reported as number and percentage of patients with type/subtype

Table 2: Risk factors, severity and outcomes in hospitalized adult patients with confirmed influenza

	Not admitted to ICU	Admitted to ICU	Total
Number of cases	1738	214	**1952**
Pregnancy	43 (3%)	7 (3%)	**50 (3%)**
Medical comorbidities	1316 (76%)	176 (82%)	**1492 (76%)**
Chronic respiratory illness	515 (30%)	71 (33%)	**586 (30%)**
Chronic cardiac disease	567 (33%)	70 (33%)	**637 (32%)**
Diabetes	383 (22%)	46 (22%)	**429 (22%)**
Chronic liver disease	74 (4%)	15 (7.0%)	**89 (5%)**
Chronic neurological illness	280 (16%)	31 (15%)	**311 (16%)**
Chronic renal disease	219 (13%)	28 (13%)	**247 (13%)**
Immunocompromised	260 (15%)	33 (15%)	**293 (15%)**
Malignancy	187 (11%)	25 (12%)	**212 (11%)**
Obesity	198 (11%)	33 (15%)	**231 (12%)**
Nursing home resident	152 (9%)	4 (2%)	**156 (8%)**
Received influenza vaccine	693/1431 (48%)	59/168 (35%)	**752/1599 (47%)**
Influenza type/subtype			
A/H1	126 (7%)	13 (6%)	**139 (7%)**
A/H3	231 (13%)	25 (12%)	**256 (13%)**
A/unknown	1260 (73%)	162 (76%)	**1422 (73%)**
B	121 (7.0%)	14 (7%)	**135 (7%)**
In hospital mortality	40/1692 (2%)	25/207 (12%)	**65/1899 (3%)**

and 2015 (n=2,070). Based on the bed capacity of sentinel hospitals, this is likely to represent around 14,000 admissions with confirmed influenza nationally. However, as influenza testing is not performed on all patients with acute respiratory presentations, and influenza may also trigger delayed respiratory presentations (e.g. secondary bacterial pneumonia) and non-respiratory complications (e.g. acute myocardial infarction), this should be regarded as a minimum estimate.

The 2016 year was the first season in which the use of quadrivalent vaccine (containing two influenza A and two influenza B strains) was funded under the National Immunisation Program. However, in comparison with the 2015 season, where more than half of admissions were due to influenza B and both Victorian and Yamagata lineages circulated, the incremental benefit of the quadrivalent vaccine would be expected to be minimal in 2016 as influenza B activity was low. Influenza vaccine effectiveness was noted to be low in the target population in this season in comparison to previous years (and absent in the elderly). Further work is being performed to explore this issue further. Vaccine effectiveness in the elderly has generally been found to be lower than in younger age groups, but a study from the United States found influenza vaccine to be cost-effective in the elderly over four seasons[6].

In recent seasons, there has been ongoing concern about mismatches between the A/H3N2 vaccine and circulating strains, due in part to antigenic change associated with egg adaptation as well as growing genetic diversity within circulating A/H3N2 strains, with North American data suggesting a higher vaccine effectiveness against 3C.3b than 3C.3a and 3C.2a clades [7, 8]. Additionally, work has suggested that the effectiveness of influenza vaccines against A/H1N1pdm may be poorer in a middle aged cohort born before 1980, who were exposed to 163Q A/H1N1/USSR types[9].

The peak incidence of confirmed influenza provides a measure of the impact of influenza. We chose to use acute hospital beds as a denominator because the number of admissions are not readily available in a timely manner, and bed numbers provide a "hard limit" of hospital capacity. The proportion of hospital beds occupied by patients with confirmed influenza can be estimated from the incidence and mean duration of stay – at Westmead Hospital in 2016, a peak weekly incidence of 9.7 per 100 beds roughly equates to 9% of the hospital bed capacity (9.7 admissions per 700 bed days x 5.6 days).

We found that around half of the influenza cases were unvaccinated. Our estimates of vaccine coverage are similar to that of previous years, where around 70-80% of the elderly, around 60% of non-elderly adults with comorbidities and around 20% in children with comorbidities[10-13]. Our estimates of influenza vaccine coverage in the elderly are consistent with recent estimates from a meta-analysis of vaccine coverage in the Australian elderly population collected by a variety of methods, providing reassurance about the validity of hospital controls for this purpose[14]. Additionally, we have recently compared vaccine coverage estimates from hospital, primary care and community-based systems and found them to be broadly consistent.[15]

The systematic review also found an increase in coverage associated with public funding of influenza vaccine since 1999; however, our findings reinforce the need to improve coverage particularly in younger populations with medical comorbidities where publicly funded vaccine has been available since 2010. We found that 93% of admissions with influenza in the elderly occurred in patients with medical comorbidities; this proportion was 71% in non-elderly adults and 44% in children. This suggests that even with an effective influenza vaccine, the current policy of vaccinating only younger individuals with comorbidities would not be expected to provide protection to more than half of children admitted to hospital.

There are several limitations to this surveillance system. There may be under-ascertainment of influenza due to poor quality sample collection or the lack of use of influenza laboratory tests,

despite the diagnosis of influenza having implications for infection control and antiviral use in hospitals. Delayed presentations or secondary bacterial pneumonia may be associated with false negative influenza tests as the influenza infection may be cleared by the time of presentation. Ascertainment in tropical regions is limited by sampling in the winter/dry season only.

In summary, we detected a large number of hospital admissions with laboratory-confirmed influenza in a national observational study in 2016 comparable to 2014 and 2015 but much higher than in prior years. A consistent finding over several years is that a high proportion of patients with severe influenza, and almost all deaths, occurred in patients with chronic comorbidities.

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Table 3: Factors associated with admission to intensive care in patients hospitalised with confirmed influenza

Variable	Crude OR	p	Adjusted OR*	p
Age				
<16 years	0.9 (0.6, 1.4)	0.713	1.1 (0.7, 1.8)	0.685
16-64 years	1		1	
65+ years	0.6 (0.5, 0.9)	0.007	0.7 (0.5, 0.9)	0.017
Medical comorbidities	1.5 (1.0, 2.1)	0.035	1.9 (1.3, 2.9)	0.001
Aboriginal or Torres Strait Islander peoples	1.6 (0.9, 2.7)	0.11	1.3 (0.7, 2.3)	0.369
Pregnancy	1.3 (0.6, 3.0)	0.488	1.0 (0.4, 2.4)	0.935
Restricted functional status	1.1 (0.8, 1.5)	0.426	1.0 (0.7, 1.4)	0.912
Nursing home resident	0.2 (0.1, 0.5)	0.002	0.2 (0.1, 0.6)	0.003
Influenza type/subtype				
A/H1	0.9 (0.4, 2.0)	0.778	0.9 (0.4, 1.9)	0.701
A/H3	0.9 (0.5, 1.9)	0.85	1.1 (0.5, 2.3)	0.761
B	1		1	
A/unk	1.1 (0.6, 2.0)	0.72	1.2 (0.7, 2.2)	0.561

* all variables included in multivariate model

Figure 2: Incidence of confirmed influenza (per 100 hospital beds) by week and year

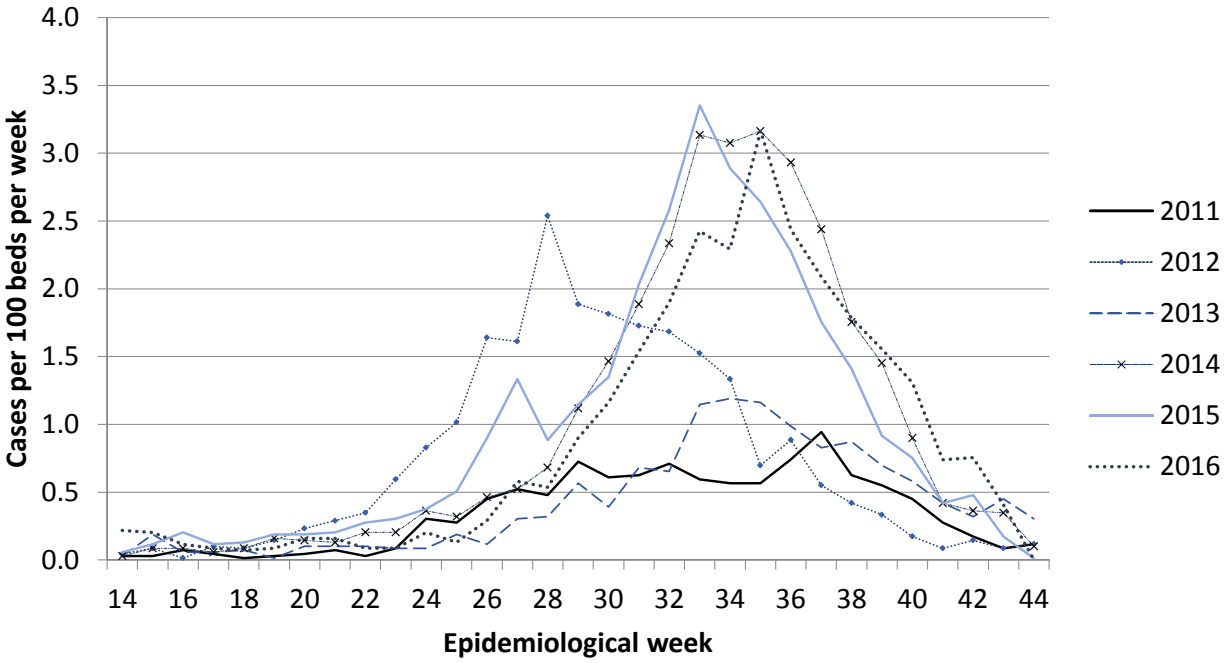
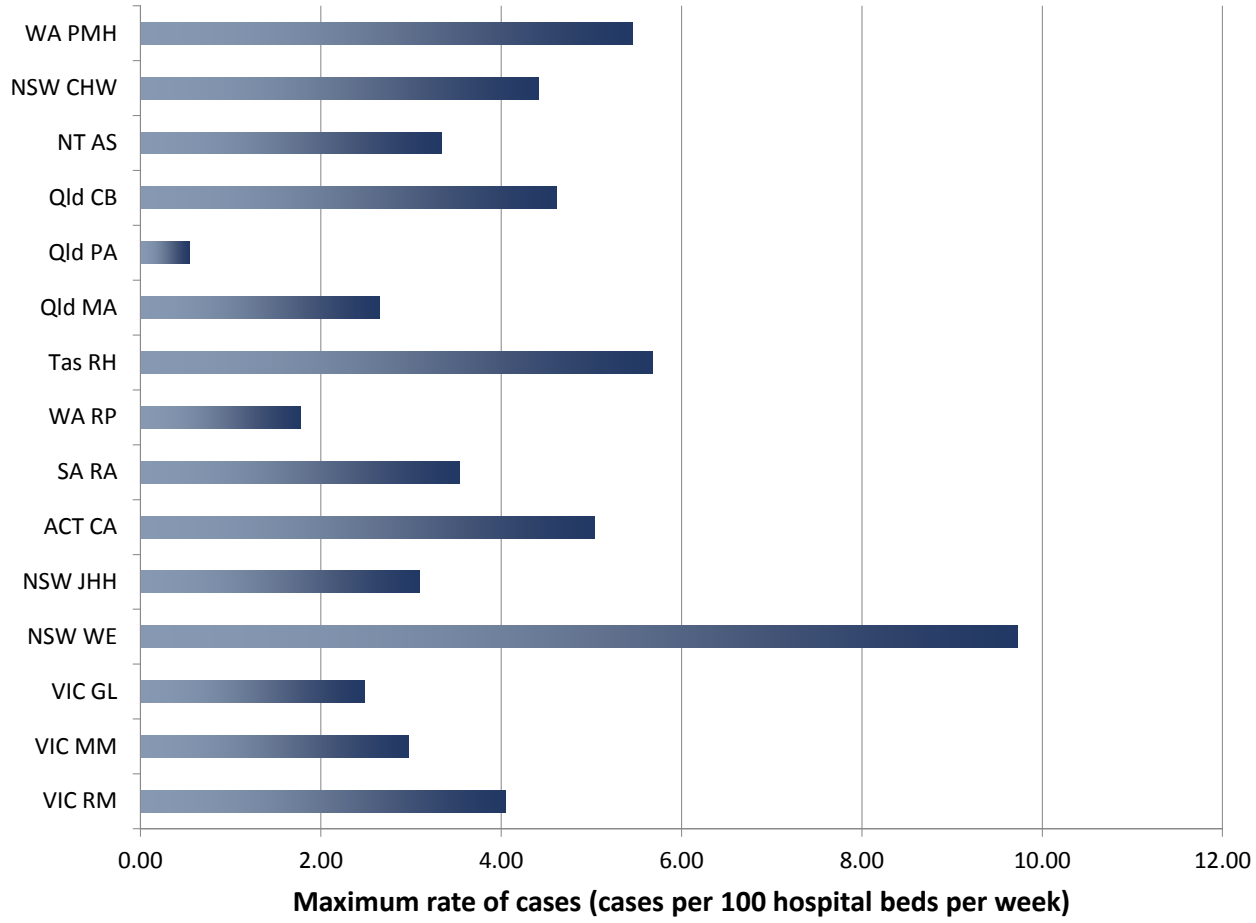


Figure 3: Peak incidence of confirmed influenza (per 100 hospital beds) by hospital



RM: Royal Melbourne; MM: Monash Medical Centre, GL: University Hospital Geelong, WE: Westmead Hospital, JHH: John Hunter Hospital, CA: Canberra and Calvary Hospitals, RA: Royal Adelaide, RP: Royal Perth Hospital, RH: Royal Hobart Hospital, MA: Mater Hospital, PA: Princess Alexandra Hospital, CB: Cairns Base Hospital, AS: Alice Springs Hospital, CHW: Children's Hospital at Westmead, PMH: Princess Margaret Hospital

Annual report

Annual report of the National Influenza Surveillance Scheme, 2010

Kellie Gavin, Rhonda Owen, Ian G Barr and the National Influenza Surveillance Committee, for the Communicable Diseases Network Australia

Abstract

The 2010 influenza season was moderate overall, with more laboratory-confirmed cases than in earlier years (with the exception of 2009). That said, self-reported influenza-like illness (ILI) was equal to or lower than 2008 and earlier years. In 2010, the number of laboratory-confirmed notifications for influenza was 0.8 times the 5-year mean. High notification rates were reflected in an increase in presentations with ILI to sentinel general practices and emergency departments. Notification rates were highest in the 0–4 year age group. Infections during the season were predominantly due to influenza A(H1N1)pdm09, with 90% of notifications being influenza A (56% A(H1N)1pdm09, 30% A(unsubtyped) and 4% A(H3N2)) and 10% being influenza B. The A(H1), A(H3) and B influenza viruses circulating during the 2010 season were antigenically similar to the respective 2010 vaccine strains. Almost all (99%) of the circulating influenza B viruses that were analysed were from the B/Victoria lineage.

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

Introduction

Influenza or 'the flu' is a common, highly infectious respiratory viral disease. The virus spreads from person to person by airborne droplets of exhaled respiratory secretions, commonly generated by coughing or sneezing.[1] Typical symptoms include sudden onset of fever, sore throat, runny nose, cough, fatigue, headache, and aches and pains.

Influenza causes annual epidemics of respiratory disease. Influenza epidemics usually occur during the winter months in temperate climates, causing an increase in hospitalisations for pneumonia, an exacerbation of chronic diseases and also contributing to increased mortality. Those most susceptible include the elderly and very young people, or people of any age who have a higher risk of complications (e.g. pneumonia, heart failure) due to certain chronic medical conditions, e.g. heart, lung, kidney, liver, immune, or metabolic diseases. Healthy children and adults usually only display minor symptoms.

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

In temperate zones of Australia, the annual influenza season runs from May to October, with notifications generally peaking in mid-August. Influenza activity varies from year to year. Australia experienced a mild season in 2006, moderate seasons in 2007 and 2008 and an extra-ordinary season in 2009 due to the influenza A(H1N1) pandemic. In years prior to 2010 (with the exception of 2008), influenza A has been the predominant type circulating in Australia. The A(H1) subtype has been the most commonly reported since the start of the 2009 (H1N1) pandemic and the A(H3) subtype the most commonly reported prior to 2009.

Surveillance methods

- Data used to describe the 2010 influenza

season were classified under the areas of epidemiology, morbidity, mortality and virology. Influenza surveillance was based on the following sources of data:

- notifications of laboratory-confirmed influenza required by legislation in all states and territories, and notified to the NNDSS;
- subtype and strain data of circulating influenza viruses provided by the World Health Organization (WHO) Collaborating Centre for Reference and Research on Influenza;
- consultation rates for influenza-like illness (ILI) identified by sentinel general practitioners (GPs);
- consultation rates for ILI identified by hospital emergency departments (EDs);
- rates of ILI and absence from work from a community survey;
- hospitalised cases of influenza from 15 sentinel hospitals across Australia through the Influenza Complications Alert Network (FluCAN);
- testing rates for influenza by sentinel laboratories in New South Wales, Victoria, Western Australia and Tasmania; and
- mortality data from the New South Wales Registry of Births, Deaths and Marriages (BDM) and Australian Bureau of Statistics (ABS).

National Notifiable Diseases Surveillance System

In 2010, laboratory-confirmed influenza was a notifiable disease under state and territory legislation in all jurisdictions. Laboratory notifications were sent to NNDSS for national collation. In this report, data were analysed by the date of diagnosis; the best substitute for the date of onset. The date of diagnosis was set as the earliest of the dates of onset, specimen collection or notification. Age, sex, Indigenous status, method of laboratory diagnosis and postcode or locality of patient residence were included in NNDSS notifications.

FluTracking

FluTracking is a project of the University of Newcastle, the Hunter New England Local Health District, the Hunter Medical Research Institute and the Australian Government Department of Health. FluTracking is an online health surveillance system established to detect epidemics of influenza and monitor the transmission and clinical severity of ILI across Australia. It involves participants from around Australia completing a simple online weekly survey, which collects data on the rate of ILI symptoms in communities.

National Health Call Centre Network

The National Health Call Centre Network (NHCCN) is a national initiative that provides information on the number and proportion of calls received by the NHCCN relating to ILI or influenza. Data are reported daily for all jurisdictions, with the exception of Queensland and Victoria.

Sentinel general practitioner surveillance

Sentinel GP surveillance schemes for influenza monitor clinical consultations for ILI. In Australia, there are two such schemes: the Australian Sentinel Practices Research Network (ASPREN), which collects infectious disease data including ILI, at a national level from approximately 100 GPs across all states and territories and the Victorian Infectious Diseases Reference Laboratory General Practice Sentinel Surveillance Program (VIDRL GPSS). The Northern Territory Tropical Influenza Surveillance Scheme, which previously reported GP ILI rates separately, joined ASPREN in March 2010. ASPREN reports ILI rates throughout the year, while the reporting period for VIDRL GPSS was from early May to late October in 2010. The national case definition of ILI is:

presentation with fever, cough and fatigue. Both sentinel surveillance schemes used the national case definition for ILI in 2010.

Emergency department surveillance

Rates for ILI presentation were collected from 56 EDs across New South Wales and eight EDs in Perth, Western Australia. Data were provided to the Office of Health Protection within the Australian Government Department of Health* (Health) on a weekly basis, through the *Weekly Influenza Report, NSW*, and the West Australian *Virus WAtch* report.

Laboratory surveillance

WHO Collaborating Centre for Reference and Research on Influenza

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

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

Sentinel laboratory networks

Laboratory testing data are collected by PathWest (Western Australia), VIDRL (Victoria), the Institute for Clinical Pathology and Medical Research, Westmead Hospital (New South Wales) and sentinel Tasmanian laboratories and reported weekly during the influenza season.

Mortality

Death certificate data from the New South Wales Registry of Births, Deaths and Marriages provided an estimate of the number of deaths from pneumonia and influenza in New South Wales and was expressed as a rate per 1,000 deaths from all-causes and compared to a predicted seasonal mean with a 95% confidence interval alert level. These were obtained weekly from the New South Wales Influenza Surveillance Report.[2]

Deaths data compiled by the ABS from information provided by the state and territory Registrars of Births, Deaths and Marriages, and coded using the 10th revision of the *International Classification of Diseases and Related Health Problems* (ICD-10) were used to estimate levels of influenza deaths. In this report, deaths for 2010 with an underlying cause of influenza and pneumonia (ICD-10 J09–J18) are presented.[3] ICD-10 code J09 was introduced in July 2009. The expanded range of codes, J09-J18, correlates with previous versions of ICD-10 codes J10-J11.

* Known at the time as Department of Health and Ageing

Morbidity data

There was no direct measure of morbidity of disease readily available during the 2010 influenza season. Instead, morbidity was assessed through a number of indicators including:

- Paediatric admissions to Intensive Care Units (ICUs) and deaths data collected by the Australian Paediatrics Surveillance Unit (APSU);
- Hospitalised cases of influenza and pneumonia from 15 sentinel hospitals across Australia through the Influenza Complications Alert Network (FluCAN);
- ED presentations for ILI in New South Wales, Western Australia and the Northern Territory; and
- ILI presentations to GP surveillance networks.

Notification rates for laboratory-confirmed influenza were calculated using the estimated 2010 December resident population supplied by the ABS.[4] All rates are represented as the rate per 100,000 population unless stated otherwise.

Results

The 2010 influenza season began in late-July, although there was a very gradual increase in notifications above non-seasonal levels from much earlier in the year. All sentinel data sources were tracking below or similar to trends seen in previous years during the main season. Between September and the end of the year, NNDSS notifications were above the 5-year mean due to notifications peaking later than recent years and unusually high activity during December.

Laboratory-confirmed cases

The first increase in notifications of laboratory-confirmed influenza in the 2010 season were registered in late June (week 26) with 99 cases diagnosed. Notifications peaked late September (week 39) and were almost back to inter-seasonal levels by the middle of November (week 47) (Figure 1). However higher than usual levels of influenza activity across all jurisdictions characterised the final weeks of 2010. The total number of notifications for the year was 13,467, which was 0.8 times the 5-year mean. This decrease was entirely due to the significantly higher number of notifications during the 2009 pandemic (n=59,023) compared with the previous four years.

Geographic spread

In 2010, 32% of laboratory-confirmed influenza notifications occurred in South Australia, 24% in Queensland, 15% in Victoria, 12% in Western Australia and New South Wales, 4% in the Northern Territory and 1% in Tasmania and the Australian Capital Territory combined (Figure 2, Table 1). The number of notifications peaked earlier in Victoria, the Australian Capital Territory and Queensland (weeks 36-37, ending 3 and 10 September respectively), New South Wales plateaued at 101 notifications for weeks 37 through to 39, while the remaining jurisdictions peaked in week 39 (in the week ending 24 September).

Laboratory-confirmed influenza rates of notification for 2010 varied across the country, ranging from 21 cases per 100,000 population in Tasmania to 259 cases per 100,000 population in South Australia. The rate of notification of influenza infection for Australia was 60 cases per 100,000 population (Table 1).

Age-sex profile

Age-specific notification rates for laboratory-confirmed influenza reported to the NNDSS in 2010 are shown in Figure 3. The highest notification rates were seen in children aged 0–4 years, which were around 1.8 times higher than the overall notification rate (111 cases per 100,000 population compared with a total rate of 60 cases per 100,000 population for all notifications). People aged 65 years or over are the target for influenza vaccination as they are at an increased risk of complications from influenza. Notification rates for people in this age group were 35 cases per 100,000 population for males

Figure 1: Laboratory-confirmed influenza notifications, 2006 to 2010, Australia, by month and year of diagnosis

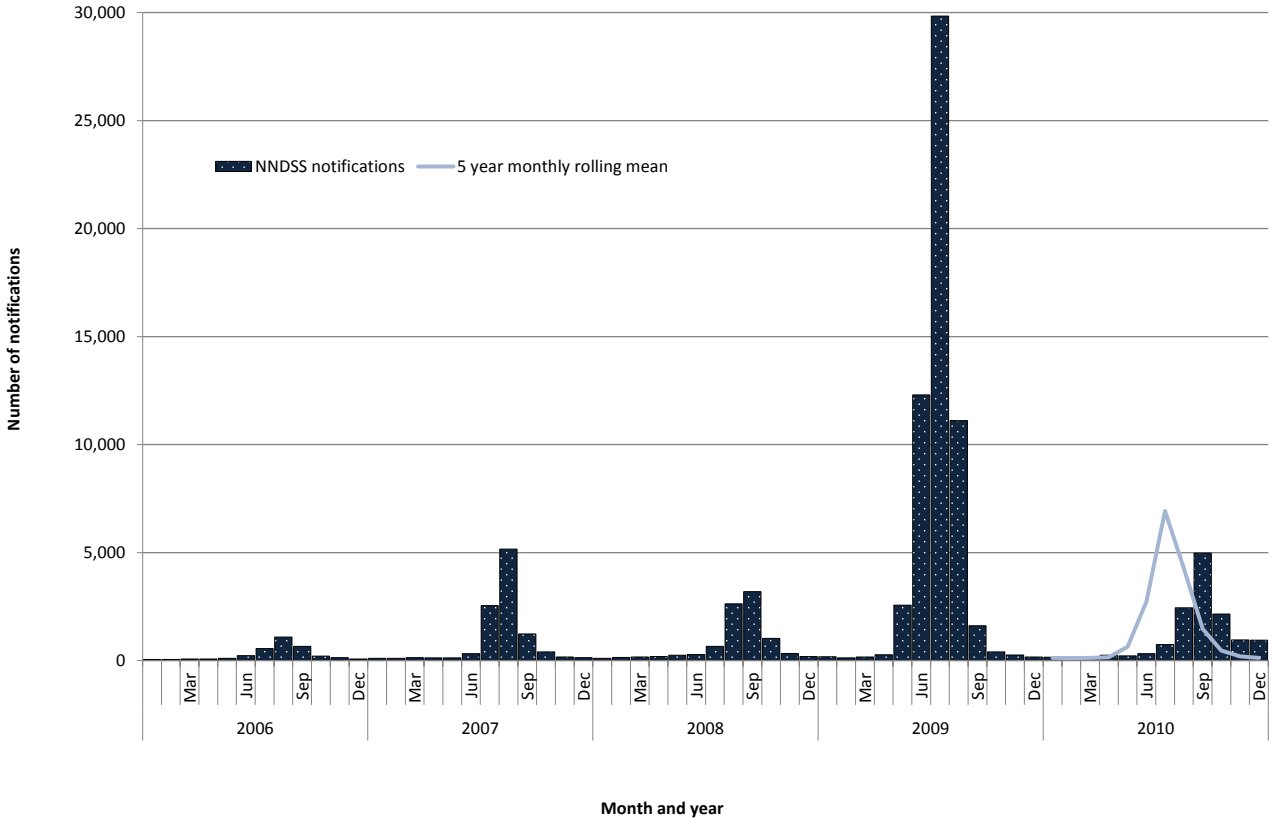


Table 1: Notifications and rates of laboratory-confirmed influenza, 2010, by state or territory and sex*

State or territory	Total notifications	% of total notifications	Notification rate (per 100,000 population)	Notifications*		Notification rate* (per 100,000 population)	
				Male	Female	Male	Female
ACT	95	1%	26	47	48	26	27
NSW	1,604	12%	22	733	847	20	23
NT	479	4%	209	248	231	208	209
Qld	3,221	24%	71	1,472	1,748	65	78
SA	4,258	32%	259	1,990	2,268	245	273
Tas.	104	1%	21	55	49	22	19
Vic.	2,081	15%	38	1,003	1,039	37	37
WA	1,625	12%	71	777	848	67	75
Australia	**13,467**	**100%**	**60**	**6,325**	**7,078**	**57**	**63**

* Excludes 64 notifications for which sex was not stated.

Figure 2: Laboratory-confirmed influenza notifications, 29 May June to 31 December 2010, by state or territory and week of diagnosis

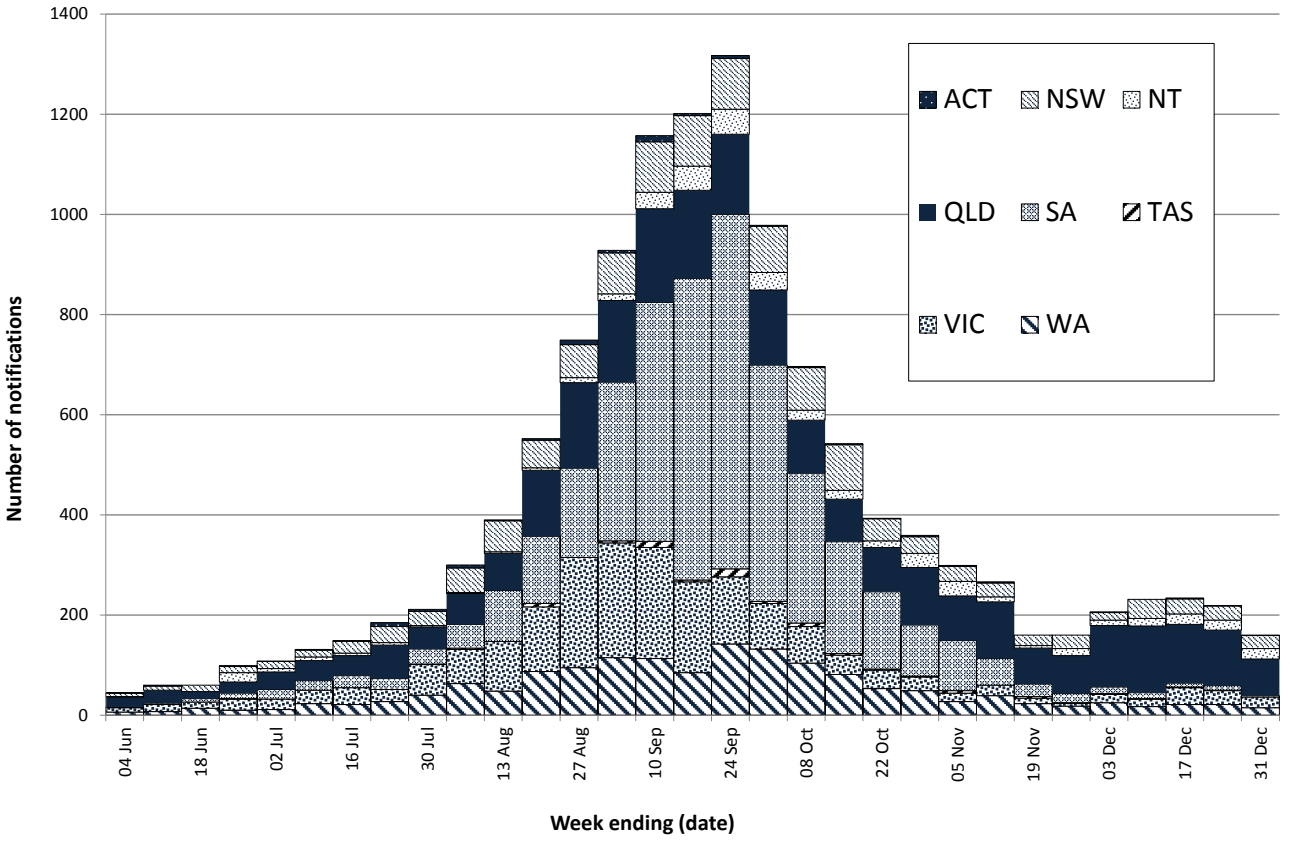
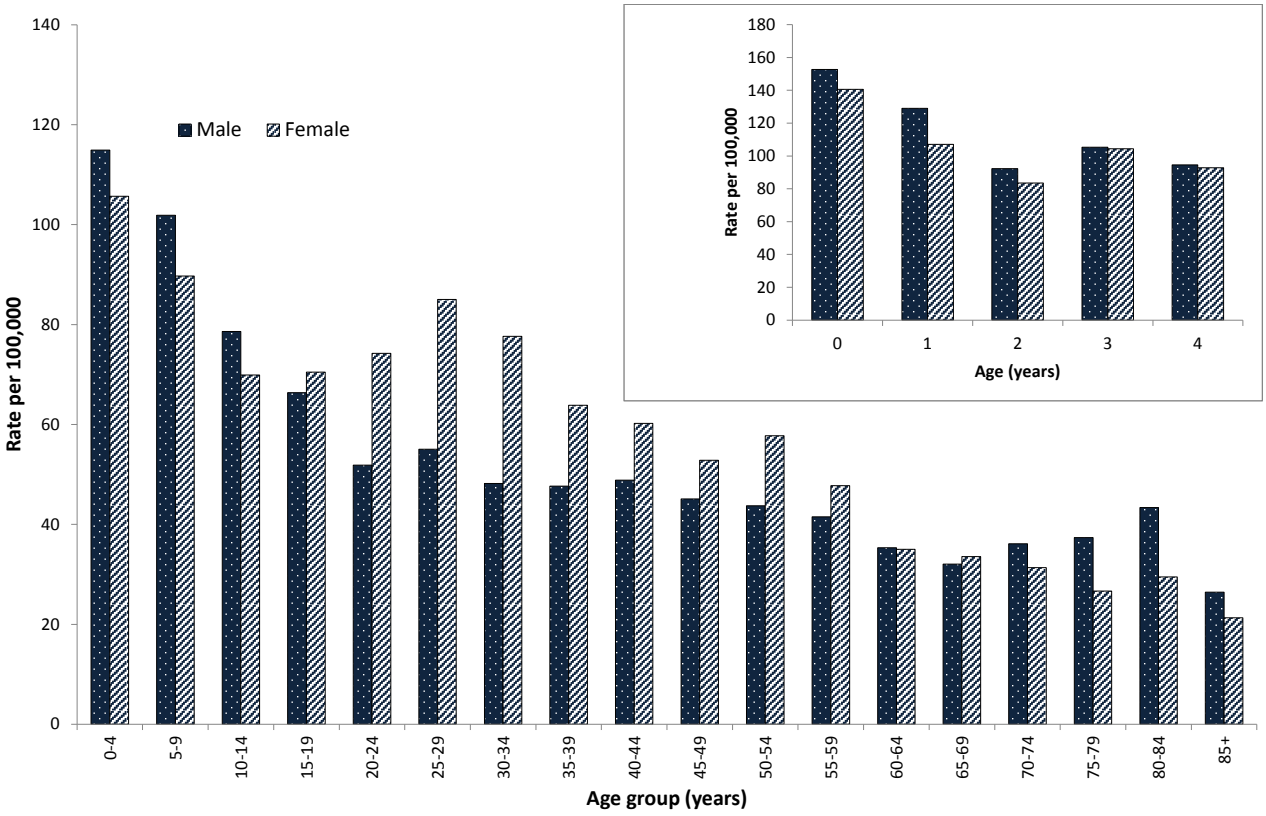


Table 2: Summary of Australian influenza viruses collected in 2010 and typed by HI or PCR at the WHO Collaborating Centre for Reference and Research on Influenza, VIDRL, by antigenic type

Type/ subtype	ACT	NSW	NT	Qld	SA	Tas.	WA	Vic.	Total
A(H1) pandemic 2009	12	122	139	335	140	1	208	249	1,206
A(H3)	3	11	1	117	3	3	30	27	195
A(NS)	0	0	2	0	0	0	0	1	3
B(VIC)	0	30	2	47	0	0	147	10	236
B(YAM)	0	0	0	0	0	0	2	0	2
Mixed A	0	0	0	1	1	0	0	2	4
Mixed A/B	0	0	0	0	0	0	0	2	2
Total	15	163	144	500	144	4	387	291	1,648

Note: If year of sample collection was unknown it was assumed based on the year it was received at the Centre or the year stated in the virus' designation. Some samples collected in 2010 may not have been received at the Centre until 2011.

Figure 3: Notification rate of laboratory-confirmed influenza, Australia, 2010, by age group (insert – single year of age) and sex*



* Notifications (n=97) with unknown age and/or sex were excluded from analysis.

and 29 cases per 100,000 population for females. This compares with 2009 pandemic year where influenza rates were higher in this age group for males (72 cases per 100,000 population) than females (64 cases per 100,000 population).

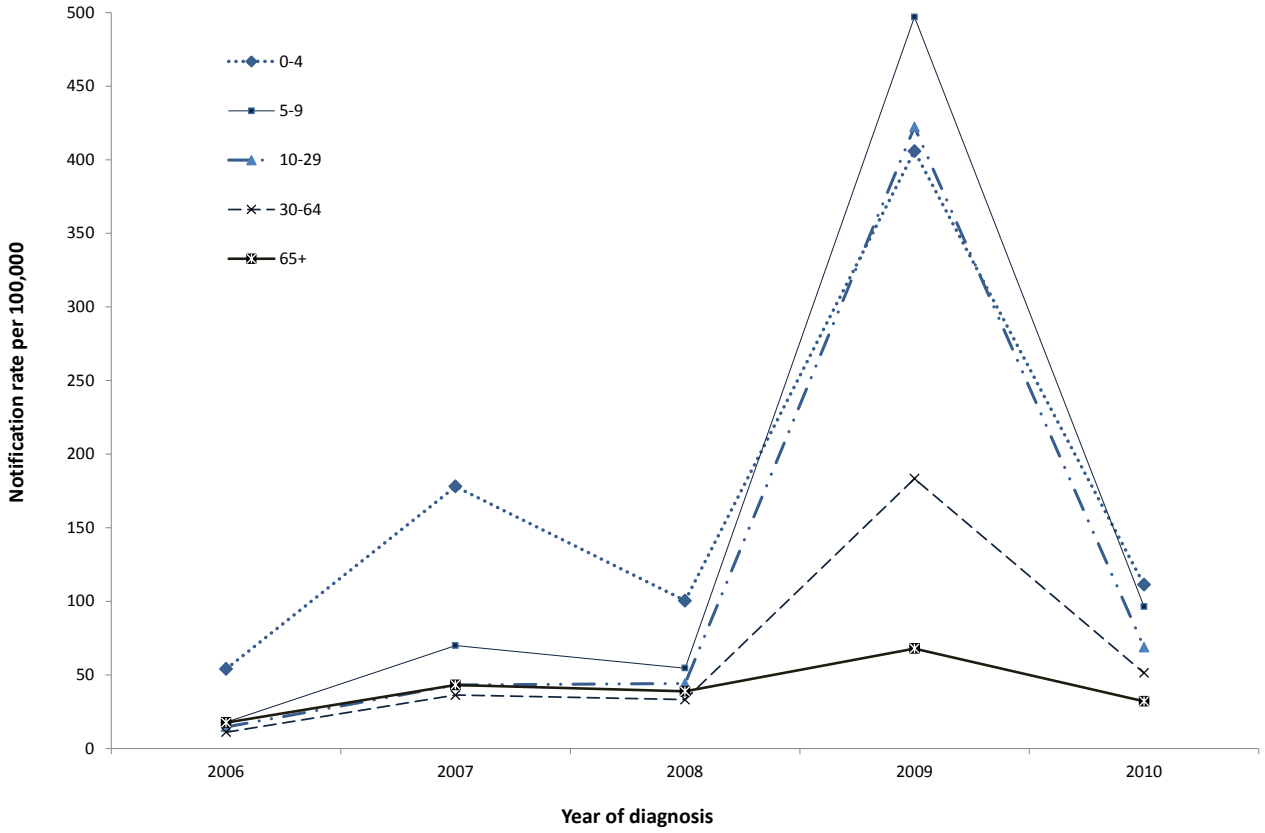
Total notifications in 2010 were approximately equal for both males (47%, n=6,312) and females (53%, n=7,058). However, notifications were slightly higher in females than in males for persons aged between 20 and 69 years, and persons aged 85 years and over. For children aged less than 15 years and persons aged 70 to 84 years, notifications for males exceeded those for females by 17%.

Figure 4 shows rates of notifications for key age groups for the years 2006 to 2010. Overall, notification rates were lower in 2010 for all age groups, compared with the 2009 pandemic year. Notification rates decreased dramatically for persons aged 0 to 29 years, from 431 cases per 100,000 population in 2009 to 80 cases per 100,000 population in 2010.

Virus type and subtype

Analysis of NNDSS influenza typing data indicated the influenza A(H1N1)pdm09 virus remained the predominant subtype in 2010. Almost all (n=13,449) of the influenza cases notified to NNDSS in 2010 included some typing data. Of those with type information, 90% (n=12,096) of notifications were type A (56% (n=7,561) were A(H1N1)pdm09, 30% (n=3,985) were A (unsubtyped) and 4% (n=549) were A(H3N2)) and 10% (n=1,302) were type B (Figure 5). Mixed influenza type A and B infections accounted for less than 1% (n=51) of notifications and typing data were not available for 18 cases.

Figure 4: Notification rate of laboratory-confirmed influenza, Australia, 2006 to 2010, by diagnosis year and age group*



* Notifications of unknown age (n=37 for 2010) were excluded from analysis.

While very little influenza B was detected in Australia in 2010, comprising 10% of influenza notifications to NNDSS (Figure 6), this was a significant increase from the previous year (Figure 5). With the predominance of the influenza A(H1N1)pdm09 virus, influenza B comprised a very small proportion (1%, n=472) of notifications in 2009, as well as a very small number of absolute notifications, compared with previous years (877 notifications in 2006 and 958 notifications in 2007 (Figure 7). In 2008, influenza B was the predominant influenza type for the first time since influenza became nationally notifiable. A breakdown of notifications by type and age indicates that the rate of influenza A was higher than influenza B in all age groups in 2010 (Figure 8).

Virology

The WHO Collaborating Centre for Reference and Research on Influenza (WHOCC) typed and subtyped 1,648 influenza virus samples that were collected in 2010 (Table 2). This represented 12% of 13,467 laboratory-confirmed cases reported to the NNDSS. Influenza A(H1) pandemic 2009 viruses comprised 73% (n=1,206) of viruses, followed by influenza B (14%, n=238; consisting of 99% B/Victoria lineage and just 1% of these were B/Yamagata lineage viruses) and influenza A(H3N2) (12%, n=195).

The 2010 Southern Hemisphere and Australian influenza vaccine included a A/California/7/2009 (H1N1)-like virus, A/Perth/16/2009 (H3N2)-like virus; and a B/Brisbane/60/2008-like virus. The WHOCC conducted antigenic characterisation by Haemagglutination Inhibition (HI) assays on 1,549 influenza virus isolates (Table 3). Nearly

Figure 5: Percentage of laboratory-confirmed influenza notifications, Australia, 2006-2010, by subtype

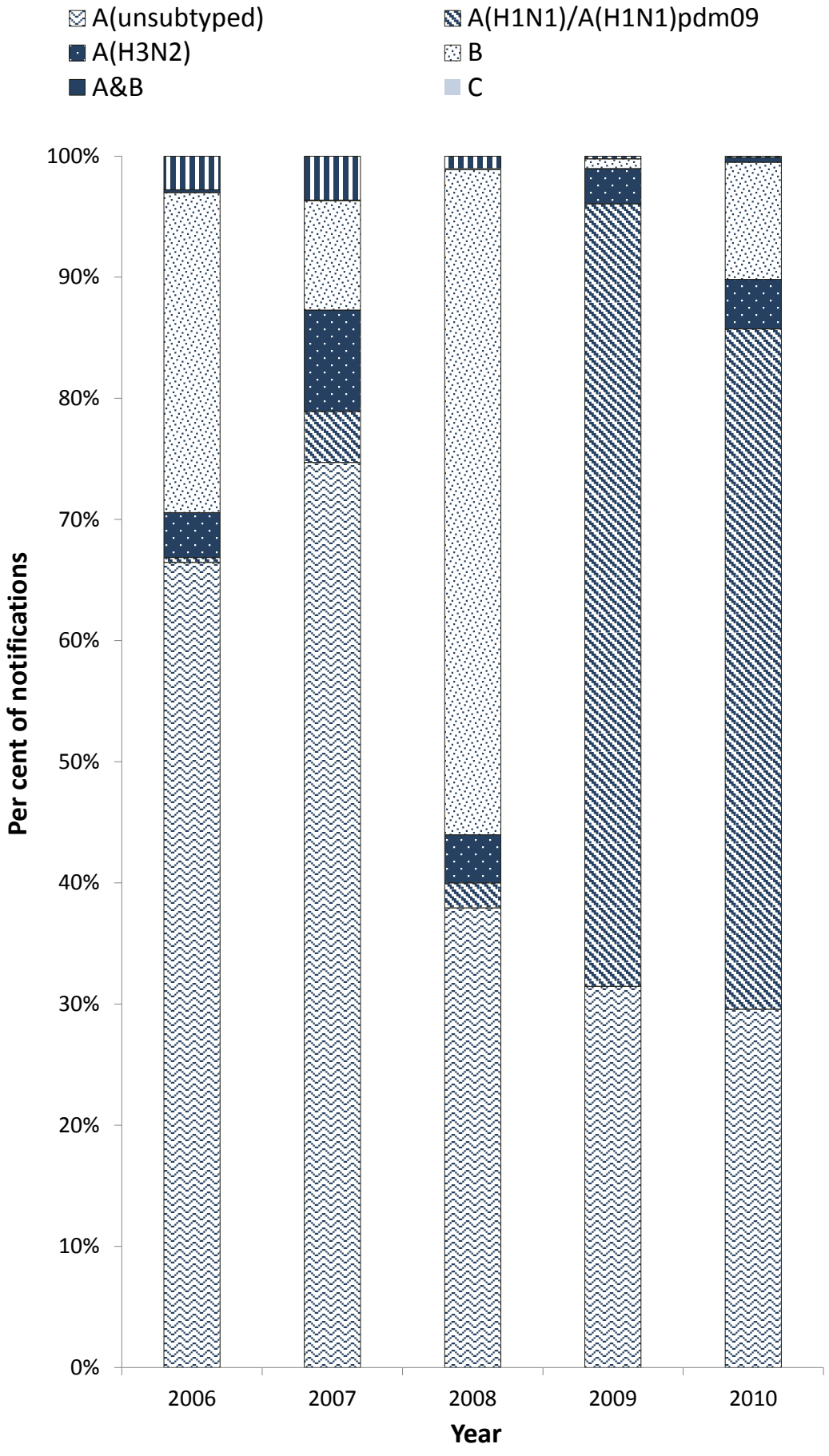
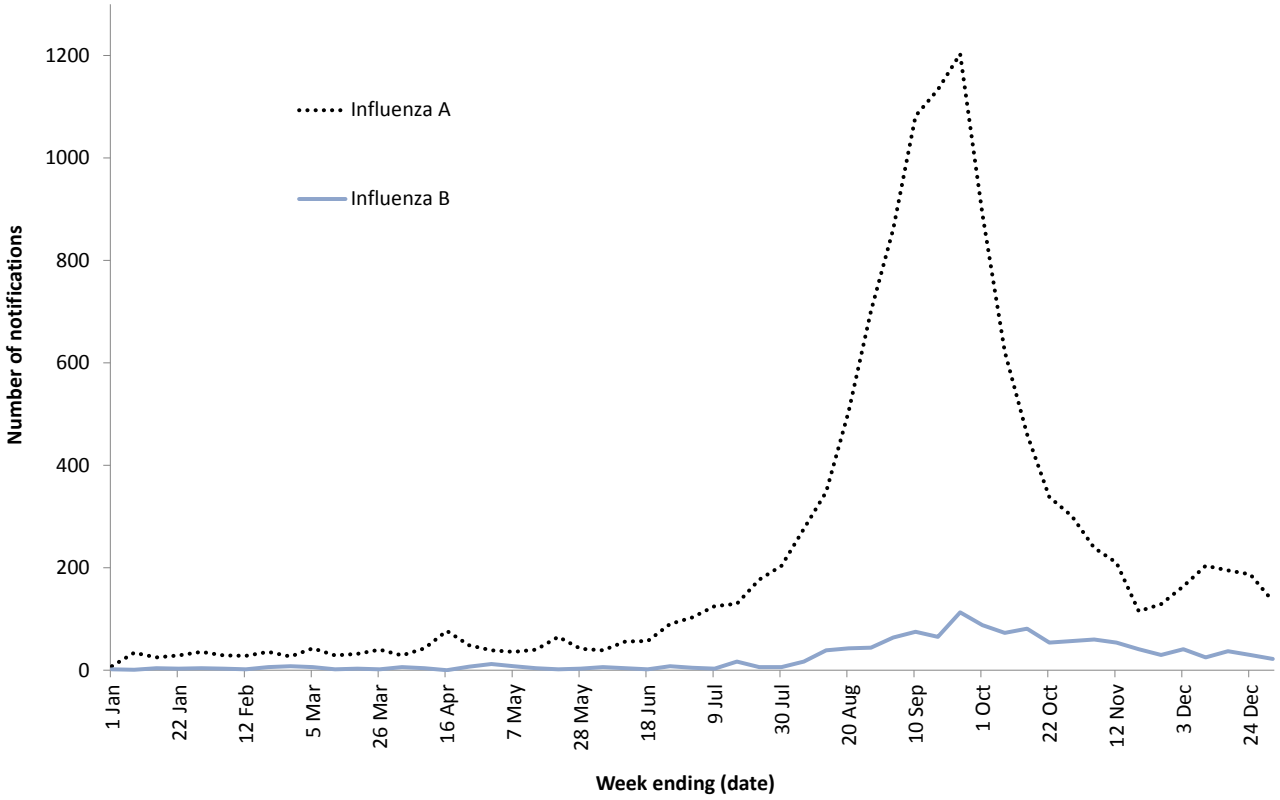


Figure 6: Laboratory-confirmed influenza notifications, Australia, 2010, by type and week of diagnosis*



* Notifications of type "A&B" (n=51) and "untyped" (n=18) influenza were excluded from this analysis.

Figure 7: Laboratory-confirmed influenza notifications, Australia, 2006 to 2010, by type and week of diagnosis

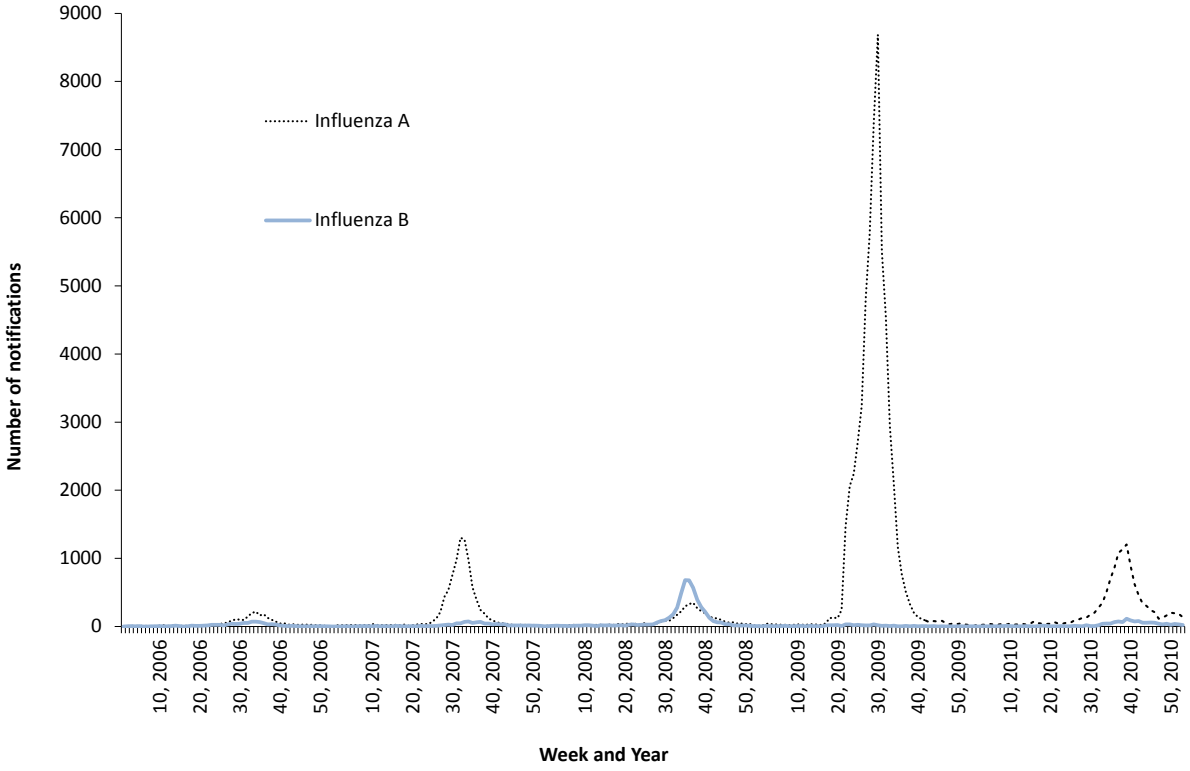


Figure 8: Notifications rate of laboratory-confirmed influenza, Australia, 2010, by type and age group

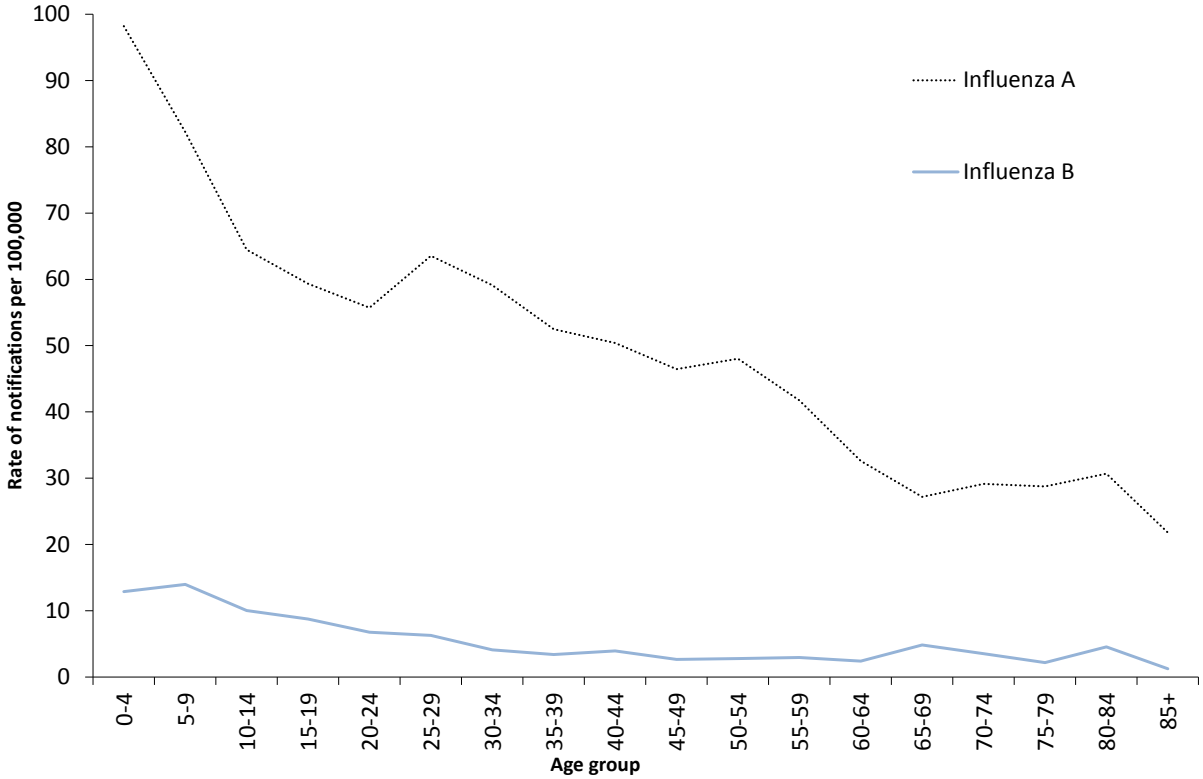


Table 3: Influenza isolates analysed by HI from samples collected by the WHOCC, VIDRL, 2010, by strain

	ACT	NSW	NT	Qld	SA	Tas.	Vic.	WA	Total
A(H1N1)pdm09	**2**	**122**	**96**	**335**	**124**	**0**	**237**	**205**	**1,121**
A/California/7/2009-like[1]	2	121	94	329	123	0	235	205	1,109
A/California/7/2009-like (low reactor)	0	1	2	6	1	0	2	0	12
A(H3N2)	**1**	**11**	**1**	**115**	**3**	**1**	**24**	**30**	**186**
A/Perth/16/2009-like[1]	0	11	0	109	3	1	23	28	175
A/Perth/16/2009-like (low reactor)	1	0	1	6	0	0	1	2	11
B	**0**	**30**	**2**	**47**	**0**	**0**	**10**	**149**	**238**
B/Florida/4/2006-like	0	0	0	0	0	0	0	0	0
B/Florida/4/2006-like (low reactor)	0	0	0	0	0	0	0	2	2
B/Brisbane/60/2008-like[1]	0	30	2	46	0	0	9	146	233
B/Brisbane/60/2008 (low reactor)	0	0	0	1	0	0	1	1	3
Mixed viruses	**0**	**0**	**0**	**0**	**0**	**0**	**4**	**0**	**4**
Mixed H3/(H1N1)pdm09	0	0	0	0	0	0	2	0	2
Mixed B/(H1N1)pdm09	0	0	0	0	0	0	2	0	2
Total	3	163	99	497	127	1	275	384	1,549

[1] Composition of the 2010 Southern Hemisphere influenza vaccine

all (99%, n=1,109) of the A(H1N1)pdm09 isolates were characterised as A/California/7/2009-like, while the remainder were characterised as 'low reactor' compared with the reference virus. Of the circulating influenza A(H3N2) viruses analysed, 94% (n=175) were antigenically similar to the A/Perth/16/2009 virus. For influenza B viruses, 98% (n=233) were closely related to the B/Brisbane/60/2008 virus (a B/Victoria lineage virus). A small number (n=2) of vaccine mismatched influenza B viruses were closely related to the B/Florida/4/2006 virus (B/Yamagata lineage) and were also detected. Thus, the majority of circulating viruses that were isolated in 2010 were antigenically similar to the 2010 vaccine viruses.

Low reactor: these viruses show ≥ 8fold reduction compared to the HI titre obtained with the reference virus and antisera.

Viruses collected in 2010 were also tested for resistance to the antiviral drugs oseltamivir and zanamivir. Neuraminidase inhibition assay (NAI) was performed on 1,541 viral isolates (Table 4). Just three of the A(H1N1)pdm2009 isolates and one of the A(H3) isolates tested, showed resistance to oseltamivir.

Influenza-like illness – national online community survey

Data from the Flutracking survey indicated that national participation more than doubled between the peak week of 2008 (n=4,827 and 207.3 respondents per 100,000 population) and the peak week of 2010 (10,773 respondents and 48.2 respondents per 100,000 population).

The peak in the proportion of Flutracking survey participants reporting a fever and cough in 2010 (4%) was lower than the two previous years (5% in 2009 and 6% in 2008) (Figure 9). The timing of the 2010 peak (week ending 5 September 2010) was eight weeks later than the peak week in 2009 (week ending 12 July 2009) and one week later than the peak week in 2008 (week ending 31 August 2008).

Influenza-like illness – National Health Call Centre Network

In 2010, 34,120 calls were made to the NHCCN relating to ILI, comprising 6% of total calls. Calls relating to ILI increased gradually throughout the year, peaking at 1,086 calls (9%) in mid-September (week 38) (Figure 10). The percentage of total calls to the NHCCN relating to ILI peaked at 9.0% in early June (week 27) and plateaued until around mid-September. While there is a seasonal trend in the number and percentage of calls to the NHCCN related to ILI, it is not as marked as the seasonal trend in notifications.

Influenza-like illness consultations from sentinel general practitioner surveillance systems

Data from ASPREN and VIDRL sentinel GPs showed that for 2010 there were 4,655 notifications for ILI. An average of 94 doctors reported to ASPREN each week (range 74 to 107), with an average of 9,172 consultations per week (range 3,082 to 11,205) across all states and territories.

Overall, the consultation rates for ILI were lower in 2010 than in years 2008 and 2009 (Figure 11). Consultation rates increased gradually through to August, increased sharply in late August (week 34) and peaked at 15 ILI cases per 1,000 consultations in late September (week 41) which was consistent with NNDSS notifications. Consultation rates for ILI then decreased through November but they were relatively stable in December. In 2010 consultations rates peaked lower and later than in 2009 and 2008, although rates were higher than the previous year during the 2010-11 inter-seasonal period (also consistent with NNDSS notifications).

At a state level, ASPREN GPs in New South Wales had higher rates of ILI notifications compared with all the other states and territories with 15 consultations for ILI per 1,000 consultations, followed by South Australia and the Australian Capital Territory (both with 10 per 1,000 consultations).

A breakdown of ASPREN data by age and sex indicates that the highest rate of ILI presentations were recorded in children under 1 year of age and aged 1–4 years (Figure 12). This is consistent with NNDSS influenza notifications. The rate of ILI presentations was higher for females aged 20 to 74 years, which is consistent with trends in laboratory-confirmed influenza notifications.

ASPREN data are not completely representative of the Australian population. In 2010, average consultation rates ranged from 7 per 1000 population in Victoria to 79 per 1000 population in Western Australia. It is also difficult to compare across different years, as representativeness varies over time, due to changes in the number of reporting doctors.

Swab tests for laboratory-confirmed influenza were performed for 19% of ILI notifications (550/2,915) through ASPREN; this excludes ILI notifications from Victoria and Western Australia that were not swab tested under similar conditions. Of these ILI notifications tested in 2010, 21% (n=114) were positive for influenza, with the majority attributable to influenza A(H1N1)pdm09 (16%, n=86).

Influenza-like illness – sentinel emergency department surveillance

Presentations to New South Wales EDs for ILI were low and relatively stable in 2010, peaking at just 2.1 presentations per 1,000 consultations in early October (week 41) compared with an interseasonal rate of 0.9 (Figure 13). The rise in laboratory-confirmed notifications of influenza to NNDSS through the 2010 season was not reflected in the presentation rates to New South Wales EDs. Presentation rates in 2010 were generally well below levels observed across the previous four years.

Presentations to emergency departments in Western Australia for ILI increased gradually from the beginning of the year, following the

Table 4: Neuraminidase inhibitor resistance in influenza viruses collected and tested at the WHOCC, 2010, by subtype

Type/subtype	NI resistant virus isolates tested by enzyme inhibition assay[1]			NI resistant clinical samples tested by pyrosequencing[2]		Frequency of oseltamivir resistance (%)
	Tested	Oseltamivir resistant	Zanamivir resistant	Tested	H275Y mutation (oseltamivir-resistant)	
A(H1) pandemic 2009	1,117	3	0	4	0	0.30%
A(H3)	185	1	0	0	0	0.50%
B(VIC)	236	0	0	0	0	0
B(YAM)	2	0	0	0	0	0
Total	1,541	4	0	4	0	0.30%

Figure 9: Percentage of Flutracking respondents reporting fever and cough, Australia, April to October, 2008 to 2010, by week of reporting

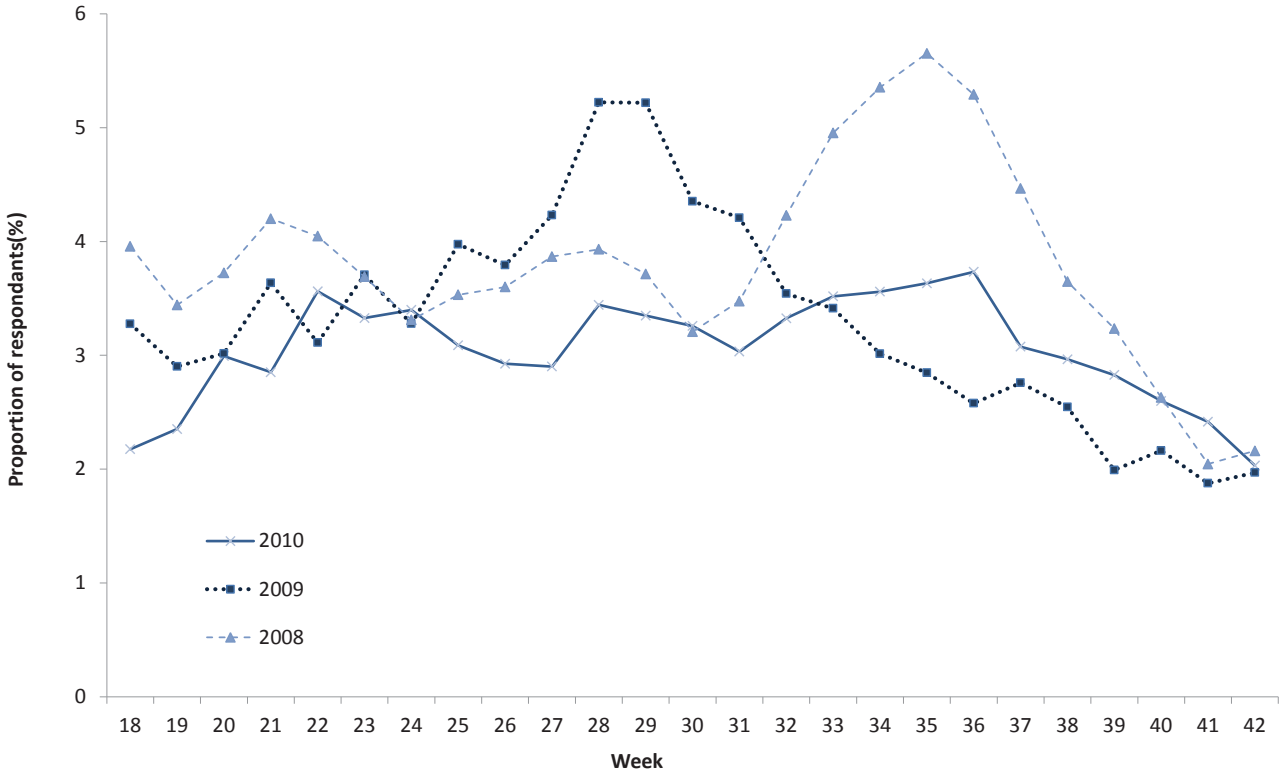
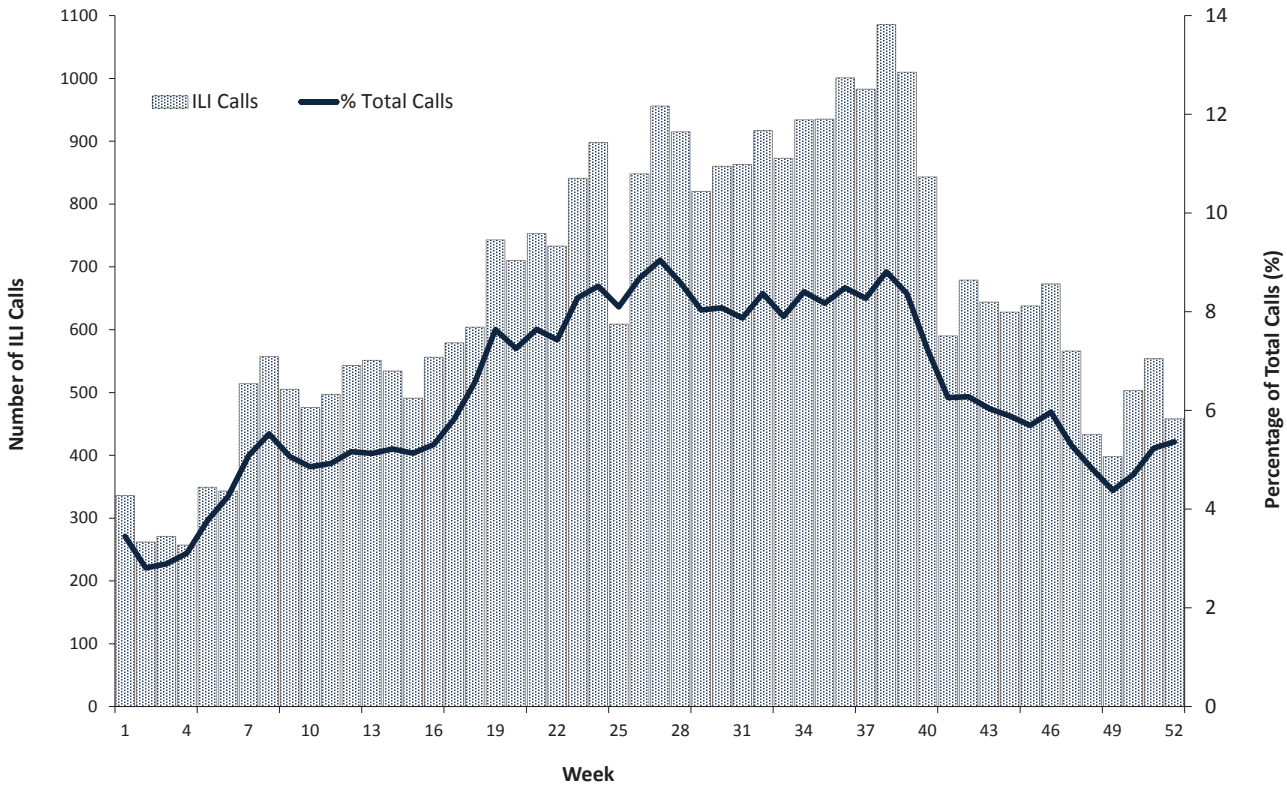


Figure 10: Number of ILI-related calls to the NHCCN compared with the percentage of total calls, Australia, 2010, by week of reporting



trend seen in 2008 (Figure 14). The number of presentations peaked at 678 in late September 2010 (week 40), which was consistent with the peak of Western Australia influenza notifications to NNDSS. The proportion of presentations admitted to hospital peaked at 11.0% (n=299) in early November (week 46). ED presentations for ILI in the peak week of 2010 were lower than the pandemic year of 2009 (n=1,266) and similar to 2008 (n=633).

Sentinel ED surveillance data were timely, and a useful indicator of seriousness of disease but, at the time, were only available from New South Wales and Western Australia. ED surveillance systems operated in other jurisdictions as well but these did not routinely report data to the National Influenza Surveillance Scheme in 2010.

Laboratory surveillance

Sentinel laboratory data from New South Wales, Western Australia, the Northern Territory**, Victoria and Tasmania showed that the number of laboratory virology tests for respiratory illness increased gradually from around 200 tests per week in March and April to a peak of 767 tests per week in mid-September (Figure 15).

The percentage of virology specimens testing positive peaked at approximately 18% in week 36 (week ending 10 September respectively) (Figure 15). The peak in week 36 confirms the increase in the number of seasonal influenza cases. Since the number of tests did not increase greatly during these weeks, it also indicates that increasing numbers were not an artefact of increased testing.

** Reported by the Western Australia National Influenza Centre.

Figure 11: GP consultation rates for influenza-like illness, Australia, 2008 to 2010, by week of reporting

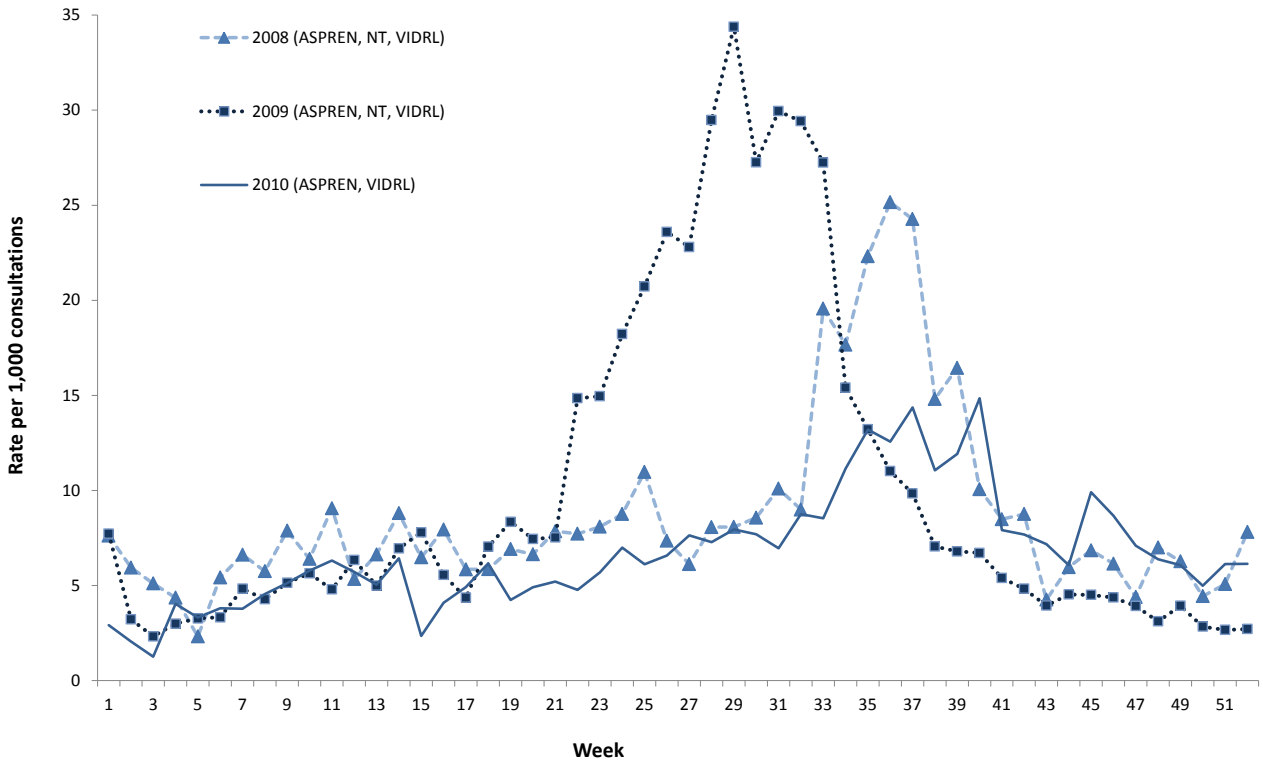
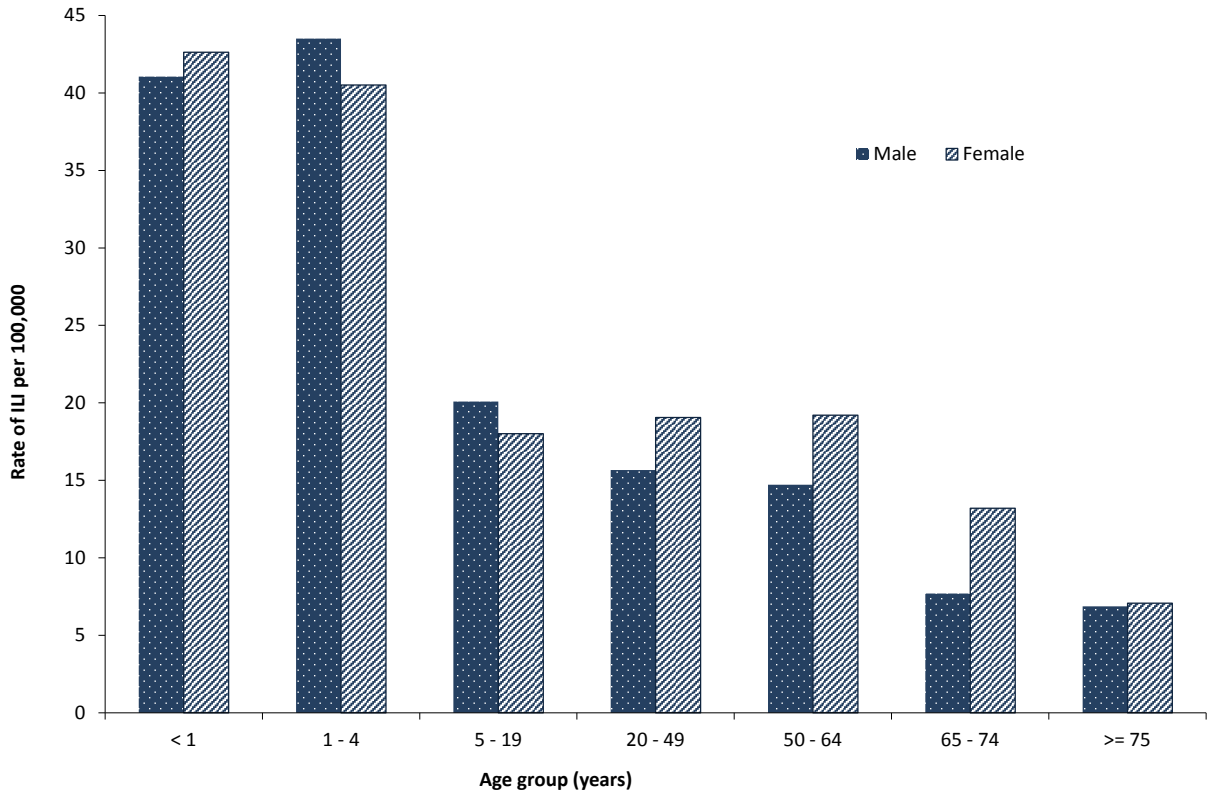


Figure 12: Consultation rates for influenza-like illness, ASPREN, 2010, by age group and sex



Morbidity

Australian Paediatric Surveillance Unit surveillance (APSU)

APSU reported that between 1 June and 25 October 2010, there were 25 cases of children aged 15 years or younger admitted to ICUs in Australia following complications due to influenza infection, seven of which were reported to have an underlying chronic condition. Of the admissions, 23 (92%) were attributed to influenza A (including 17 H1N1pdm09 and six unsubtyped) and two (8%) were influenza B. The ages at the time of admission ranged from six days to 11 years, with a median age of 3 years.

FluCAN

FluCAN reported 296 influenza-associated hospitalisations from sentinel hospitals between 13 February and 29 October 2010, including 76 admitted directly to the ICU. Weekly hospitalisations peaked at 36 admissions in mid-September, in line with the peak in laboratory-confirmed influenza notifications (Figure 16).

The majority of admissions (n=231 or 78%) were associated with influenza A(H1N1)pdm09. Risk factors were present for 70% (n=207) of hospitalisations and around 7% (n=20) were pregnant.

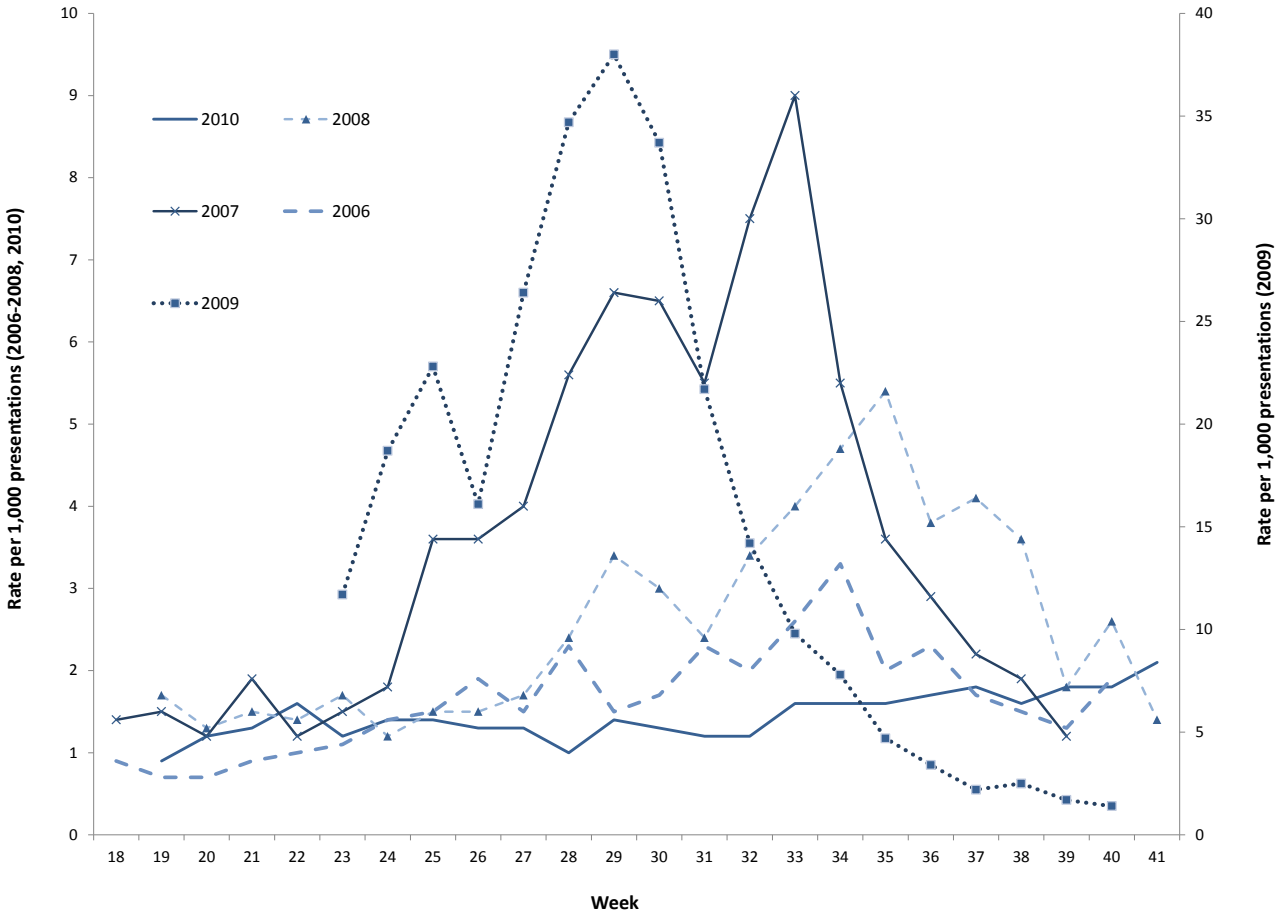
Mortality

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

Deaths from pneumonia and influenza – New South Wales

Mortality rates from influenza in New South Wales reported by the Registry of Births, Deaths and Marriages showed that rates of deaths from influenza and pneumonia peaked in early September at approximately 140 per 1,000 deaths (Figure 17).[2] The combined pneumonia and influenza death rates were equal to or below

Figure 13: Presentation rates for influenza-like illness at hospital emergency departments New South Wales, April to October, 2006 to 2010, by week of reporting



the predicted seasonal baseline for the majority of the season. Death rates were well below the upper 95% confidence interval of the predicted seasonal baseline during 2010.

Australian Bureau of Statistics death data

Influenza and pneumonia (ICD-10 codes J09–J18)[5] were noted as the underlying cause of death for 2,364 persons in 2010 (1.6% of all deaths), of which 56% (n=1,324) were female.[3] The rate of influenza and pneumonia deaths was 8.8 per 100,000 deaths. The standardised death rate was higher in males, with 10.4 per 100,000 deaths, compared to females at 7.8 per 100,000 deaths.

Discussion

The seriousness of disease and impact of influenza are difficult to measure due to the nature of the illness and limitations of surveillance systems. Influenza surveillance in Australia relies on a network of data sources and systems, varying in their ability to detect true cases of influenza. Ideally, the number of laboratory-confirmed notifications would include all cases, rather than just those that have been tested, and sentinel GP and ED surveillance systems would indicate the burden of disease on health systems and the community. Hospitalisation and death data could also be improved to allow for true indicators of clinical severity, morbidity and mortality due to influenza infection. It is possible that notifications in 2010 may have been affected by heightened media attention and public awareness following the 2009 pandemic, however it is not currently possible to measure the extent of this impact (if any).

Based on available data, the 2010 influenza season in Australia was considered moderate overall in comparison with previous seasons.

Figure 14: Number of influenza-like illness consultations in hospital emergency departments, Western Australia, January to December, 2008 to 2010, by week of reporting

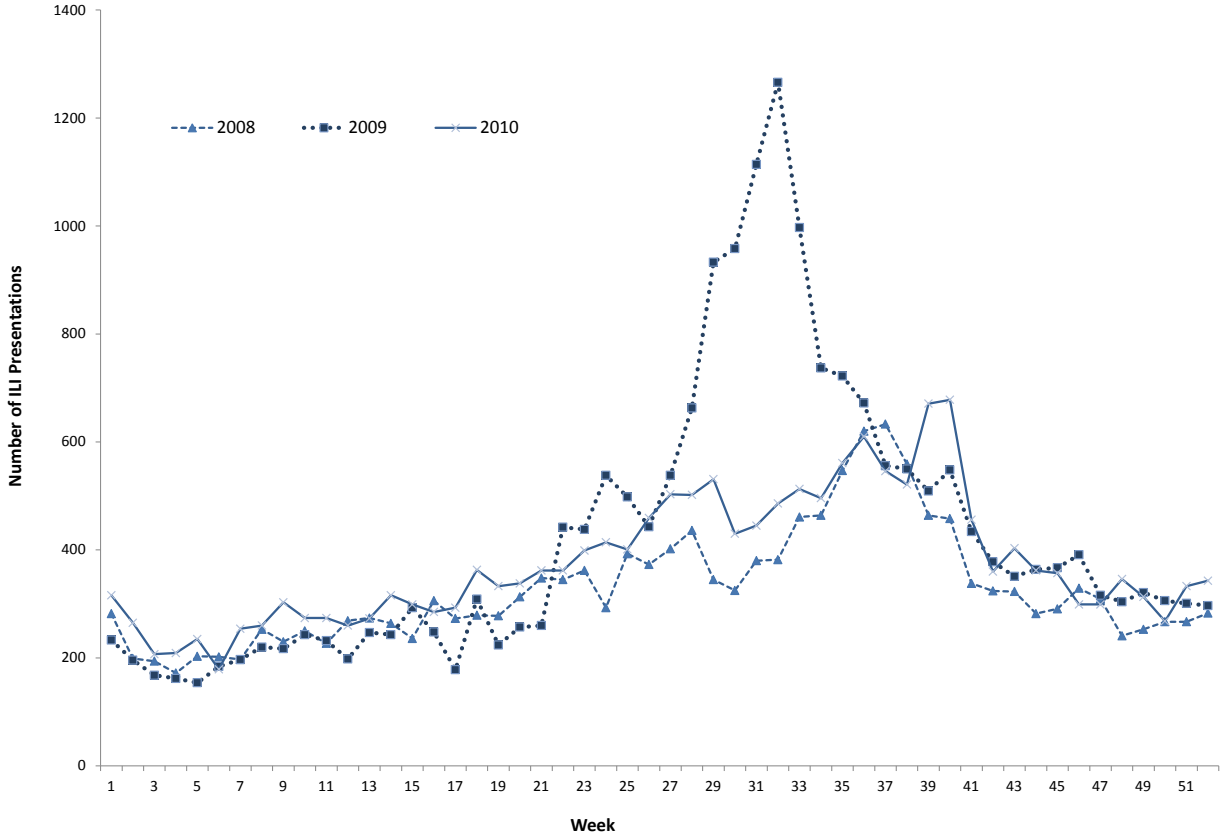
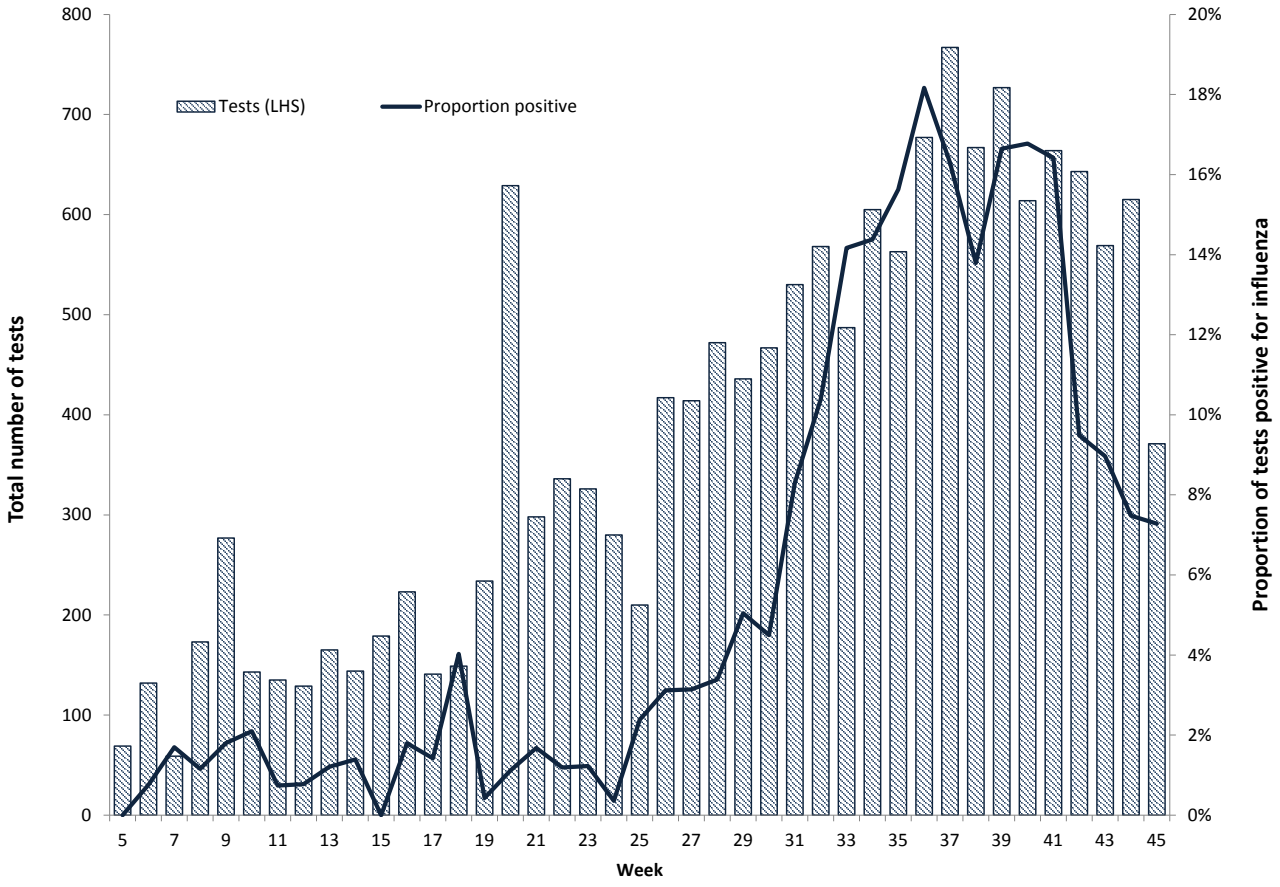


Figure 15: Number of virology specimens tested and percentage testing positive for influenza, February to November, 2010, by week of reporting



Laboratory-confirmed influenza notifications were around 0.8 times the 5-year mean, however excluding the 2009 pandemic year, 2010 notifications were almost double the previous four years. While generally the larger jurisdictions recorded higher case numbers and smaller jurisdictions had higher rates, as per previous seasons, a significant difference in 2010 was the proportion of notifications attributed to South Australia (comprising almost a third of total notifications with a rate of 259 notifications per 100,000 population).

As observed during previous years (with the exception of 2009), the highest rates of laboratory-confirmed influenza occurred in children under 5 years of age, especially in those aged less than 1 year. While notification rates for people in the 5-9, 10-29 and 30-64 year age groups decreased significantly compared with 2009, the predominance of the influenza A(H1N1)pdm09 strain ensured that the rates for these age groups remained high compared with those recorded in earlier years.

All sentinel ILI data sources (including GP and ED presentations, the Flutracking community survey and calls to the National Health Call Centre Network) indicated that the 2010 season was low to moderate overall. Trends in ILI were largely consistent with laboratory-confirmed influenza data, with the majority of data sources peaking in September.

The 2010 season was predominantly attributed to influenza A(H1N1)pdm09, which accounted for 56% of all laboratory-confirmed influenza notifications (followed by 30% unsubtyped influenza A). Of influenza viruses circulating during the 2010 season, A(H1) viruses were mostly antigenically similar to the vaccine strain A/California/7/2009 (H1N1) and A(H3) strains were mostly similar to the vaccine strain A/Perth/16/2009 (H3N2). While influenza B only accounted for 10% of notifications in 2010, this compares with just 1% during the previous year. Around 99% of influenza B viruses characterised by the WHOCC were from the Victoria lineage and most were similar to the B/Brisbane/60/2008 vaccine strain.

Figure 16: Number of influenza hospitalisations at sentinel hospitals, 13 February to 29 October 2010, by week of reporting and influenza subtype

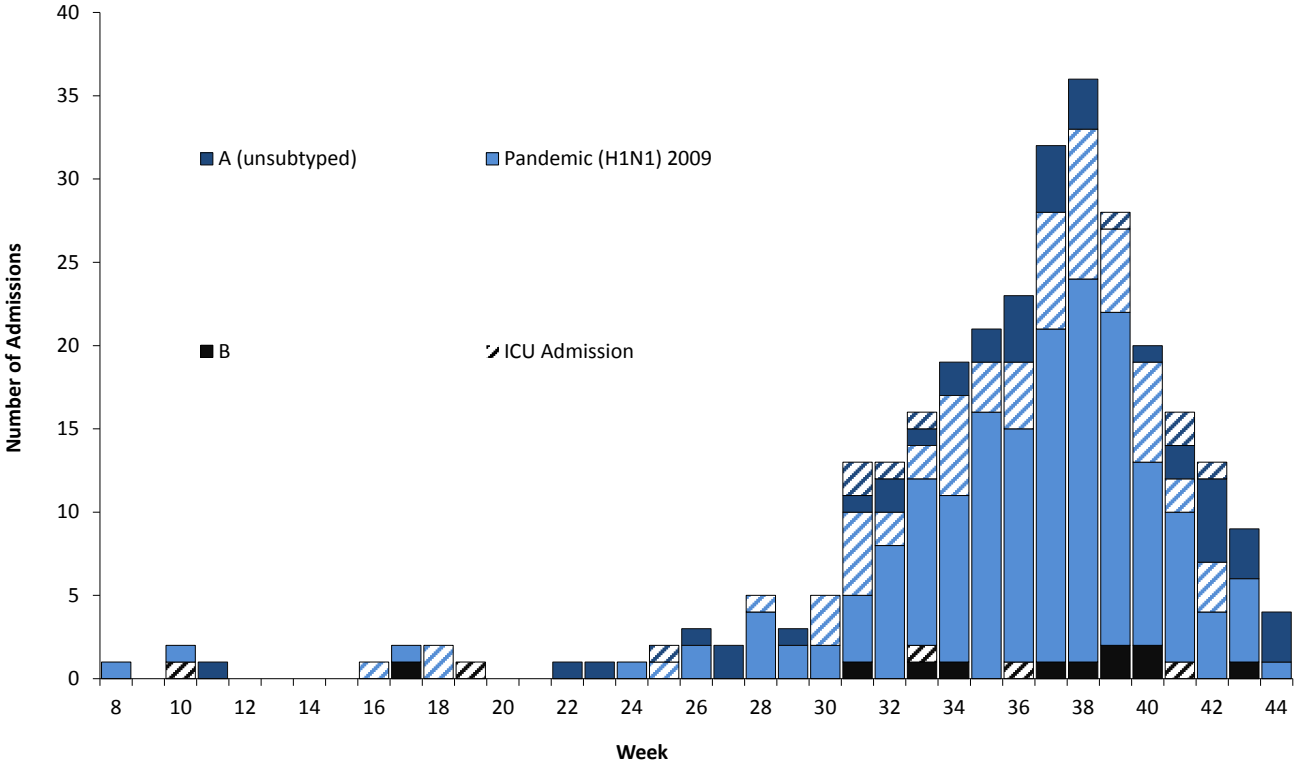
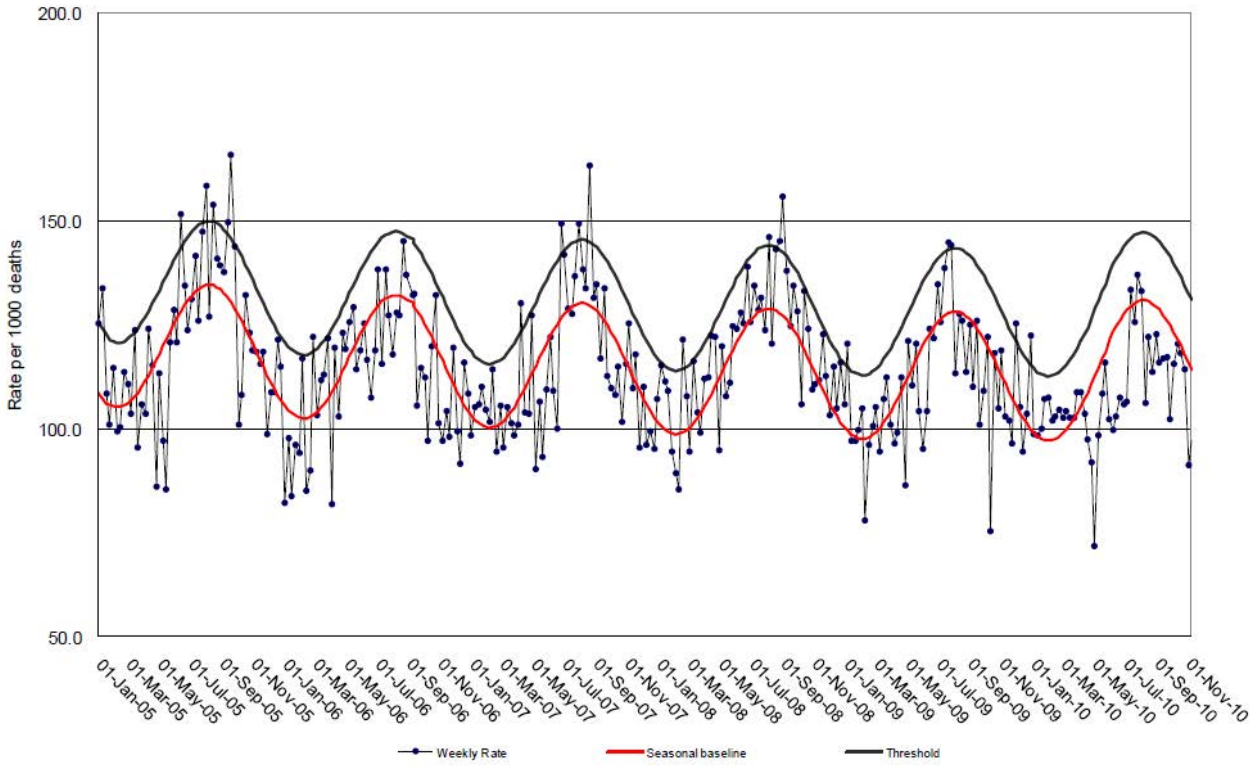


Figure 17: Observed and predicted rate of influenza and pneumonia deaths as per New South Wales registered death certificates, 1 January 2005 to 1 November 2010



Based on data generated during the 2010 Southern Hemisphere influenza season, at their technical meeting in September 2010, the WHO recommended the following influenza virus strains for inclusion in the 2011 Southern Hemisphere seasonal influenza vaccine:

- A/California/7/2009-like virus (H1N1);
- A/Perth/16/2009-like virus (H3N1); and
- B/Brisbane/60/2008-like virus.

The recommendation for the 2011 Southern Hemisphere vaccine was identical to the recommended composition of the 2010 Southern Hemisphere vaccine.

Overall, all data sources were consistent in indicating trends in influenza activity and support the characterisation of the 2010 influenza season as moderate.

Acknowledgements

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Annual report

Australian Meningococcal Surveillance Programme annual report, 2016

Monica M Lahra, Rodney Enriquez for the National Neisseria Network

Abstract

In 2016, there were 243 laboratory-confirmed cases of invasive meningococcal disease analysed by the Australian National Neisseria Network. This number was the highest number of laboratory confirmed cases since 2008. Probable and laboratory confirmed invasive meningococcal disease (IMD) are notifiable in Australia, and there were 252 IMD cases notified to the National Notifiable Diseases Surveillance System in 2016, the highest number reported since 2010. Meningococcal serogrouping was able to be determined for 98% (237/243) of laboratory confirmed IMD cases. Serogroup B infections accounted for 87 cases (37%), the lowest number and proportion reported since inception of the Australian Meningococcal Surveillance Programme (AMSP) in 1997. In contrast, the number and proportion of serogroup W infections (44%, 107 cases) in 2016 was the highest since the AMSP began. In addition, the number and proportion of serogroup Y infections (16%, 40 cases) was also the highest recorded by the AMSP. Molecular typing results were available for 225 of the 243 IMD cases. Of the serogroup W IMD strains that were able to be genotyped, 92% (97/105) have the PorA antigen encoding gene type P1.5,2 and of these, 72% (70/97) were sequence type 11, the same type as the hypervirulent serogroup W strain that has been circulating in the UK and South America since 2009. The primary IMD age peak was observed in adults aged 45 years or more, whilst secondary disease peaks were observed in those aged less than 5 years, and in adolescents aged 15–19 years. Serogroup B infections predominated in the age groups less than 1 year and 20–24 years, whereas serogroup W infections predominated in those aged 45 years or more. For all other age groups, distribution of serogroup B and W infections was roughly equal. Of the IMD isolates tested for antimicrobial susceptibility, 6% (11/189) were resistant to penicillin, and decreased susceptibility to penicillin was observed in a further 90% (170/189) of isolates. One Men W isolate demonstrated an elevated minimum inhibitory concentration (MIC) to ceftriaxone (0.125mg/L), the highest reported in Australia. All isolates tested were susceptible to rifampicin and ciprofloxacin.

Keywords: antibiotic resistance; disease surveillance; meningococcal disease; Neisseria meningitidis

Introduction

Australia's National Neisseria Network (NNN) is an established, collaborative network of reference laboratories in each state and territory that contribute to the laboratory surveillance system of the pathogenic *Neisseria* species *(N. meningitidis* and *N. gonorrhoeae).* Since 1994 the NNN has coordinated laboratory data from the examination of *N. meningitidis* cases of invasive meningococcal disease (IMD) for the Australian Meningococcal Surveillance Programme (AMSP).[1] The AMSP is funded by the Australian Government Department of Health. The NNN laboratories supply phenotypic and genotypic data of invasive meningococci for the AMSP. These data supplement the notification data from the National Notifiable Diseases Surveillance System (NNDSS), which includes cases of probable and laboratory confirmed IMD. The characteristics of meningococci responsible for IMD, and the associated demographic information, are important considerations for management of individual patients and their contacts; and

to inform public health responses for outbreaks or case clusters, locally and nationally. The introduction of the publicly funded conjugate serogroup C meningococcal vaccine onto the National Immunisation Program in 2003 has seen a significant and sustained reduction in the number of cases of serogroup C IMD after 2003.[2] However, IMD remains an issue of public health concern in Australia and continued monitoring of phenotypic and genotypic features of IMD strains is critical to monitor, plan and inform clinical management and public health interventions.

Methods

Case confirmation of invasive meningococcal disease

Case confirmation is based on isolation of *N. meningitidis*, or a positive nucleic acid amplification testing (NAAT) from a normally sterile site, defined as laboratory definitive evidence of IMD by the Communicable Diseases Network Australia (CDNA) criteria [3]. Information regarding the site of infection, age and sex of the patients is collated by the NNN for the AMSP.

IMD cases are categorised on the basis of the site from which *N. meningitidis* was isolated, or from which meningococcal DNA was detected (blood, joint fluid, vitreous fluid). When *N. meningitidis* is detected from both blood and cerebrospinal fluid (CSF) from the same patient, the case is classified as one of meningitis.

Phenotyping and genotyping of *Neisseria meningitidis*

Phenotyping is limited to the determination of the serogroup by detection of soluble polysaccharide antigens. Genotyping of both isolates and DNA extracts is performed by sequencing of products derived from amplification of the porin genes *por*A, *por*B and *fet*A.

Antibiotic susceptibility testing

Isolates were tested to determine their minimum inhibitory concentration (MIC) values to antibiotics used for therapeutic and prophylactic purposes: ceftriaxone, ciprofloxacin; rifampicin. This program defines the penicillin categories as: sensitive (MIC ≤ 0.03 mg/L); less sensitive (MIC 0.06–0.5 mg/L) and resistant (MIC ≥ 1 mg/L).

Results

In 2016, there were 243 laboratory-confirmed cases of IMD analysed by the NNN, and 252 cases notified to the NNDSS. Thus, laboratory data were available for 96% of notified cases of IMD in Australia in 2016 (Figure 1). This number of laboratory-confirmed cases of IMD was the highest reported since 2008, with an increase of 40% from the previous year (n=174). The number of cases notified to the NNDSS was the highest reported since 2010 (n=226), with an increase of 39% from the previous year (n=182). In 2016 the peak incidence for IMD occurred in mid-spring and early summer (1 October to 31 December 2016) (Table 1). This was different to previous years where the peak incidence occurred in mid-winter and early spring.

Victoria reported the highest number of cases (76 cases) in 2016, an increase from 54 cases in 2015 and the highest number of cases reported from this state since 2005 (n=80) (Table 2). New South Wales had the second highest number of IMD cases in 2016 (69 cases) and this was the highest number of cases reported in this state since 2010 (n=76). All jurisdictions, with the exception of South Australia and the Australian Capital Territory, recorded a rise in IMD cases in 2016 compared with 2015.

Age distribution

The peak incidence of IMD in 2016, as in 2015, occurred in adults aged 45 years or more. This age group represented 35% (86/243) of IMD cases in 2016 (Table 3). Within this age group, 50 cases were in those aged 65 years or more, which was the highest number and proportion of cases for this age group reported by the AMSP. Prior to 2015, the primary peak incidence of IMD was in children less than 5 years of age, however in 2016, they represented 21% of IMD cases, the lowest proportion of cases noted by the AMSP

Figure 1: Number of invasive meningococcal disease cases reported to the National Notifiable Diseases Surveillance System compared with laboratory confirmed data from the Australian Meningococcal Surveillance Programme, Australia, 2016

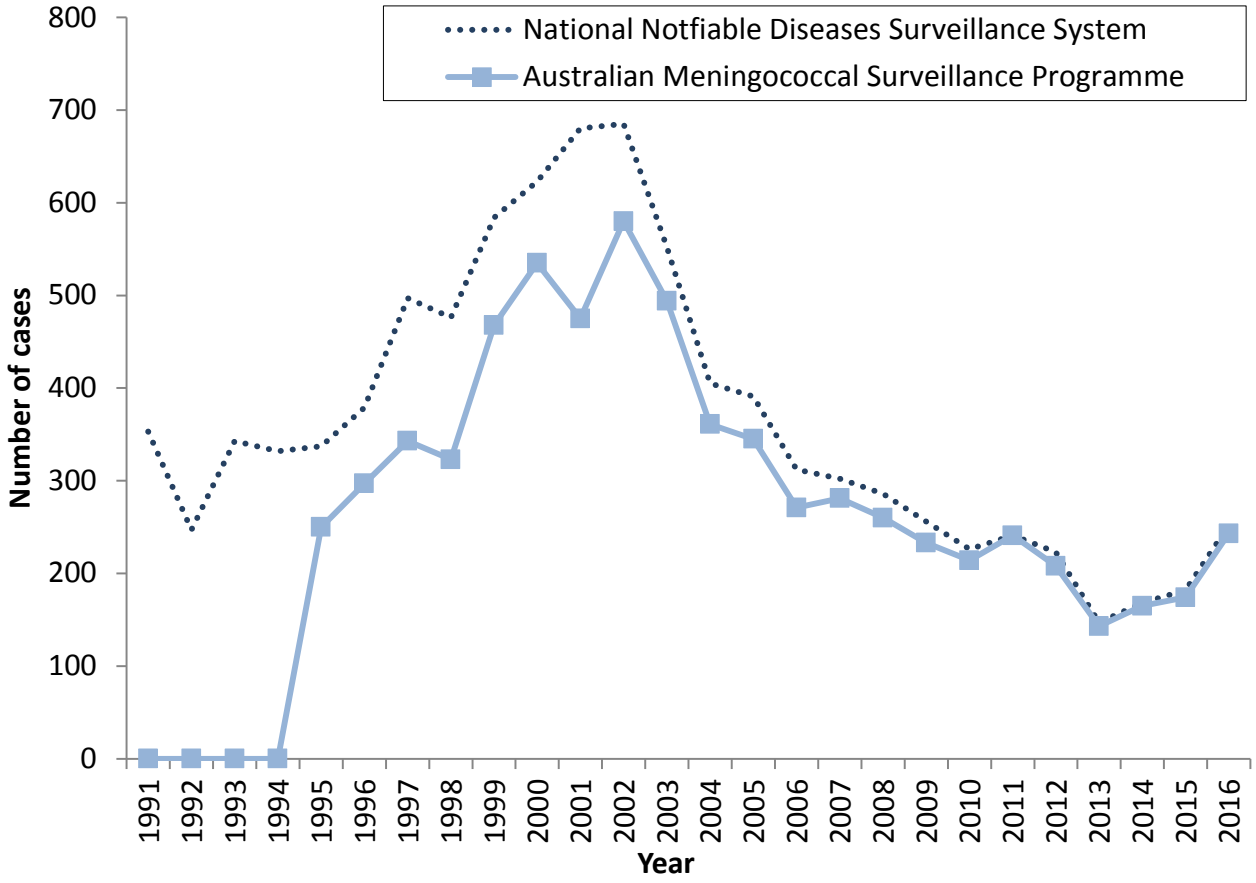


Table 1: Laboratory confirmed cases of invasive meningococcal disease, Australia, 2016, by quarter

Serogroup	01 January - 31 March	01 April - 30 June	01 July – 30 September	01 October - 31 December	2016 Total
B	19	18	28	22	87
C	2	0	0	1	3
Y	4	8	10	18	40
W	14	17	36	40	107
NG	2	0	1	0	3
ND	1	0	1	1	3
Total	42	43	76	82	243

NG: non groupable.
ND: not determined.

Table 2: Number of laboratory confirmed cases of invasive meningococcal disease, Australia, 2016, by state or territory and serogroup

State or territory	Serogroup						Total
	B	C	Y	W	NG	ND	
ACT	1	0	0	1	0	0	2
NSW	25	2	15	23	3	1	69
NT	2	0	0	0	0	0	2
Qld	15	0	13	14	0	0	42
SA	22	0	0	4	0	0	26
Tas.	0	0	1	4	0	0	5
Vic.	17	1	9	47	0	2	76
WA	5	0	2	14	0	0	21
Australia	**87**	**3**	**40**	**107**	**3**	**3**	**243**
Proportion of all IMD cases	35.8%	1.2%	16.5%	44.0%	1.2%	1.2%	

NG: non groupable
ND: not determined

Table 3: Laboratory-confirmed cases of invasive meningococcal disease, Australia, 2016, by age and serogroup

Serogroup	Age group									Total
	<1	1 to 4	5 to 9	10-14	15-19	20-24	25-44	45-64	65+	
B	14	11	3	1	14	18	15	6	5	87
C	0	0	0	0	1	0	1	1	0	3
Y	2	1	1	0	5	4	2	10	15	40
W	8	11	3	0	13	7	16	19	30	107
NG	0	2	0	0	0	0	1	0	0	3
ND	0	1	0	0	1	1	0	0	0	3
Total	24	26	7	1	34	30	35	36	50	243
%B of within age group	58.3	42.3	42.9	100.0	41.2	60.0	42.9	16.7	10.0	

NG: non groupable
ND: not determined

for this age group in any year. Between 2003 and 2014, the proportion of IMD that occurred in children aged less than 5 years ranged from 28% to 36% of cases. A secondary disease peak has also been observed in previous years amongst adolescents aged 15–19 years. Of the total cases of IMD in 2016, 14% (34/243) were in those aged 15–19 years, which was less than the proportion reported in the period 2006 to 2011 (16–20%), and 2013–2015 (17–20%).

Anatomical site of samples for laboratory confirmed cases

In 2016, diagnosis was made by a positive culture in 78% (189/243) of cases and 22% (54/243) of cases were confirmed by NAAT testing alone (Table 4).

There were 45 diagnoses of meningitis based on cultures or NAAT examination of CSF either alone or with a positive blood sample. There

Table 4: Number of laboratory-confirmed cases of invasive meningococcal disease, Australia, 2016, by anatomical source and method of confirmation

Specimen type	Isolate of NM*	PCR positive**	Total
Blood	162	26	188
CSF +/- Blood	17	28	45
Other	10	0	10
Total	189	54	243

* NM: *Neisseria meningitidis*
** NAAT: nucleic acid amplification testing.

were 188 diagnoses of septicaemia based on cultures or NAAT examination from blood samples alone (Table 4). There were 8 IMD diagnoses by positive joint fluid culture, 1 IMD diagnosis by positive tissue culture, and 1 IMD diagnosis by positive abscess culture.

Serogroup data

Number and proportions of cases of serogroup B, C, Y, W invasive meningococcal disease

The serogroup was able to be determined for 237 of 243 laboratory-confirmed cases of IMD (98%) in 2016 (Tables 2 and 3). The overall decrease in IMD cases since 2002 was initially predominantly due to a reduction in the number of cases of IMD caused by serogroup C from 2003 to 2007 following the introduction of the serogroup C vaccine. After 2009, a decline in the numbers IMD cases caused by serogroup B was reported, from 194 cases in 2009 to 104 cases in 2013. In 2014, there was an increase in the numbers of IMD cases caused by serogroup B (n=129), however, in 2015 the numbers of IMD cases caused by serogroup B was similar to 2013. In the years 2006–2012 the proportion of IMD cases caused by serogroup B was 84%–88%, in 2013–2014 it was 75–80%, and in 2015 it was 64%. The number and proportion of IMD cases caused by serogroup B declined further in 2016 to the lowest number (n=87) and proportion of total IMD (36%) reported by the AMSP. The number of IMD cases caused by serogroup C (3 cases) in 2016 was similar to the previous year 2015 (2 cases), which was the lowest total reported by the AMSP. Since 2014, the rise in the total number of IMD cases has been due to a rise in the number of cases of IMD caused by serogroup W and serogroup Y (Figure 2).

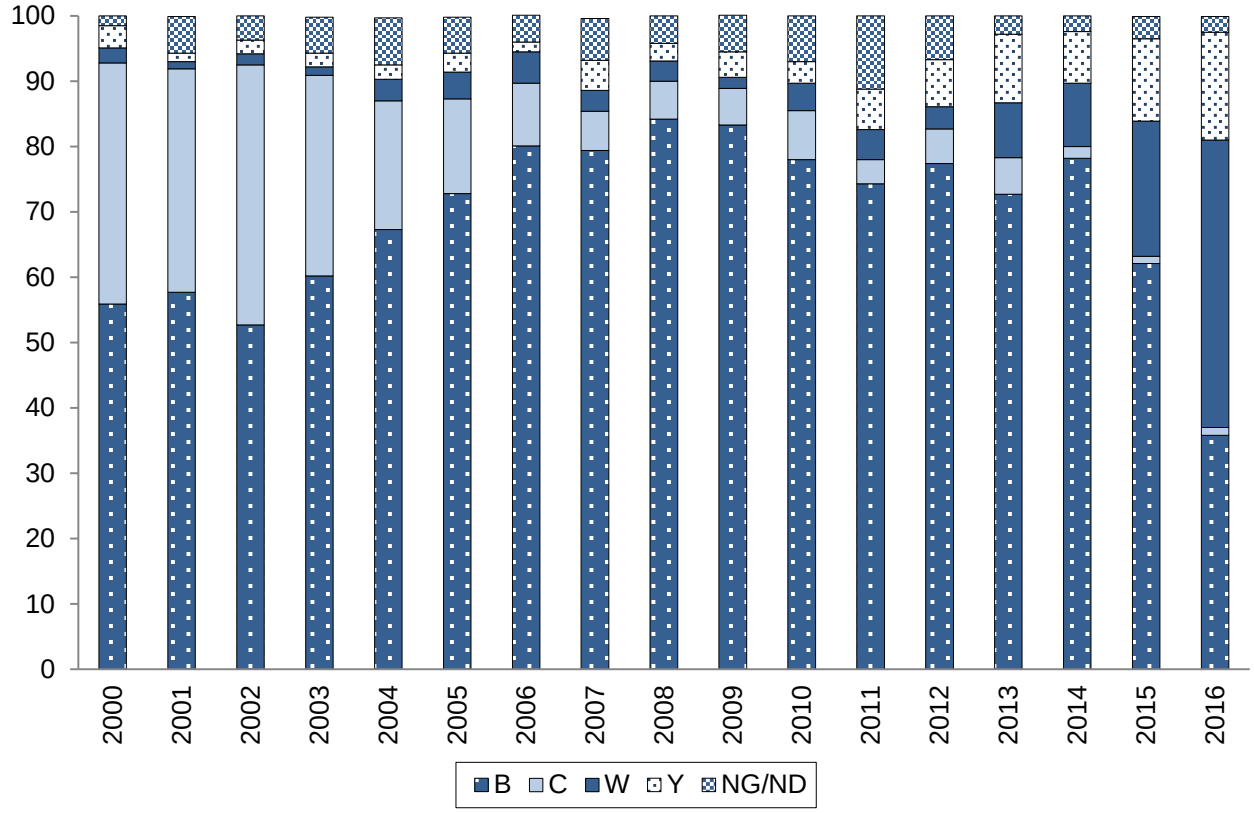
The number and proportion of cases of IMD caused by serogroup W in 2016 (107 cases, 44% of total IMD) was the highest reported by the AMSP, and was almost triple the number of cases reported in 2015 (36 cases), and a ten-fold increase in the average number of annual IMD cases caused by serogroup W reported before 2015. Prior to 2016, the proportion of cases of IMD caused by serogroup W ranged from 1–5% in the period 1997–2012, 8–10% in 2013–2014, to 21% of the total cases of IMD in 2015.

The number and proportion of cases of IMD caused by serogroup Y in 2016 (n=40, 17% of total IMD) was also the highest reported by the AMSP, almost double the number of IMD cases reported in the previous year (22 cases), and a four-fold increase in the average number of annual serogroup Y cases reported before 2015. Prior to 2016, the proportion of cases of IMD caused by serogroup Y ranged from 1–5% in the period 1997–2010, 6–11% in 2011–2014, and was 13% of the total cases of IMD in 2015.

Of the 107 cases of IMD cases caused by serogroup W in 2016, 47 cases (44%) were reported from Victoria, where serogroup W represented 62% (47/76) of cases, 23 cases (22%) were reported in New South Wales, representing 33% (23/69) cases and 14 cases (13%) were reported in Western Australia, representing 67% (14/21) cases. This is in contrast to previous years where, serogroup B has been predominant in these states. Serogroup W was reported in all jurisdictions, except in the Northern Territory in 2016.

Of the 40 cases of IMD caused by serogroup Y in 2016, 15 cases (38%) were reported from New South Wales, where this serogroup represented 22% (15/69 cases) of cases. Thirteencases (33%) were reported in Queensland, representing 31%

Figure 2: Proportion of serogroups of laboratory-confirmed invasive meningococcal disease, Australia, by year



(13/42 cases) of cases. Serogroup Y was reported in all jurisdictions except in the Northern Territory, Australian Capital Territory, and South Australia. Serogroup B was reported in all jurisdictions except in Tasmania, and continued to be the predominant serogroup amongst IMD cases reported in South Australia.

In 2016, the predominant serogroup for children less than 5 years continued to be serogroup B, however the proportion was the lowest since 2000. (Table 3, Figure 3). In young adults aged 20–24 years, IMD caused by serogroup B was also the predominant serogroup, however this proportion was lower than in 2014 (83%), 2007–2010 and 2012 (72-88%), but similar to 2011, 2013, and 2015 (62-67%). The proportion of IMD caused by serogroup B in the all other age groups was less compared with previous years, due to the large increase in the number of IMD cases caused by serogroup W and Y in these age groups.

In 2016, there was an increase in the number of IMD caused by serogroup W across all age groups except those aged 10–14 years. For those aged more than 45 years, IMD caused by serogroup W was the predominant serogroup (49/83 cases or 57%). This is in contrast with previous years, where serogroup Y was predominant for this age group. There was also a large increase in the number and proportion of IMD cases caused by serogroup W (16/35 cases, 46%) in those aged 25–44 years, compared with previous years, where serogroup B was predominant (68–87% since 2007).

Genotyping

In 2016, genotyping results were available for 93% (225/243) of IMD cases (Tables 5 and 6). The predominant *por*A genotype for IMD cases caused by serogroup B was P1.7-2,4 (25 cases, 33% of serogroup B that were typeable), which is similar to 2015 (Figure 4). The predominant *por*A genotype for serogroup Y IMD cases was P1.5-1,10-1 (34 cases, 85% of serogroup Y IMD cases that were typeable).

Figure 3: Number of serogroups B, Y and W cases of laboratory-confirmed invasive meningococcal disease, Australia, 2016, by age

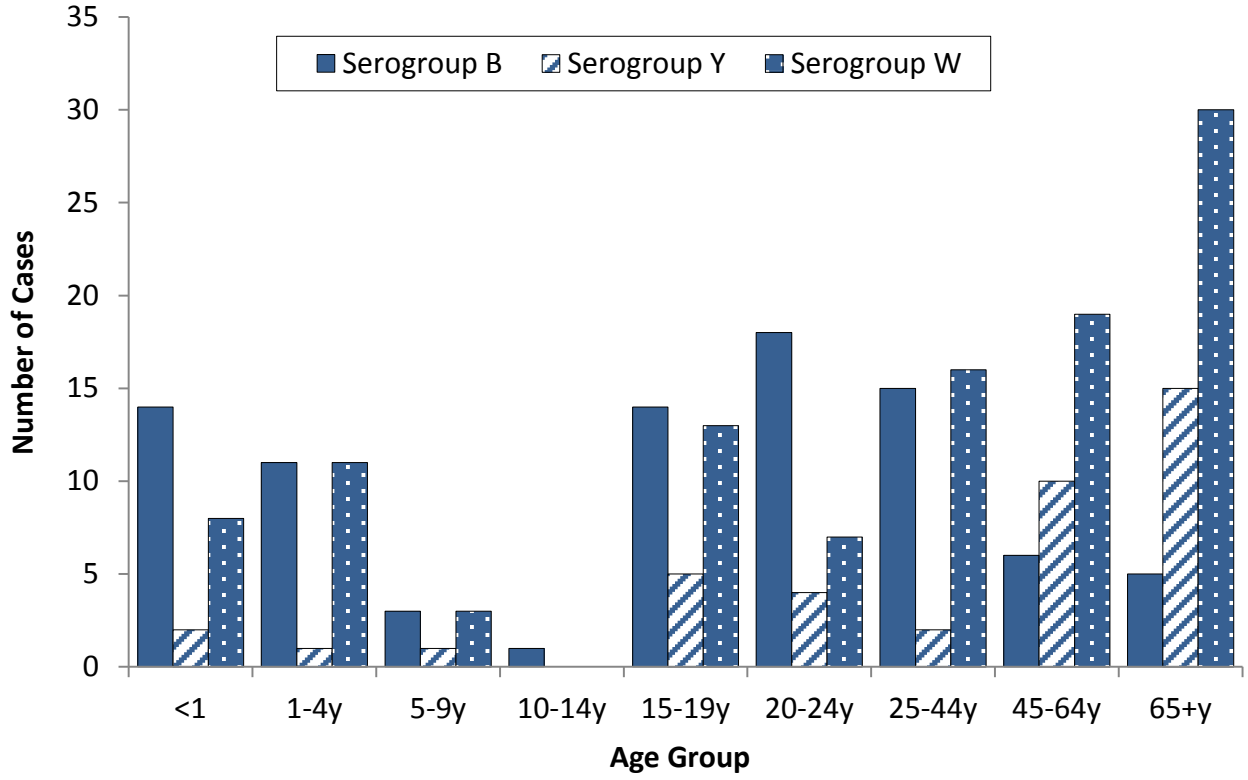


Figure 4: Number of porA genotypes for serogroup B in laboratory- confirmed cases of invasive meningococcal disease Australia, 2016

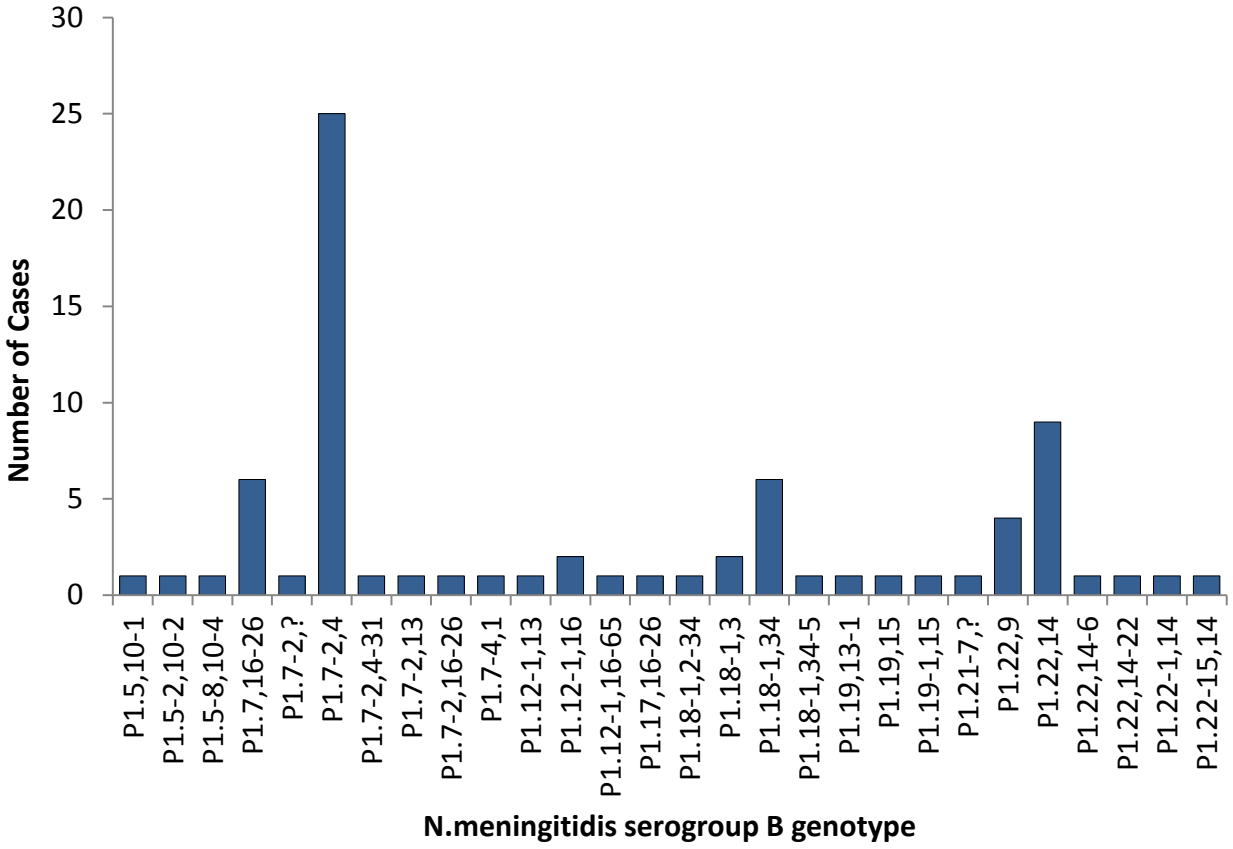


Table 5: Laboratory-confirmed cases of invasive meningococcal disease, Australia, 2016, by *por*A genotype

2016 AMSP	No. PER SEROGROUP					
GenotypeporA	**B**	**C**	**Y**	**W135**	**ND**	**TOTAL**
P1.5,2	0	2	0	96	2	**100**
P1.5,2-59	0	0	0	1	0	**1**
P1.5,10-1	1	0	0	0	0	**1**
P1.5-1,2-2	0	0	2	0	0	**2**
P1.5-1,10-1	0	0	34	0	0	**34**
P1.5-1,10-4	0	0	0	2	0	**2**
P1.5-2,10-1	0	0	3	0	0	**3**
P1.5-2,10-2	1	0	0	0	0	**1**
P1.5-2,10-29	0	0	1	0	0	**1**
P1.5-8,10-4	1	0	0	0	0	**1**
P1.5-9,2	0	0	0	1	0	**1**
P1.5-11,2	0	0	0	1	0	**1**
P1.7,16-26	6	0	0	0	1	**7**
P1.7-2,?	1	0	0	0	0	**1**
P1.7-2,4	25	0	0	0	0	**25**
P1.7-2,4-31	1	0	0	0	0	**1**
P1.7-2,13	1	0	0	0	0	**1**
P1.7-2,16-26	1	0	0	0	0	**1**
P1.7-4,1	1	0	0	0	0	**1**
P1.12-1,13	1	1	0	0	0	**2**
P1.12-1,16	2	0	0	0	0	**2**
P1.12-1,16-65	1	0	0	0	0	**1**
P1.17,16-26	1	0	0	0	0	**1**
P1.18-1,2-34	1	0	0	0	0	**1**
P1.18-1,3	2	0	0	3	0	**5**
P1.18-1,34	6	0	0	0	0	**6**
P1.18-1,34-5	1	0	0	0	0	**1**
P1.19,13-1	1	0	0	0	0	**1**
P1.19,15	1	0	0	0	0	**1**
P1.19-1,15	1	0	0	0	0	**1**
P1.21-7,?	1	0	0	0	0	**1**
P1.22,9	4	0	0	0	0	**4**
P1.22,14	9	0	0	0	0	**9**
P1.22,14-6	1	0	0	0	0	**1**
P1.22,14-22	1	0	0	0	0	**1**
P1.22-1,14	1	0	0	0	0	**1**
P1.22-15,14	1	0	0	0	0	**1**
TOTAL	75	3	40	104	3	**225**

Table 6: Distribution of *porA* genotype laboratory-confirmed cases of invasive meningococcal disease, Australia, 2016, by state or territory

Genotype *porA*	No. PER SEROGROUP PER STATE							
	NSW	QLD	VIC	SA	WA	ACT	TAS	NT
P1.5,2	21W, 1C	12W	44W, 1C, 1NG	4W	14W	1W	1W	
P1.5,2-59			1W					
P1.5,10-1				1B				
P1.5-1,2-2		1Y		1Y				
P1.5-1,10-1	15Y	10Y	8Y		1Y			
P1.5-1,10-4		2W						
P1.5-2,10-1		2Y	1Y					
P1.5-2,10-2		1B						
P1.5-2,10-29							1Y	
P1.5-8,10-4			1B					
P1.5-9,2	1W							
P1.5-11,2			1W					
P1.7,16-26		3B	4B					
P1.7-2,?	1B							
P1.7-2,4	3B	3B	3B	15B	1B			
P1.7-2,4-31			1B					
P1.7-2,13					1B			
P1.7-2,16-26		1B						
P1.7-4,1					1B			
P1.12-1,13	1C							1B
P1.12-1,16	2B							
P1.12-1,16-65		1B						
P1.17,16-26	1B							
P1.18-1,2-34			1B					
P1.18-1,3			1B				3W	1B
P1.18-1,34	4B	1B			1B			
P1.18-1,34-5			1B					
P1.19,13-1				1B				
P1.19,15		1B						
P1.19-1,15	1B							
P1.21-7,?	1B							
P1.22,9	2B		1B	1B				
P1.22,14	4B	2B	3B					
P1.22,14-6		1B						
P1.22,14-22	1B							
P1.22-1,14	1B							
P1.22-15,14			1B					

There were 105 serogroup W IMD cases that were able to be genotyped, 97 of these (92%), had the *por*A antigen encoding gene type P1.5,2. Multilocus sequence typing showed that, of these 72% (70/97) were sequence type (ST)-11 — the same strain type as the hypervirulent serogroup W strain reported in the UK and South America since 2009[4, 5] (Table 7).

Antibiotic susceptibility testing

Antimicrobial susceptibility testing was able to be performed for 77% (189/243) of the IMD cases in 2016. Of these, 6% (11/189) were resistant to penicillin (MIC ≥ 1 mg/L), the highest number and proportion of isolates with penicillin resistance reported by the AMSP. Only 4% of isolates tested were fully susceptible to penicillin (MIC ≤0.03 mg/L), and 90% (170/189) of isolates were less sensitive to penicillin (MIC=0.06–0.5 mg/L). Of the isolates that were resistant to penicillin, all were serogroup W, and 9/11 were P1.5,2: ST-11, one was P.1.5,2: ST-12351, and one was P.1.5,2-59: ST-11. This represented 11% penicillin resistance in all serogroup W isolates available for testing (n=99). There was one isolate that had an elevated MIC to ceftriaxone (MIC=0.125 mg/L), the highest recorded by the AMSP. This isolate was W:P1.5,2, ST-11 and was less sensitive to penicillin. All isolates tested were susceptible to rifampicin and ciprofloxacin.

Discussion

In 2016, there were 243 cases of laboratory confirmed IMD, representing 96% of the number of notifications to the NNDSS.[2] The number of laboratory-confirmed IMD in 2016 represents a 40% increase cases compared with the previous year. The number of IMD cases has been steadily rising since the nadir in 2013, however the number of cases reported this year remains less than half of the peak number of IMD cases reported in Australia in 2002 (n=684). The introduction of the serogroup C vaccine to the national immunisation schedule in 2003 has resulted in a very large and sustained reduction in the number and proportion of serogroup C IMD cases in this country, and in 2015 the number and proportion of IMD cases caused by serogroup C was the lowest ever reported by the AMSP. In early 2014, a recombinant multicomponent meningococcal B vaccine became available in Australia. [6] This vaccine is not on the national immunisation program but is available for purchase privately. Therefore uptake is elective and the impact of its introduction is yet to be determined in this country.

A number of changes in IMD epidemiology were observed in Australia in 2016. There was a notable increase in the number and proportion of IMD cases caused by serogroup W and serogroup Y, and a marked decrease in the number and proportion of cases caused by serogroup B. The incidence of serogroup W and serogroup Y was the highest ever reported by the AMSP.

In addition, as reported by the AMSP in 2015, the primary peak of IMD was observed in adults aged 45 years or older and was due to the increased number of IMD cases caused by serogroup W and serogroup Y in this age group. However these serogroups also increased in frequency in all age groups in 2016. Secondary disease peaks were observed in those aged less than 5 years, and in adolescents aged 15–19 years.

Serogroup W represented 44% of all laboratory-confirmed IMD cases in 2016, with the highest proportions in Victoria (44%), Queensland (33%), New South Wales (22%) and Western Australia (13%). Typing of these isolates found that the predominant circulating strain of serogroup W, had the *por*A antigen encoding gene type P1.5,2 and was sequence type (ST)-11. This is same genotype as the hypervirulent serogroup W strain that emerged in the United Kingdom and South America in 2009[4, 7] and has spread to now account for 25% of IMD in the UK in 2014/15, and 59% of all cases in Chile in 2012. This serogroup W strain is now considered endemic in these regions and is associated with atypical presentations, more severe clinical disease and a higher case fatality rate.[7] The initial increase in serogroup W in these regions, as is now being demonstrated in Australia, was seen in older adults, but was subsequently reported in all age groups, particularly in adolescents and

Table 7: Laboratory-confirmed cases of serogroup W IMD, Australia, 2016, by sequence type (ST)

Sequence Type	W Genotype							
	P1.5,2	P1.5,2-59	P1.5-1,10-4	P1.5-9,2	P1.5-11,2	P1.18-1,3	Not typeable	Total
ST11	70	1	0	0	0	0	0	**71**
ST22	0	0	0	0	0	3	0	**3**
ST23	0	0	2	0	0	0	0	**2**
ST1287	7	0	0	0	0	0	0	**7**
ST3298	1	0	0	0	0	0	0	**1**
ST8345	1	0	0	0	0	0	0	**1**
ST8857	0	0	0	0	1	0	0	**1**
ST10651	0	0	0	1	0	0	0	**1**
ST12351	8	0	0	0	0	0	0	**8**
ST12590	1	0	0	0	0	0	0	**1**
Not typeable	9	0	0	0	0	0	2	**11**
Total	**97**	**1**	**2**	**1**	**1**	**3**	**2**	**107**

infants[8]. In response, vaccination programs have been implemented in both the United Kingdom and in Chile [4, 9]

An increase in IMD cases caused by serogroup Y was also observed in the eastern states of New South Wales, Queensland and Victoria in 2016. The predominant serogroup Y genotype (85%, 34/40) was P1.5-1,10-1, which increased in number and proportion compared with 2015 (70%, 14/20) and 2014 (50%,6/12) whereas in previous years the serogroup Y genotype distribution was more heterogeneous. The emergence of serogroup Y and genotype Y:P1.5-1,10-1 has also been reported recently in Europe [10]. The phenotypic and genotypic characterization of the serogroup Y isolates is ongoing by the NNN.

Antimicrobial susceptibility testing of IMD isolates in 2016 showed an increase in penicillin resistance, the highest annual number and proportion recorded by the AMSP. The incidence of penicillin resistance in *N. meningitidis* in Australia has been less than 1% annually of IMD isolates tested in 1996-2014, rising to 3% in 2015, and 6% in 2016. The proportion of IMD isolates with penicillin MIC values in the less sensitive category has been increasing in recent years. These proportions ranged from 62–75% in 1996–2006; 67%–79% in 2007–2009; 78%-88% in 2010–2015, and in 2016, was 90%. In 2016, all isolates resistant to penicillin were serogroup W, representing 11% of all serogroup W isolates tested. Additionally, there was another serogroup W isolate with the highest ceftriaxone MIC recorded (0.125mg/L) by the AMSP. All IMD isolates were susceptible to rifampicin and ciprofloxacin.

The increase in IMD cases caused by serogroup W and serogroup Y, and the observed increase in antimicrobial resistance in serogroup W isolates are of significant concern. The NNN is continuing to lead further investigations with the Department of Health and the CDNA in to these observed changes and is closely monitoring the phenotypic and genotypic features of *N. meningitidis* causing IMD in Australia. Additional investigations including whole genome sequencing are in place to enhance IMD surveillance. The AMSP data are used for informing treatment guidelines and disease prevention strategies; and to monitor the effect of interventions.

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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

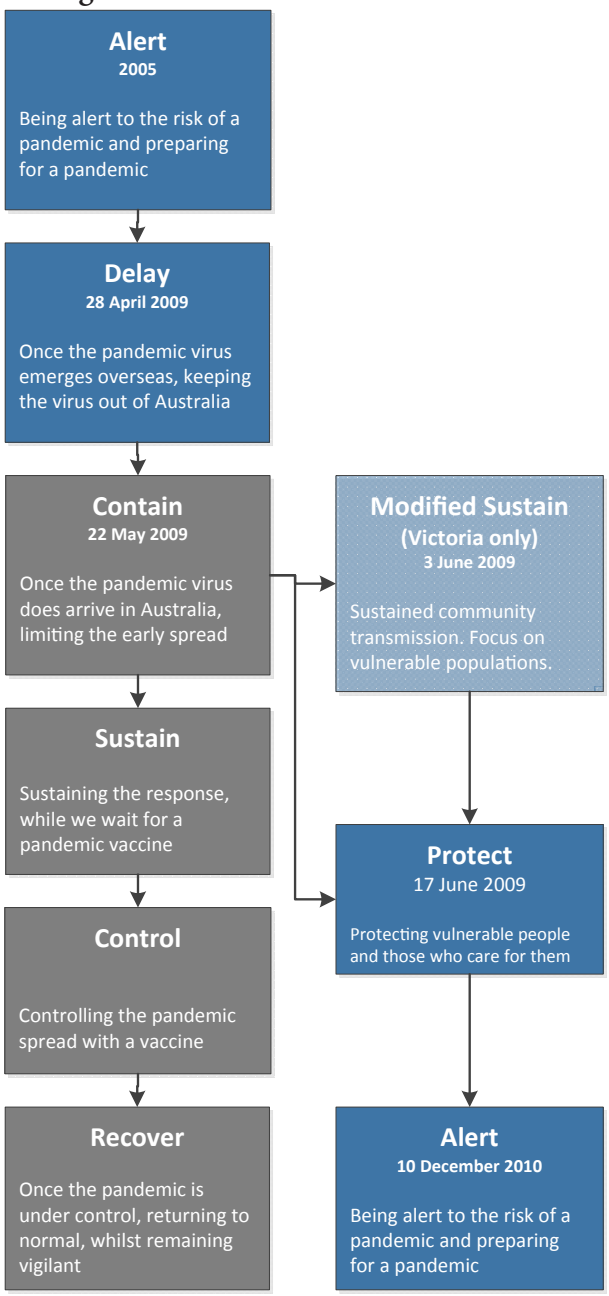
In March 2009, cases of a novel influenza virus began to emerge in Mexico and the United States of America. By mid-April 2009, atypical cases and clusters of severe pneumonia were occurring mainly among previously healthy young people in different areas of Mexico.[6] These cases were subsequently determined 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

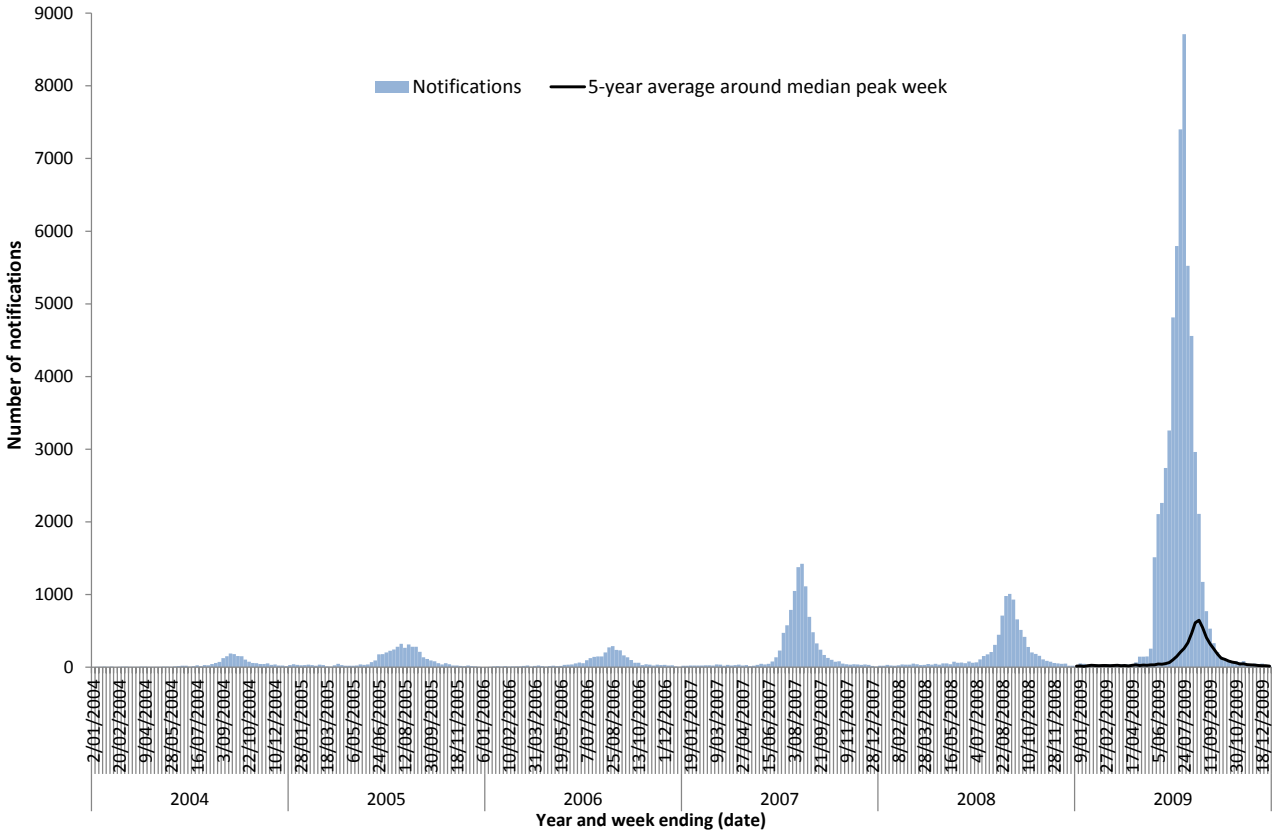
Generally the influenza season in Australia commences between late June and early July, peaking in mid-August and lasting on average approximately 17 weeks (Figure 2). In comparison, the 2009 influenza season began in early May following the emergence of the pandemic influenza virus overseas in late 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).

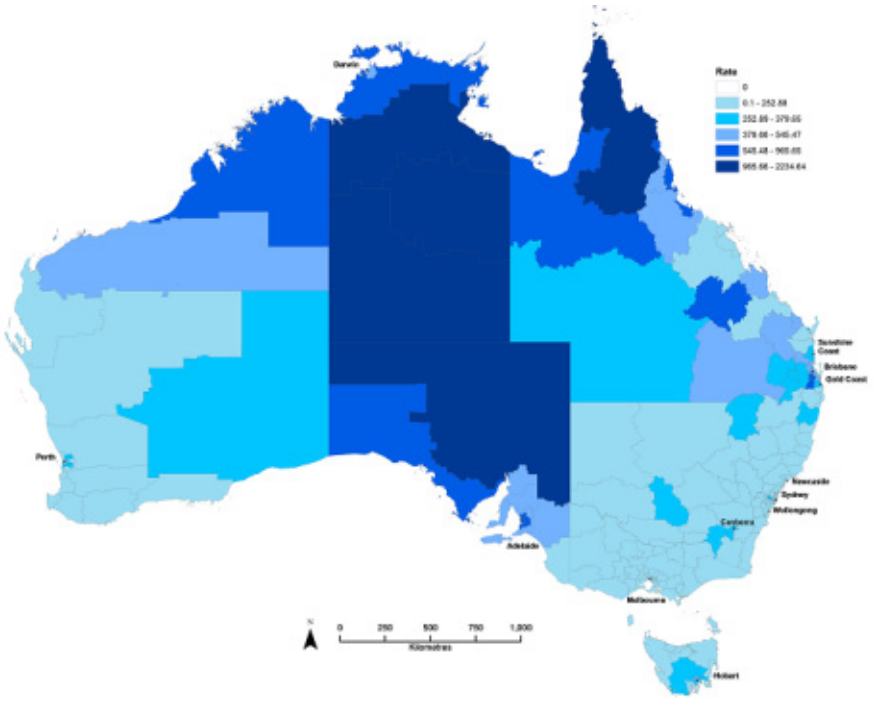
The timing and size of influenza activity increases and peaks varied across states and territories in 2009 (Figure 4). For example, Victoria experienced an overwhelming number of influenza cases from late May, much earlier

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

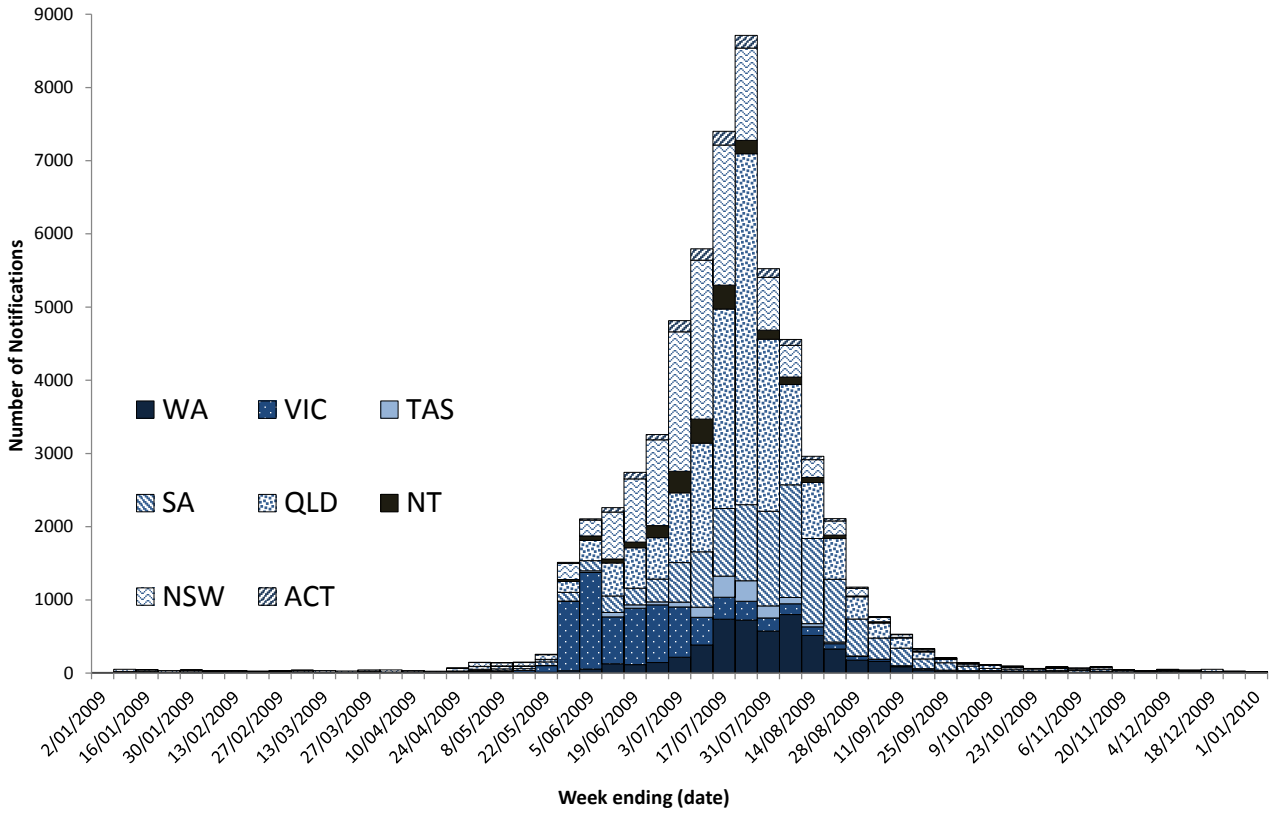
than other jurisdictions and, as a result, testing recommendations were focussed towards vulnerable individuals; therefore resulting in an overall relative reduction in the number of notifications compared to other jurisdictions.[9]

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.

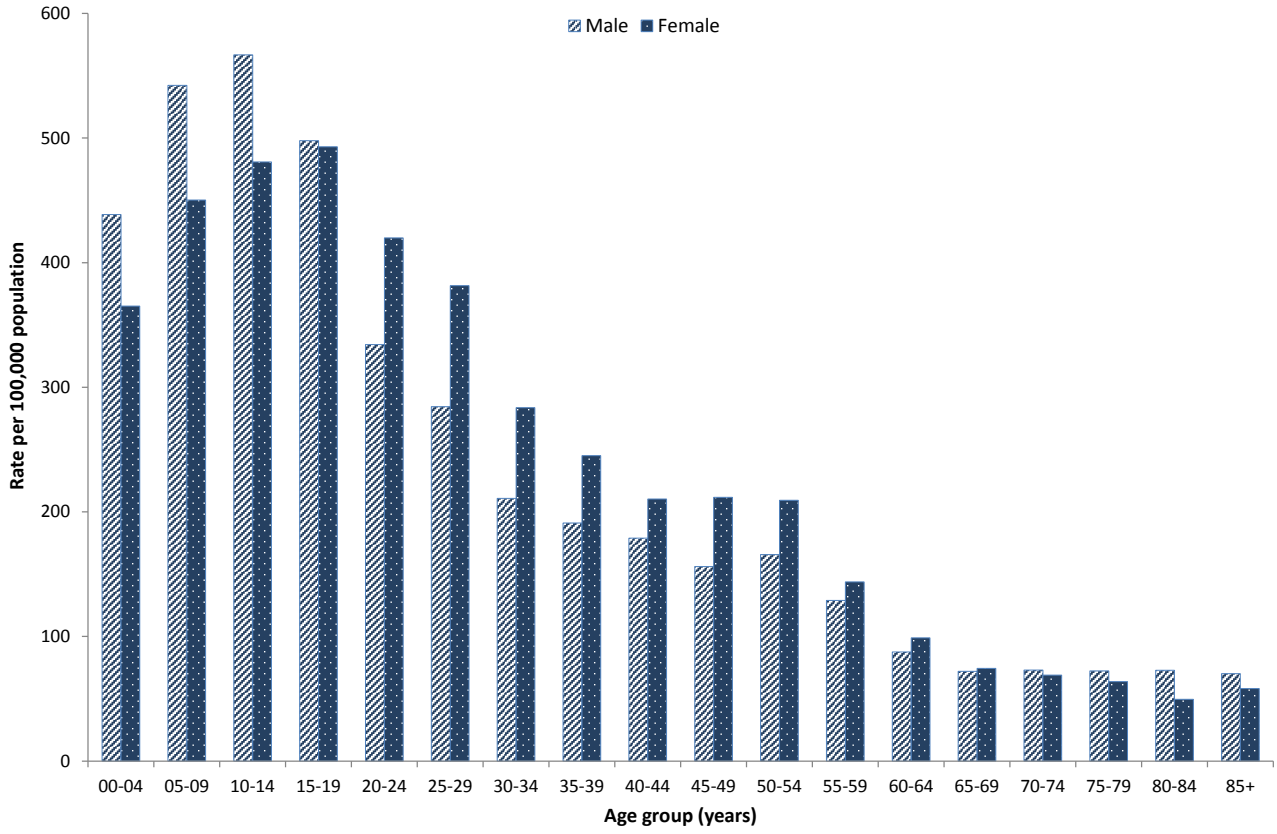
The median age of notifications in 2009 was 21 years (IQR 11-38), which is lower than the median age (26 years) among notifications for the period 2004-2008. Age-specific notifications

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



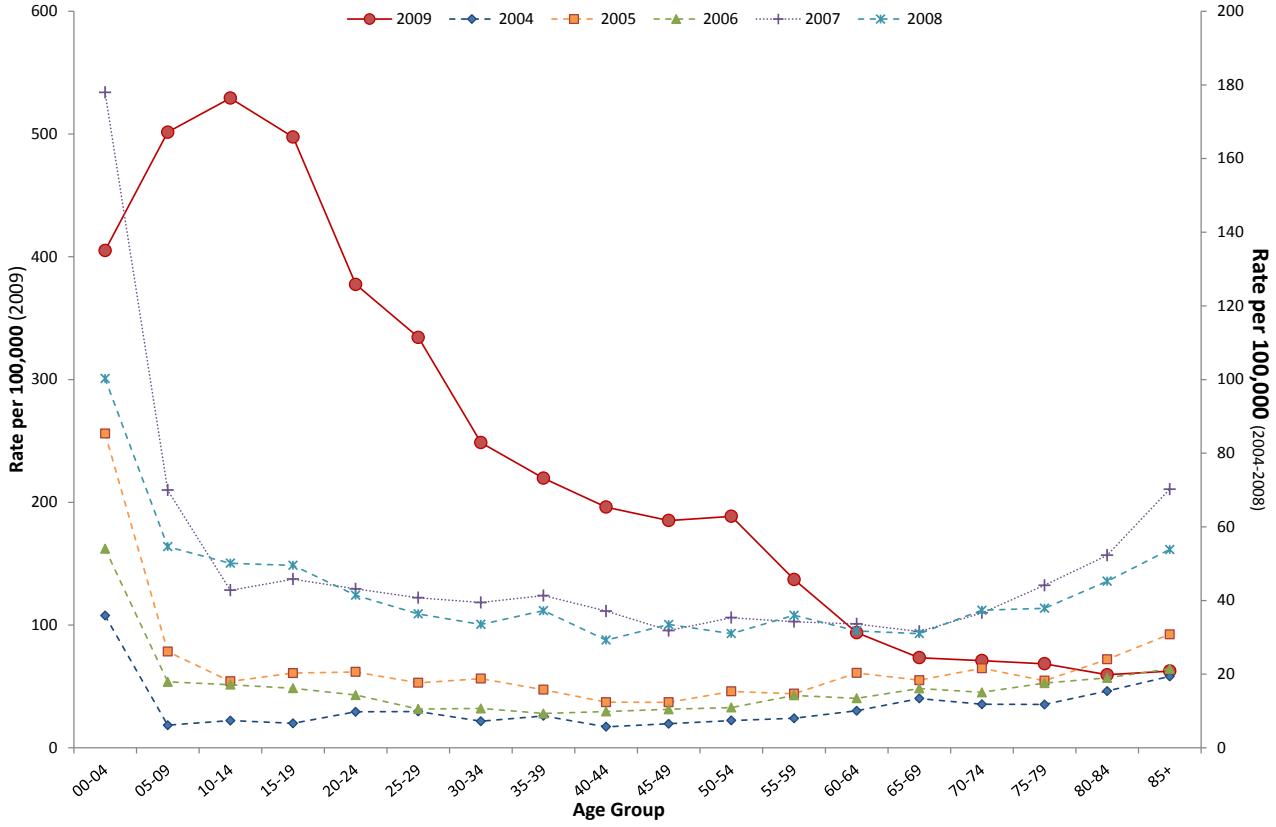
Source: NNDSS

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



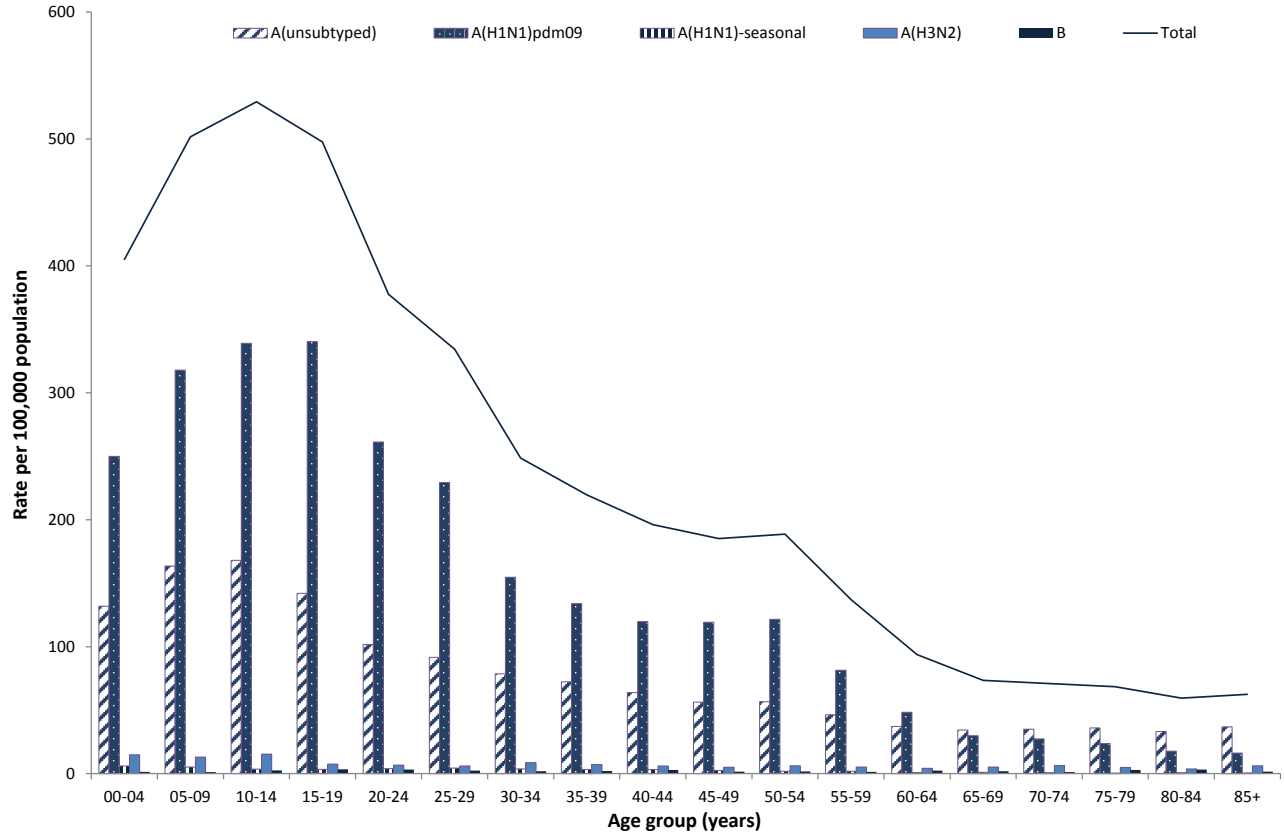
Source: NNDSS *Excludes 427 cases where age and/or sex were not reported.

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

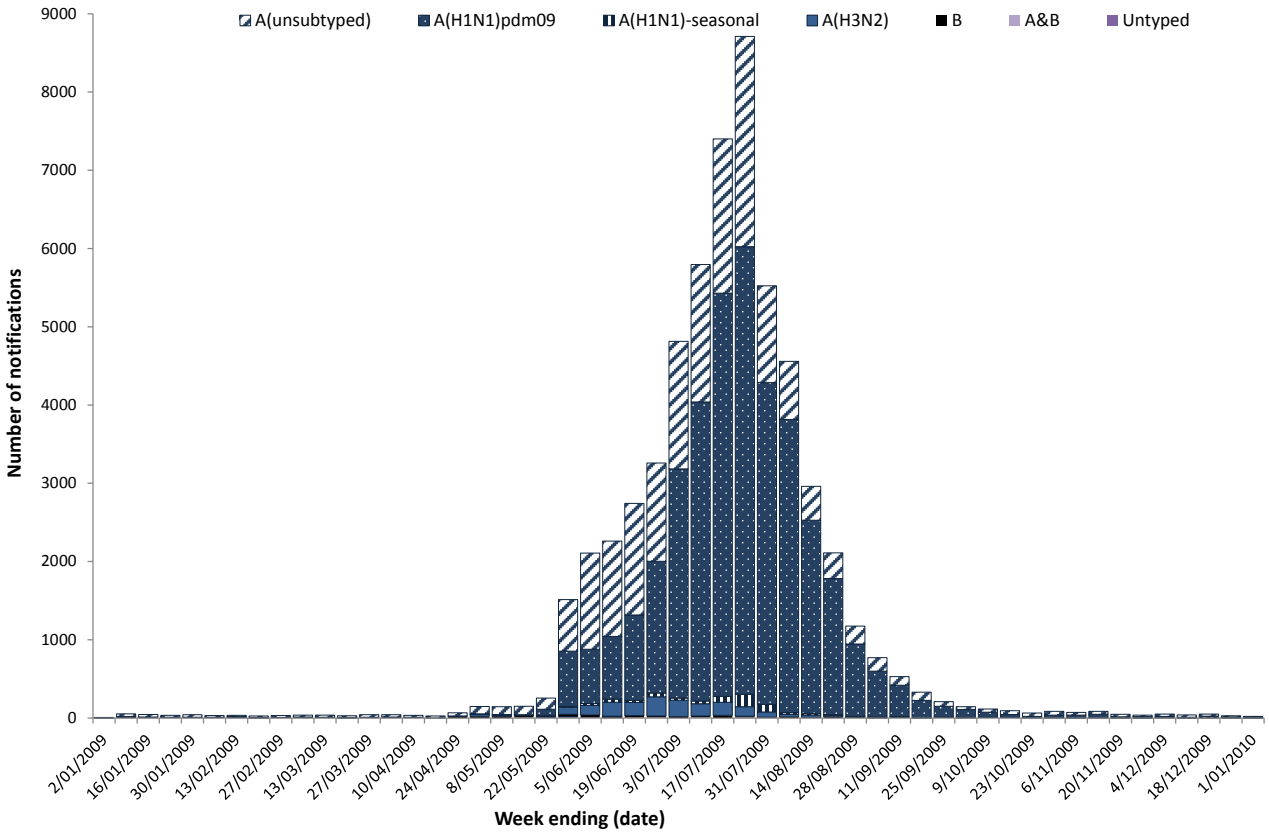
Figure 7: Rates of laboratory confirmed influenza, Australia, 2009, by subtype and age group*



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).

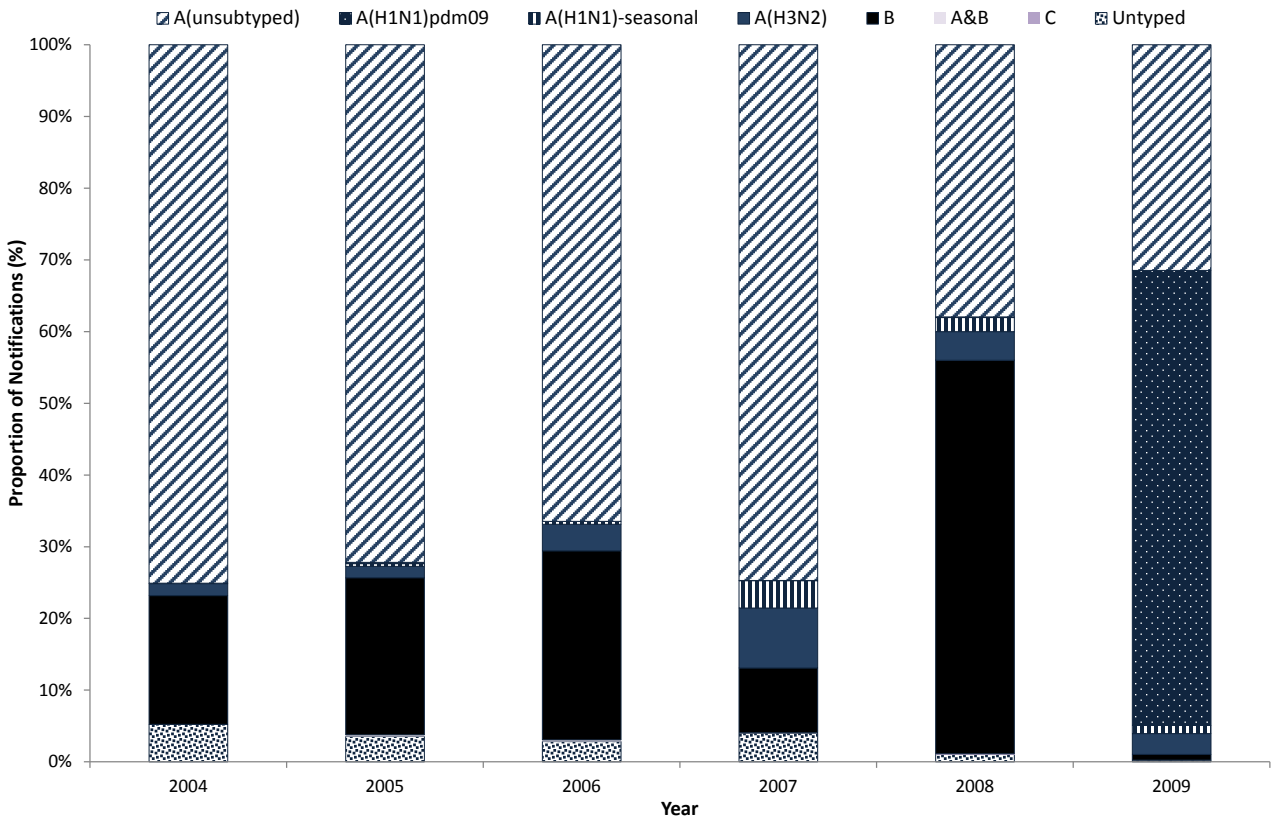
As a proportion, in 2009 there was a very small amount of influenza B infections (1%) reported compared with previous years where influenza B represented between 9 and 55% of overall notifications (Figure 9). Analysis of influenza A

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

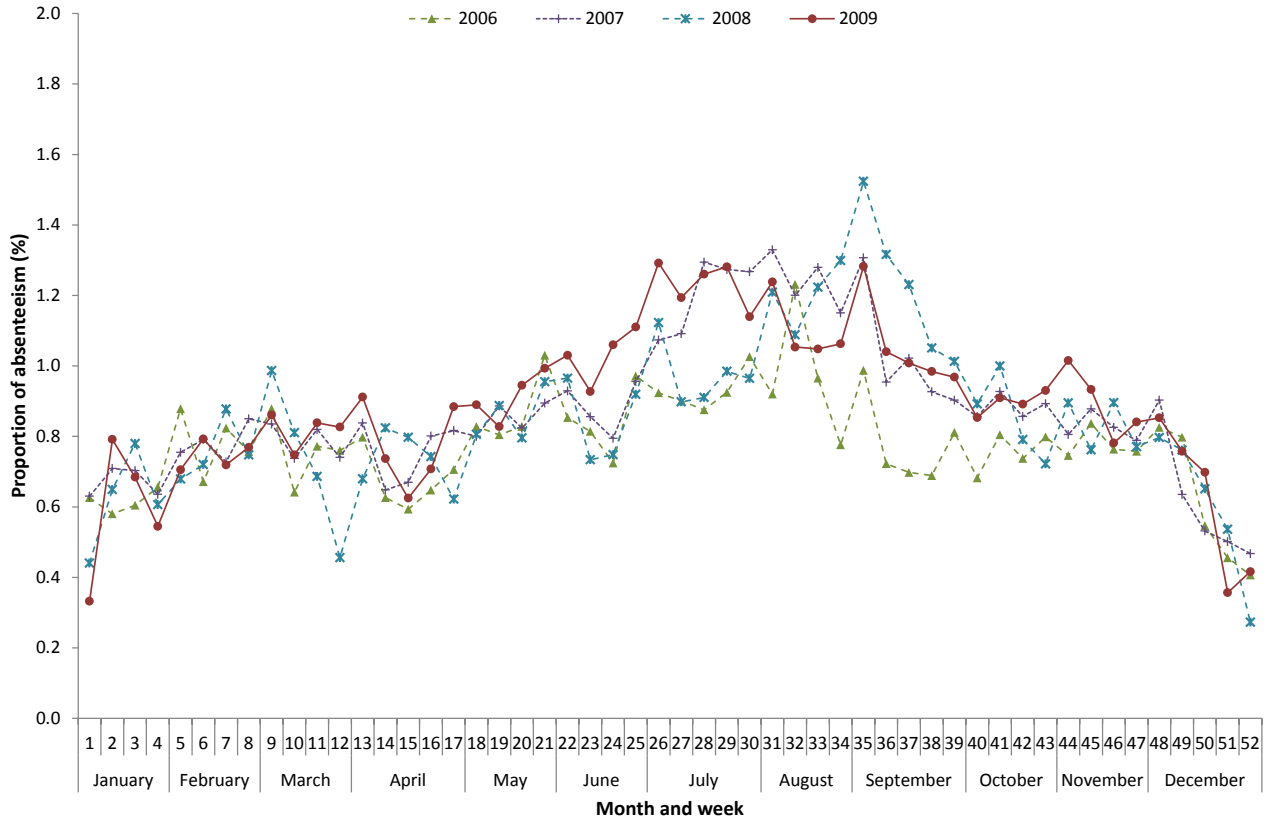
Community level influenza-like illness surveillance

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.

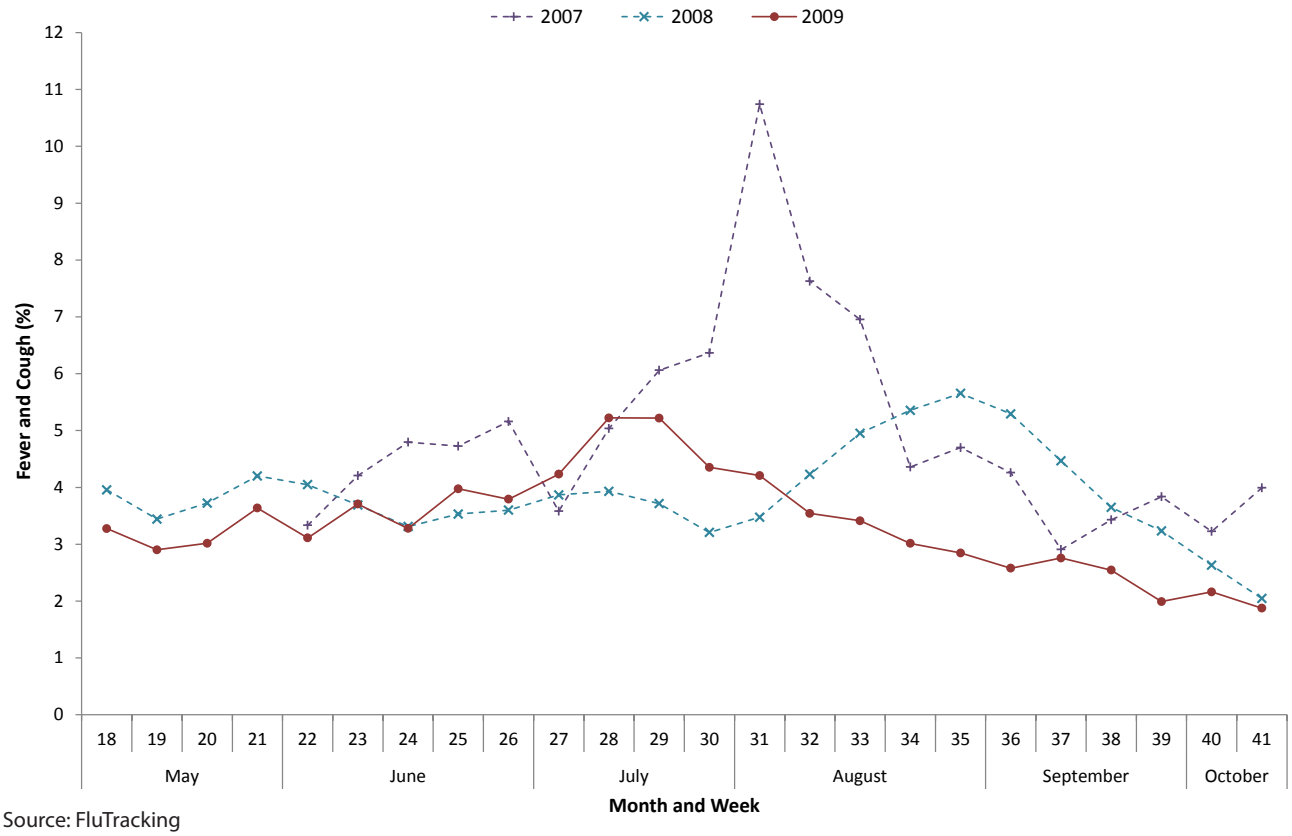
The FluTracking weekly online survey for ILI among participating community members in 2009 showed that rates of fever and cough among participants essentially peaked in mid-July at 5.2% and peaked much earlier than in 2007 and 2008 (Figure 11). Overall however, peak cough

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



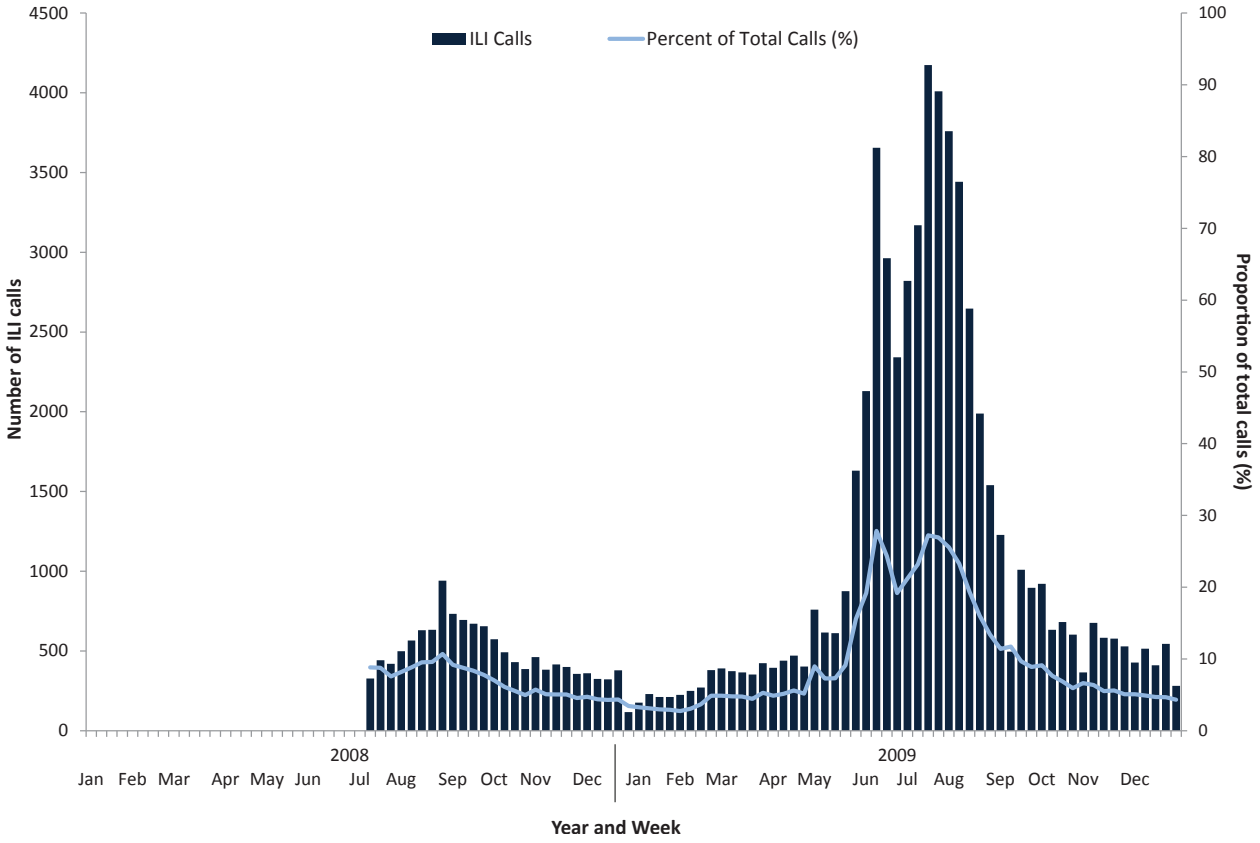
Source: A national employer (not disclosed)

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.

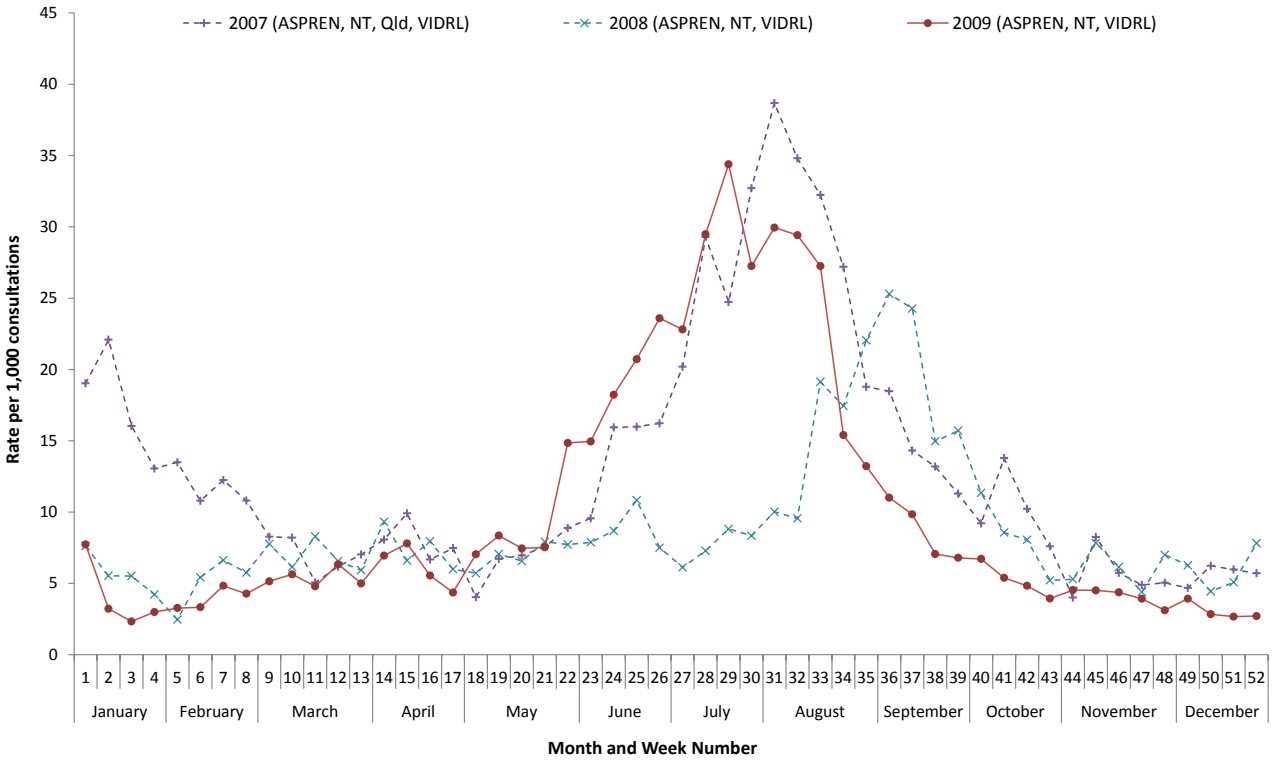
The number and proportion of calls to the National Health Call Centre Network (NHCCN) started to increase in early May 2009, had an initial peak in the week ending 14 June 2009 (27.8% of total calls; n=3,655 calls), followed by a peak in the week ending 19 July 2009 (4,174 calls; 27.2% of total calls) (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.

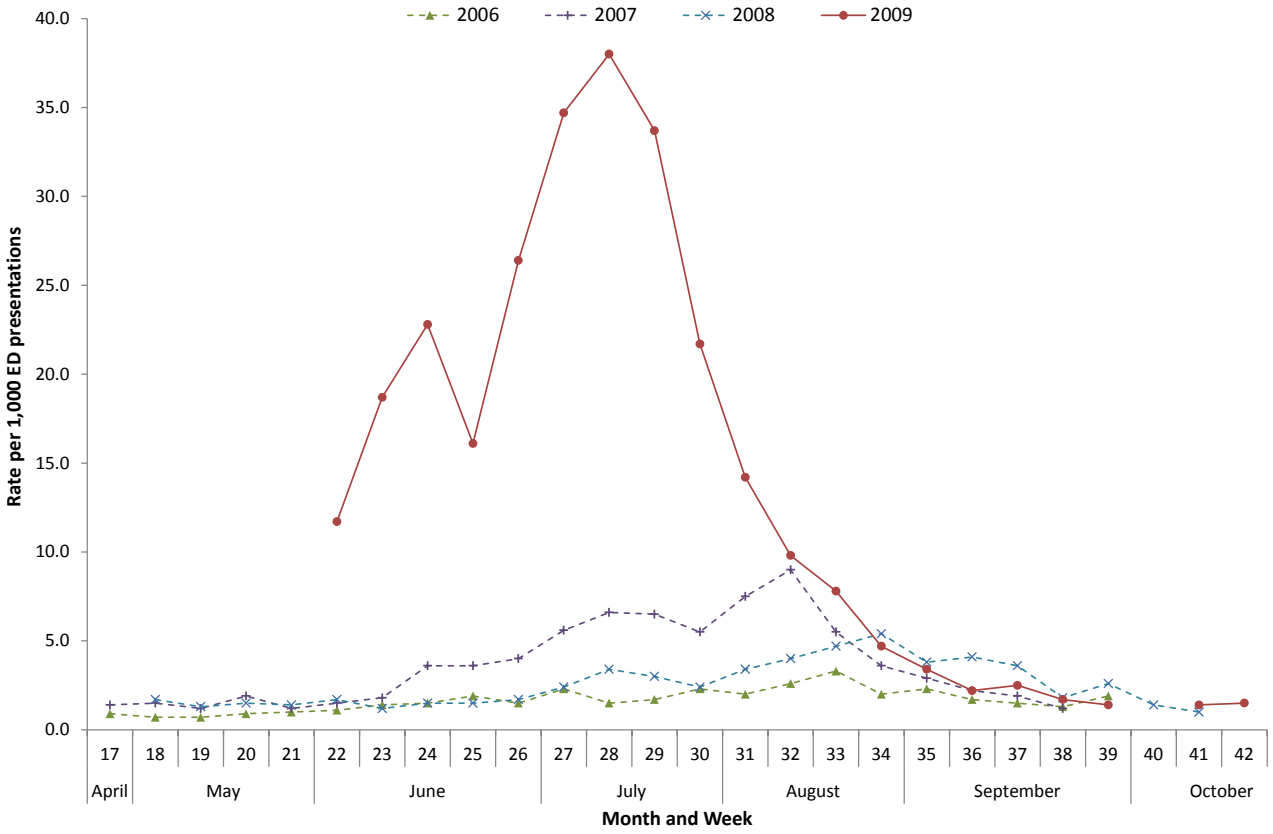
In terms of the 2009 ILI consultation rates, it is difficult to measure whether the initial community concern associated with the emergence of the pandemic influenza virus might have led to increased presentations to general practitioners, especially in people who may not have normally sought medical 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

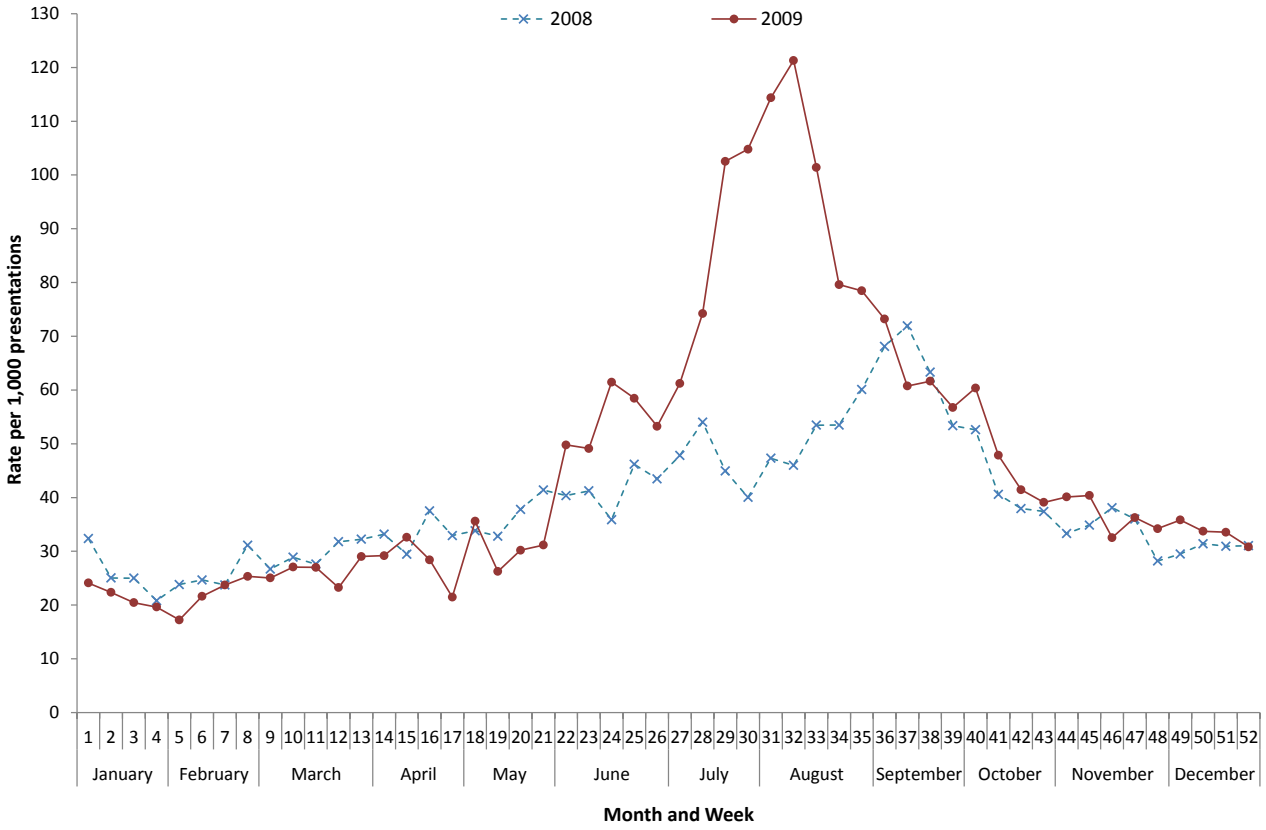
Admitted Patient Care Dataset

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

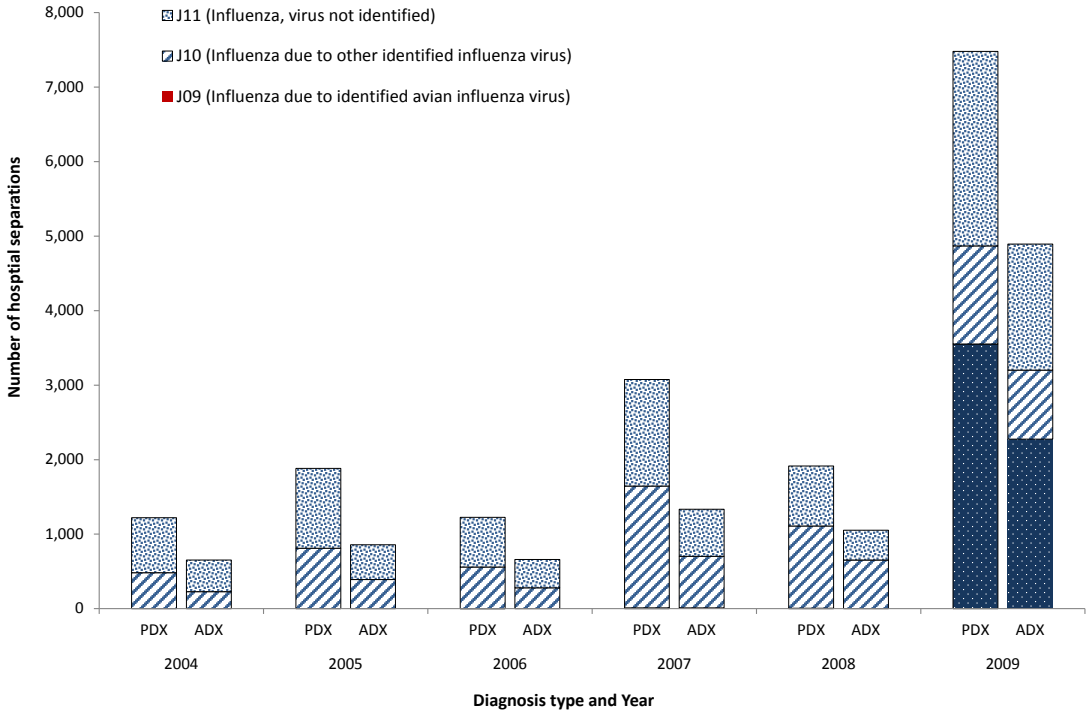
2,776 (range 1,876-2,966) respectively (Figure 16). In comparison, in 2009 there was almost a five-fold increase (n=8,069) in the number of separations for virologically confirmed influenza and a three-fold increase (n=12,374) for influenza with or without virological confirmation. Additionally the ratio 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).

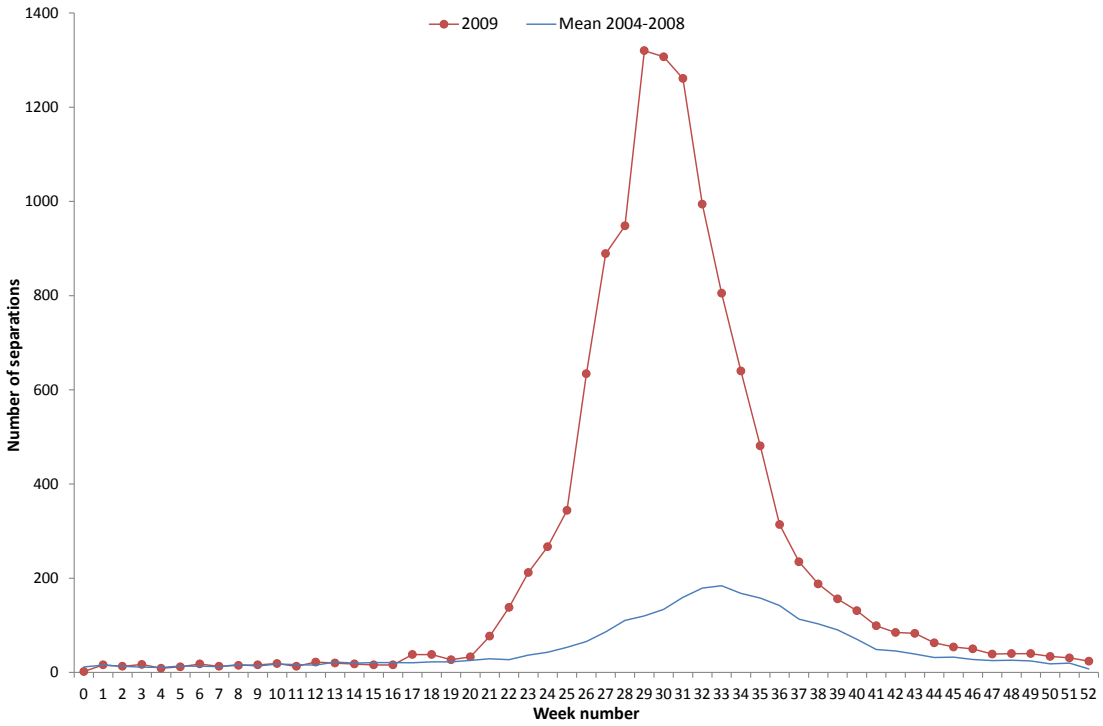
Between 2004 and 2009, hospital separations remained highest for children aged less than 5 years (Figure 18). The age-specific hospitalisation incidence profile suggests that hospitalisation

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

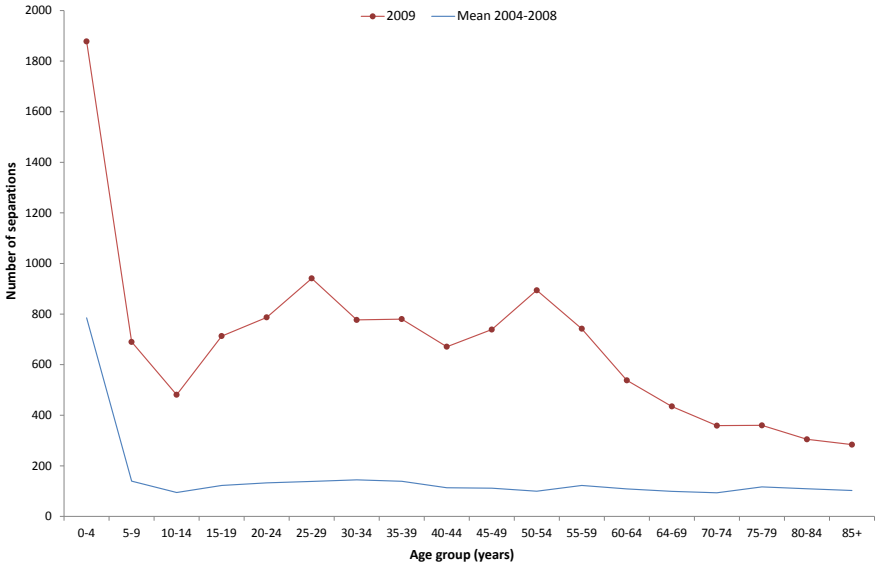
Figure 17: Number of hospital separations for all diagnoses* ICD-10-AM coded as J09, J10 or J11, Australia, 2004-2009, by week of separation



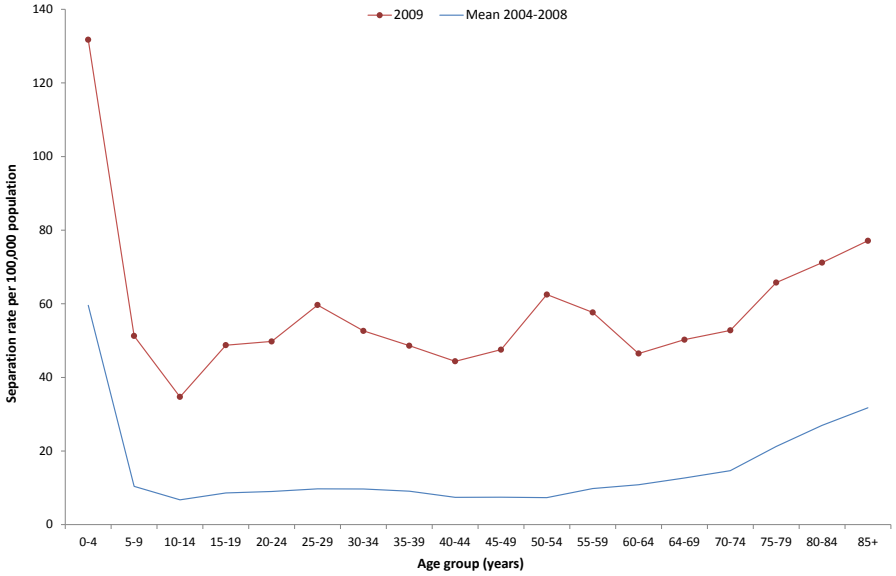
Source: Admitted Patient Care Data collection 2003-04 to 2009-10
*Includes both principal diagnosis and additional diagnoses. Where principal diagnosis is the diagnosis established after study to be chiefly responsible for occasioning a patient's service event or episode, as represented by a code; and 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

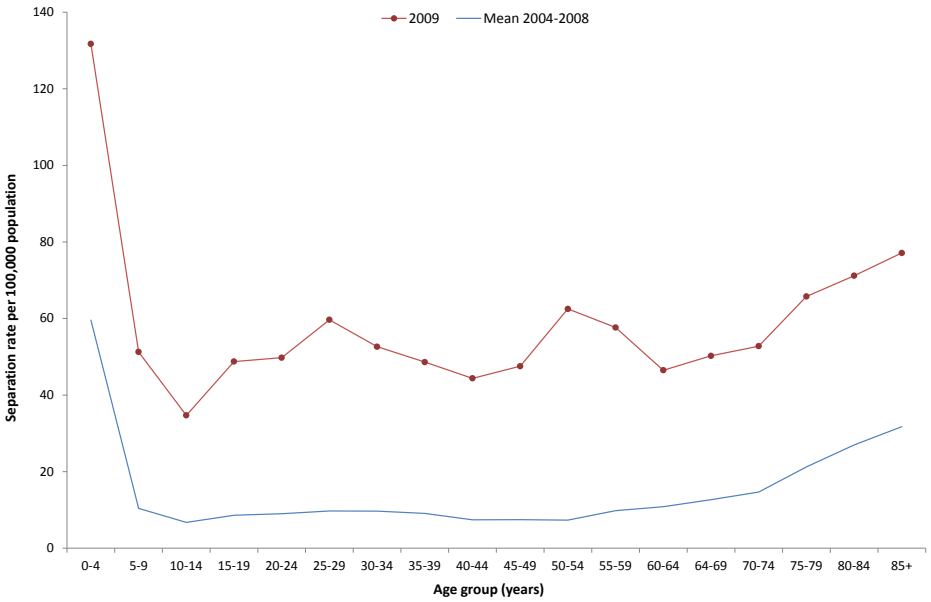
(i) Number of hospital separations.



(ii) Rate per 1,000 separations.

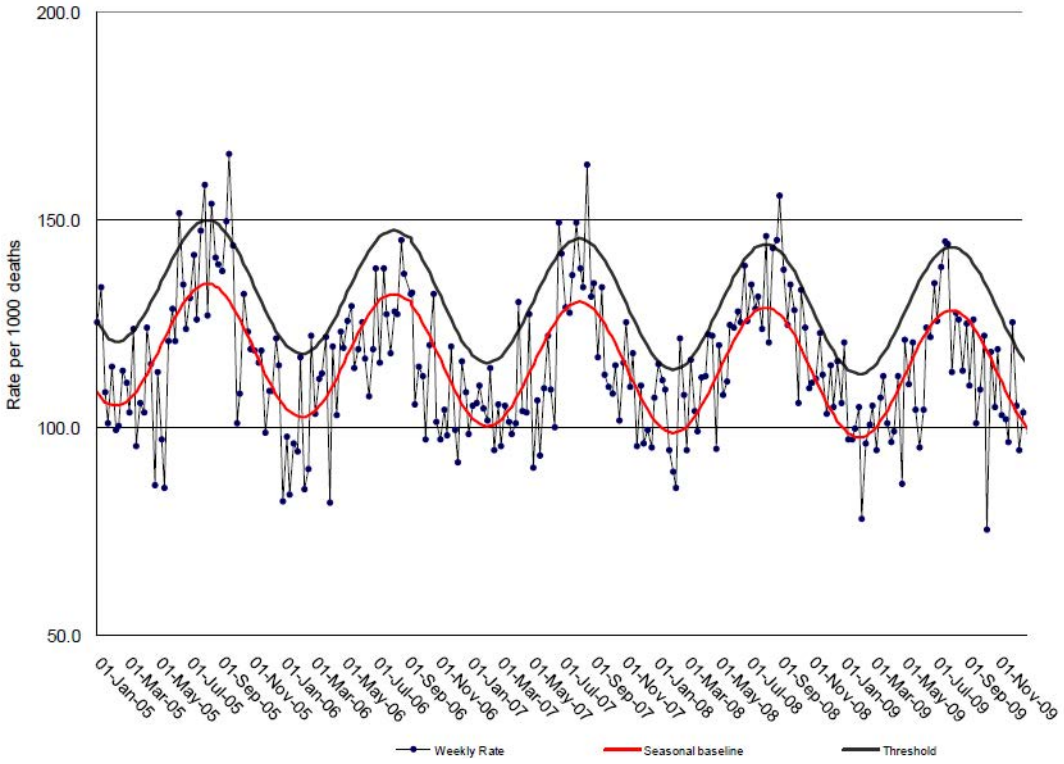


(iii) Rate per 100,000 population.
Source: Admitted Patient Care Data collection 2003-04 to 2009-10



*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

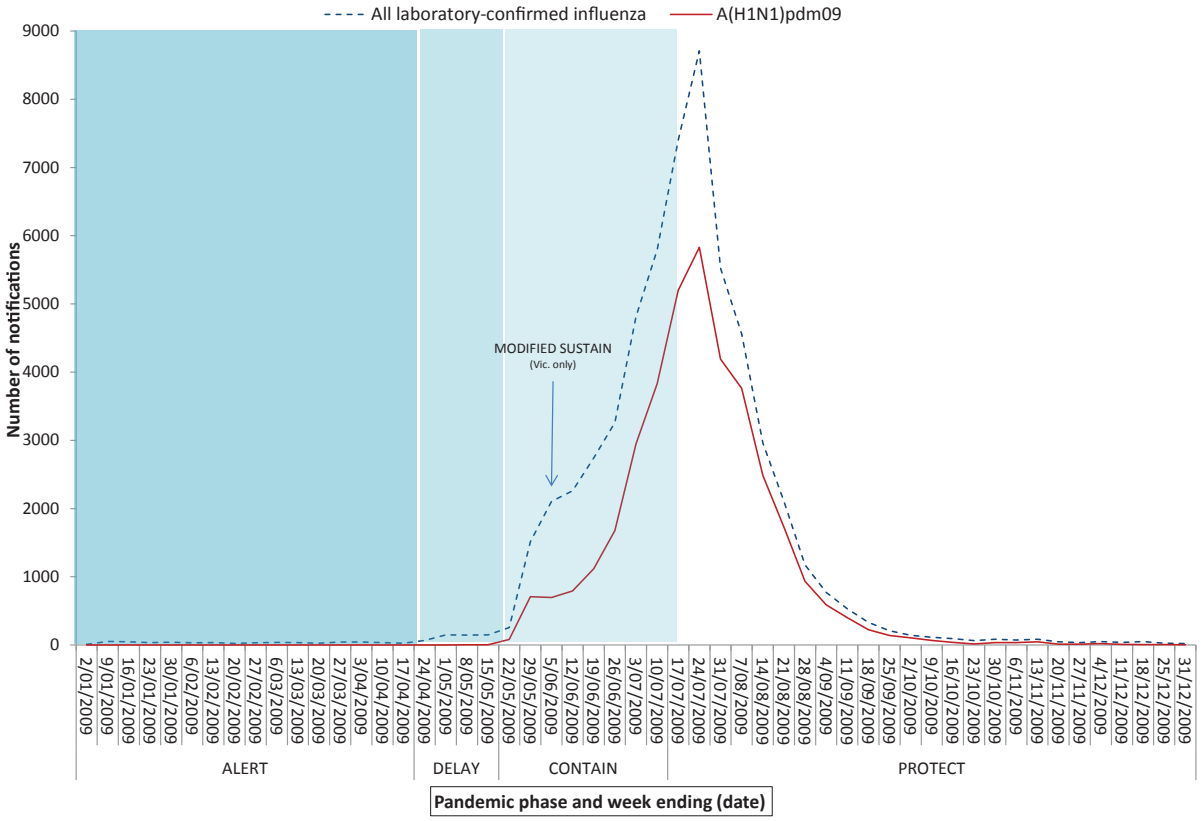
Indirect information derived from New South Wales Registry of Births, Deaths and Marriages death certificate surveillance, indicated that rates of deaths from influenza and pneumonia peaked and slightly exceeded the epidemic threshold during July 2009 at approximately 145 deaths per 1,000 NSW population (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).

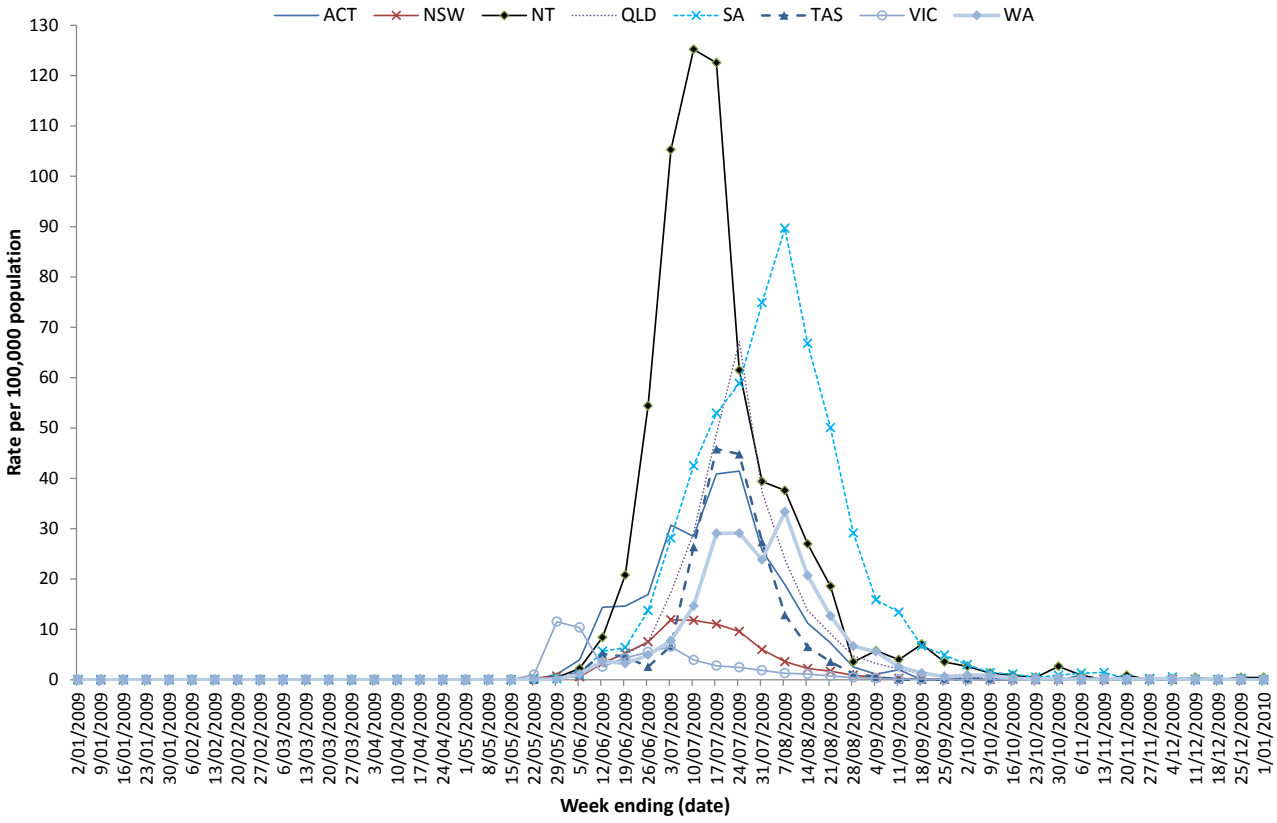
Some commentary is provided on analyses of the enhanced data during the PROTECT phase (17 June to 31 December 2009), where case ascertainment strategies were focussed towards those at risk for severe disease or those with severe disease and the conclusions are supported by sentinel systems that continued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

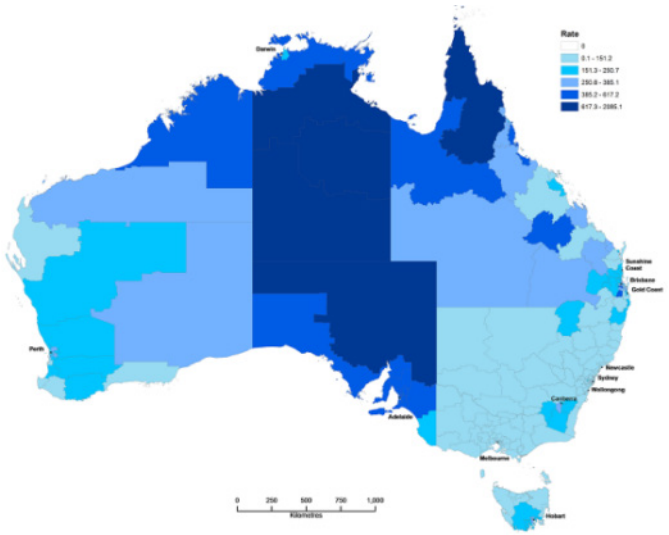
The first case of confirmed influenza A(H1N1) pdm09 infection in Australia was notified on 7 May 2009 in a traveller who had returned from the United States of America to Queensland. Following the detection of this imported case, the first case of locally acquired infection was identified on 16 May 2009. 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



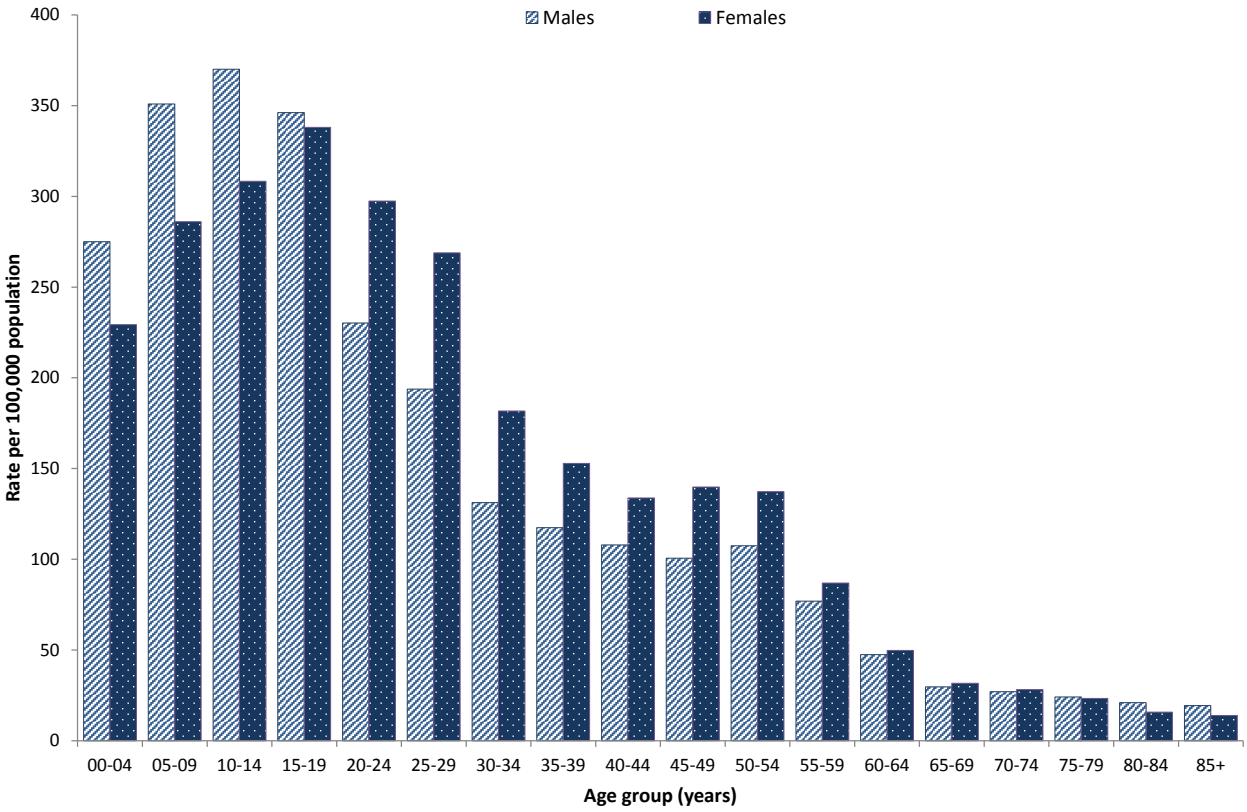
Source: NetEpi and NNDSS

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

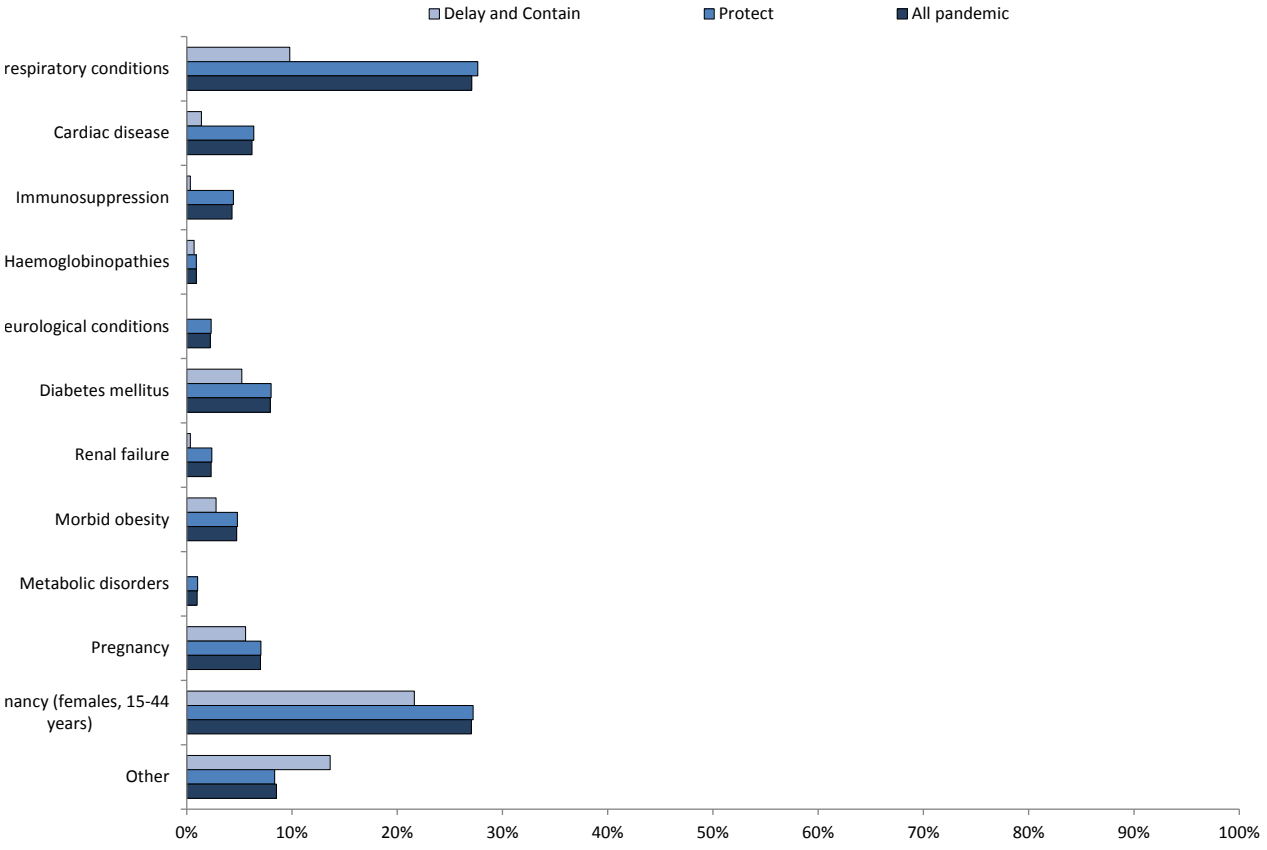
Whilst the main source of the virus' initial introduction into Australia is likely to have been through air travel; cruise ships[50] were identified as an important source of potential virus introduction, especially given that the duration of cruises can allow for multiple generations of influenza infections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

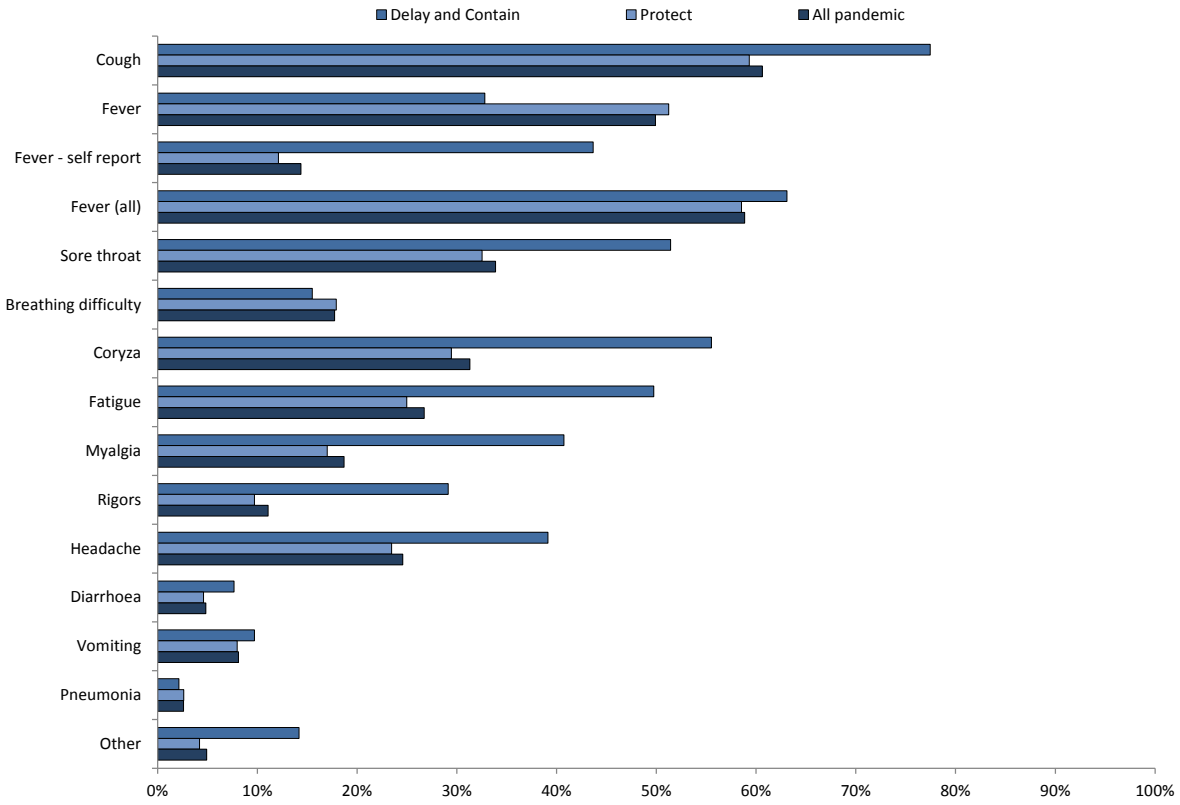
The most commonly reported underlying medical condition during these early phases were chronic respiratory conditions (9.8%), which included asthma and chronic obstructive pulmonary disease, followed by diabetes mellitus (5.2%). A total of 16 cases were pregnant (21.6%

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 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 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.

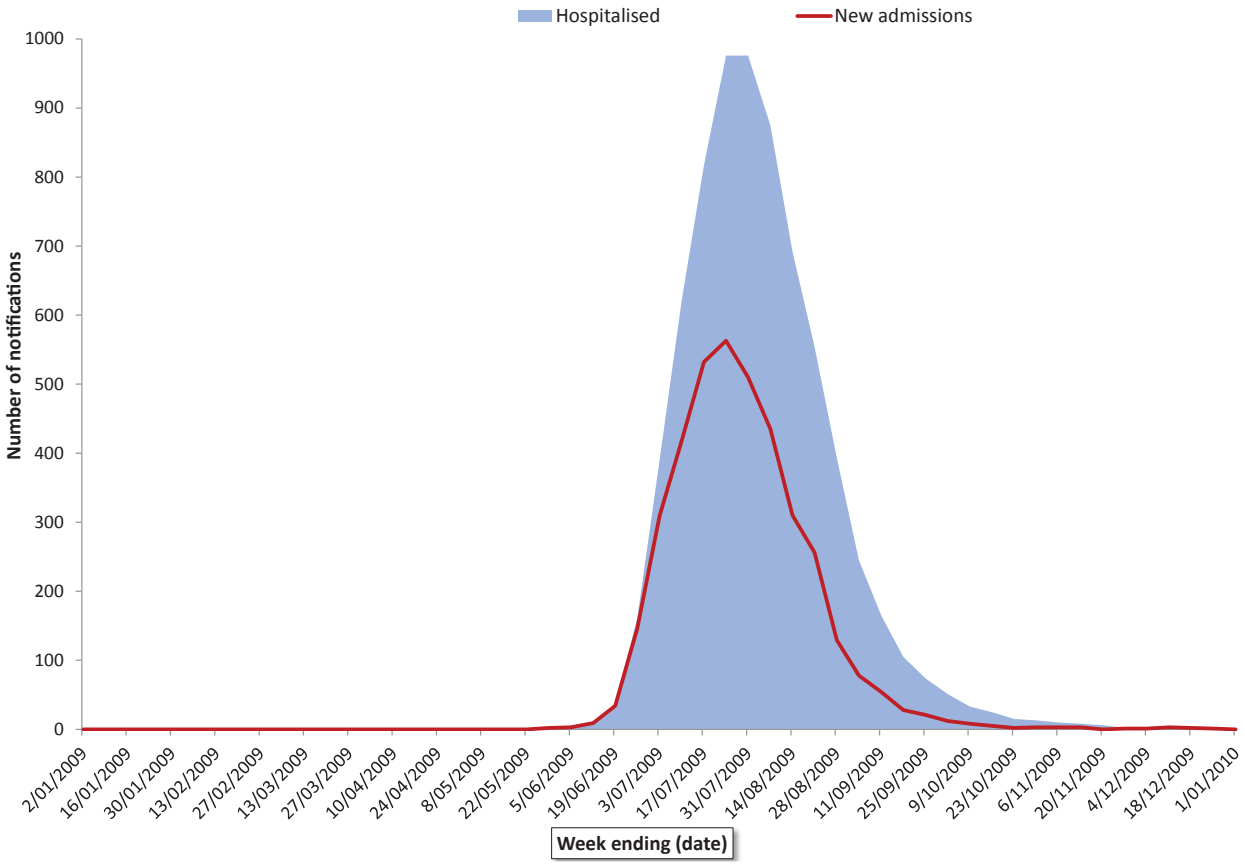
obesity and who were pregnant was much higher than the expected population prevalence for these conditions (Table 4).

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).

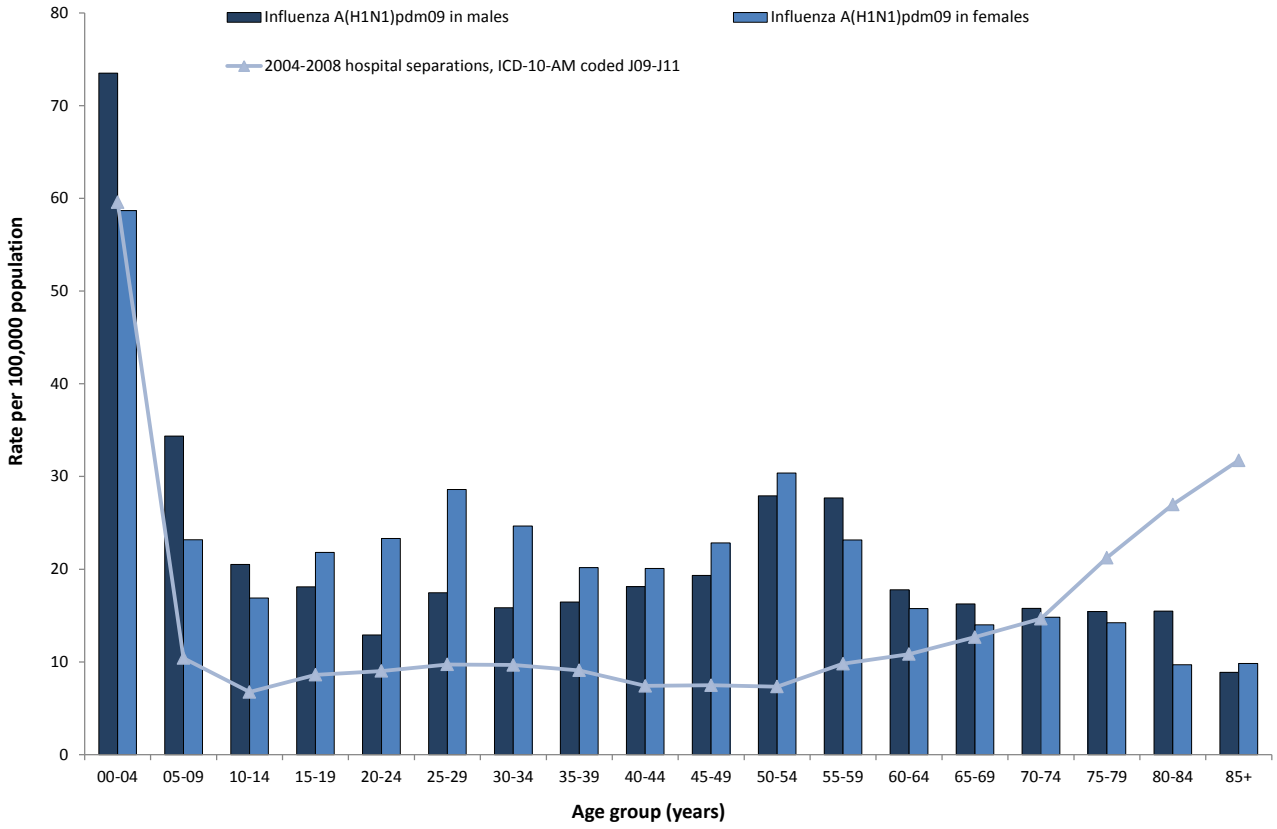
Severity and complications

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.

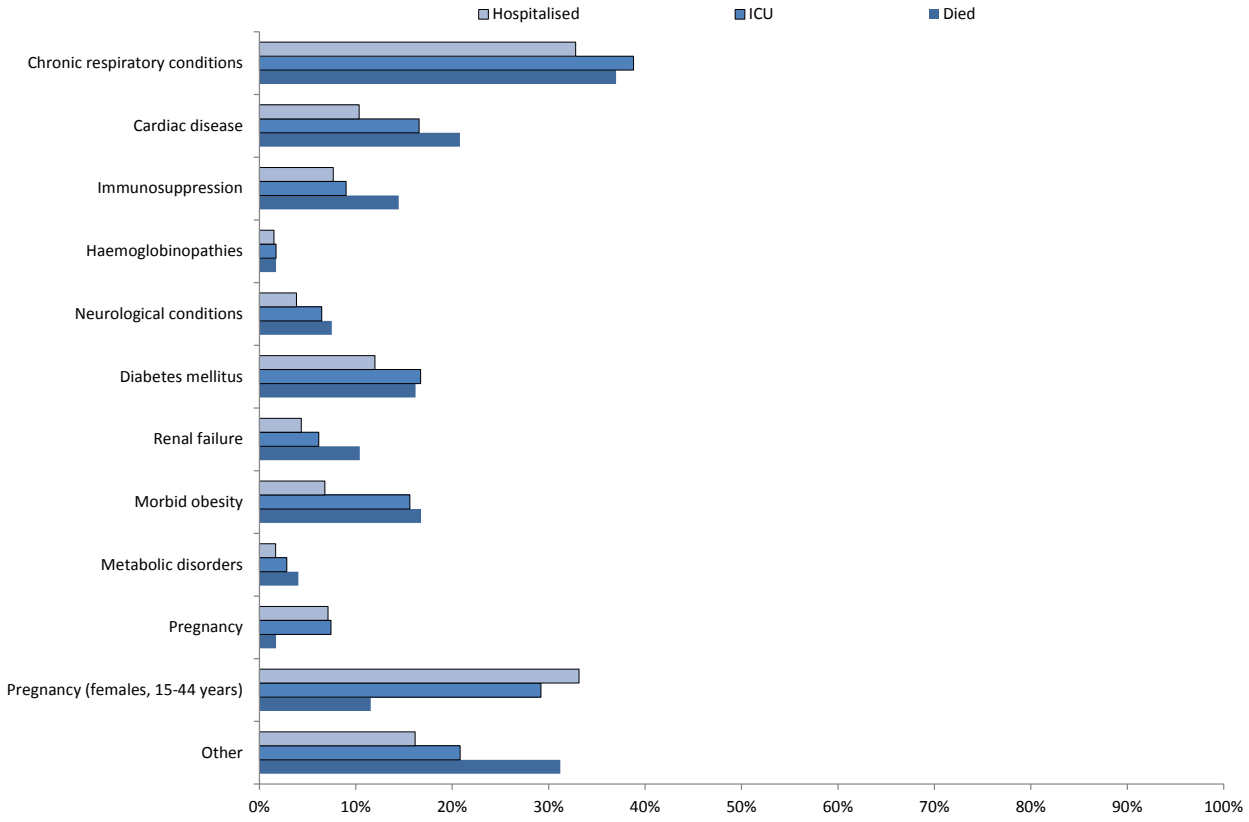
The number of hospital admissions peaked in the week ending 24 July 2009 at 562 (Figure 26). During this peak week of hospital admissions, there were over 970 people with confirmed influenza A(H1N1)pdm09 being cared for in hospital, representing 1.2% of beds available in both private and public acute 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

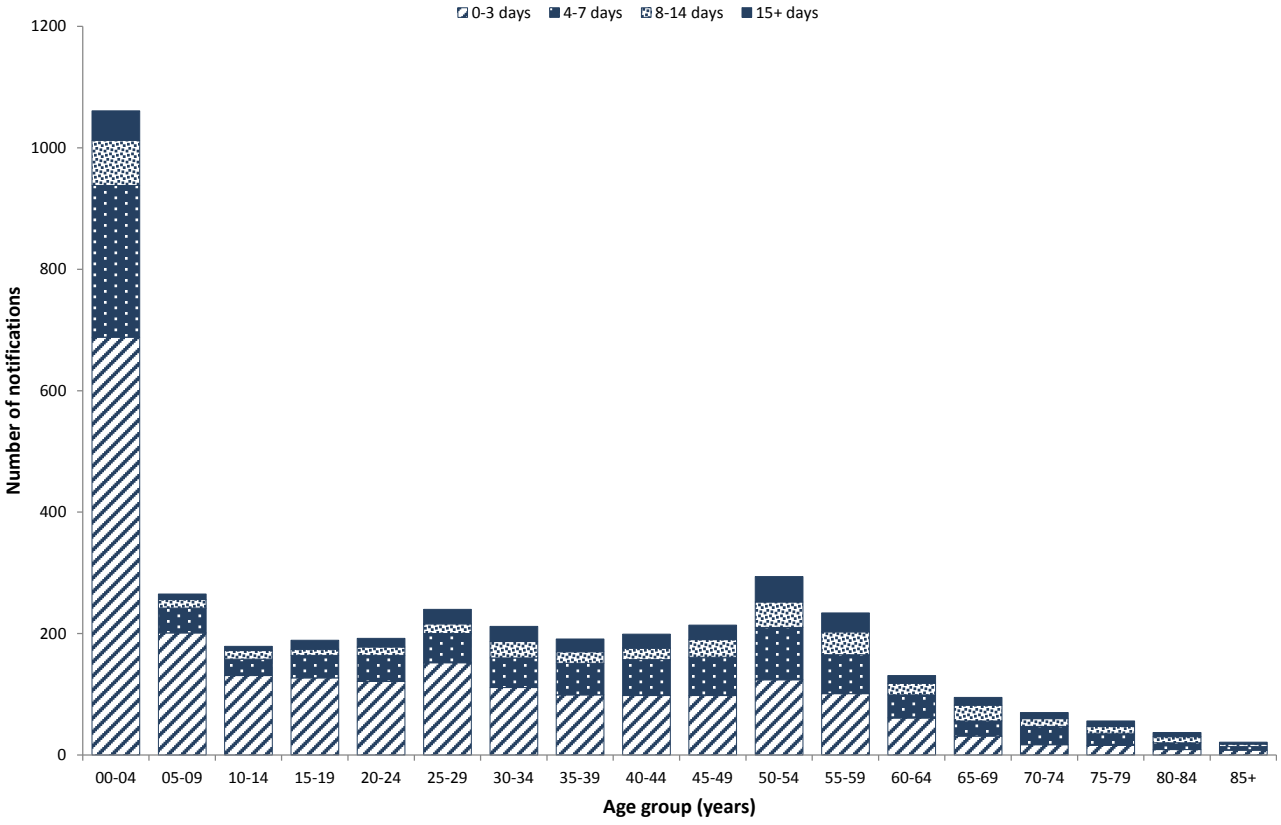
Information on length of stay was available for 69% (3,881/5,085) of hospitalised cases, which includes those cases also admitted to an ICU. A case may have already been hospitalised due to another condition; therefore if the period between date of admission and onset was greater than 7 days, date on 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).

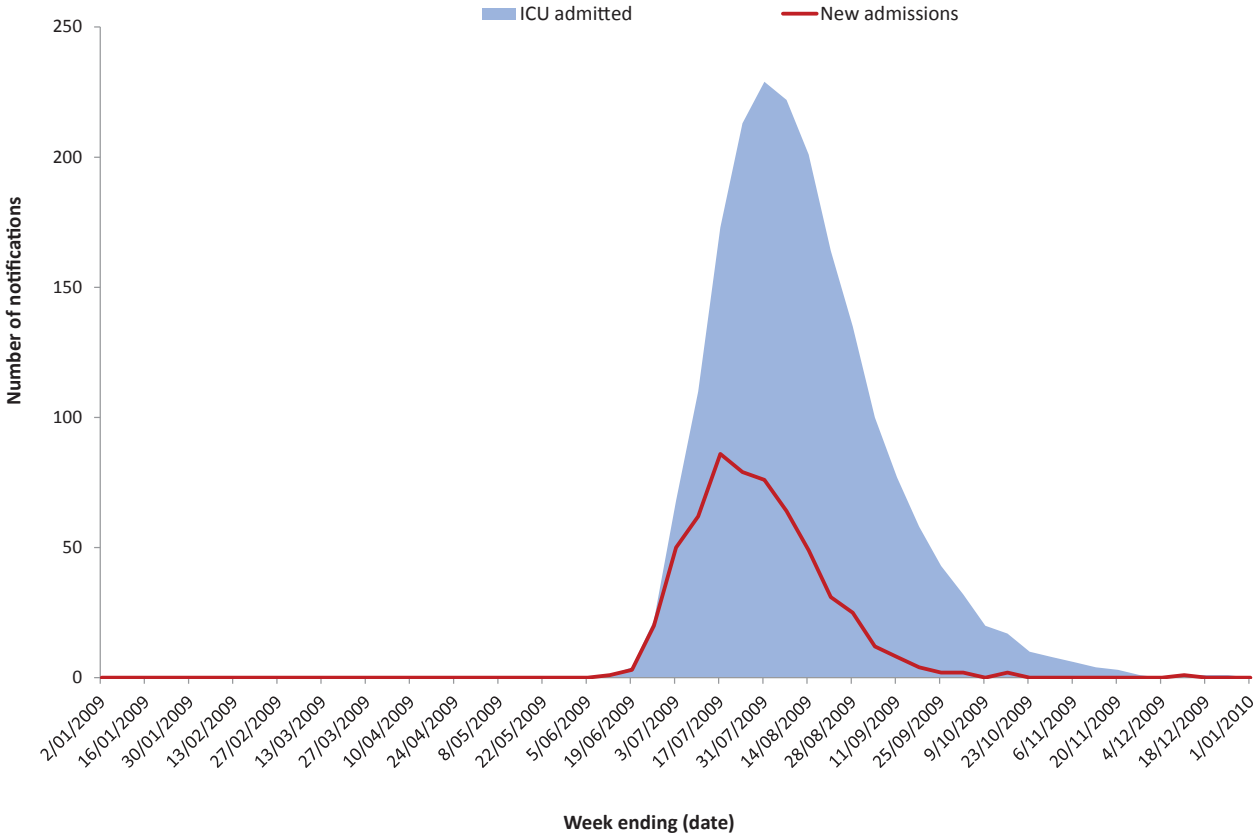
Of the hospitalised cases with clinical presentation data (3,181/5,085), three-quarters of cases presented with cough or fever (74.5% and 76.3% respectively) and almost half with breathing difficulty (45.6%). Twelve per cent of cases (371/3,181) presented with pneumonia. Data on the duration of each 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

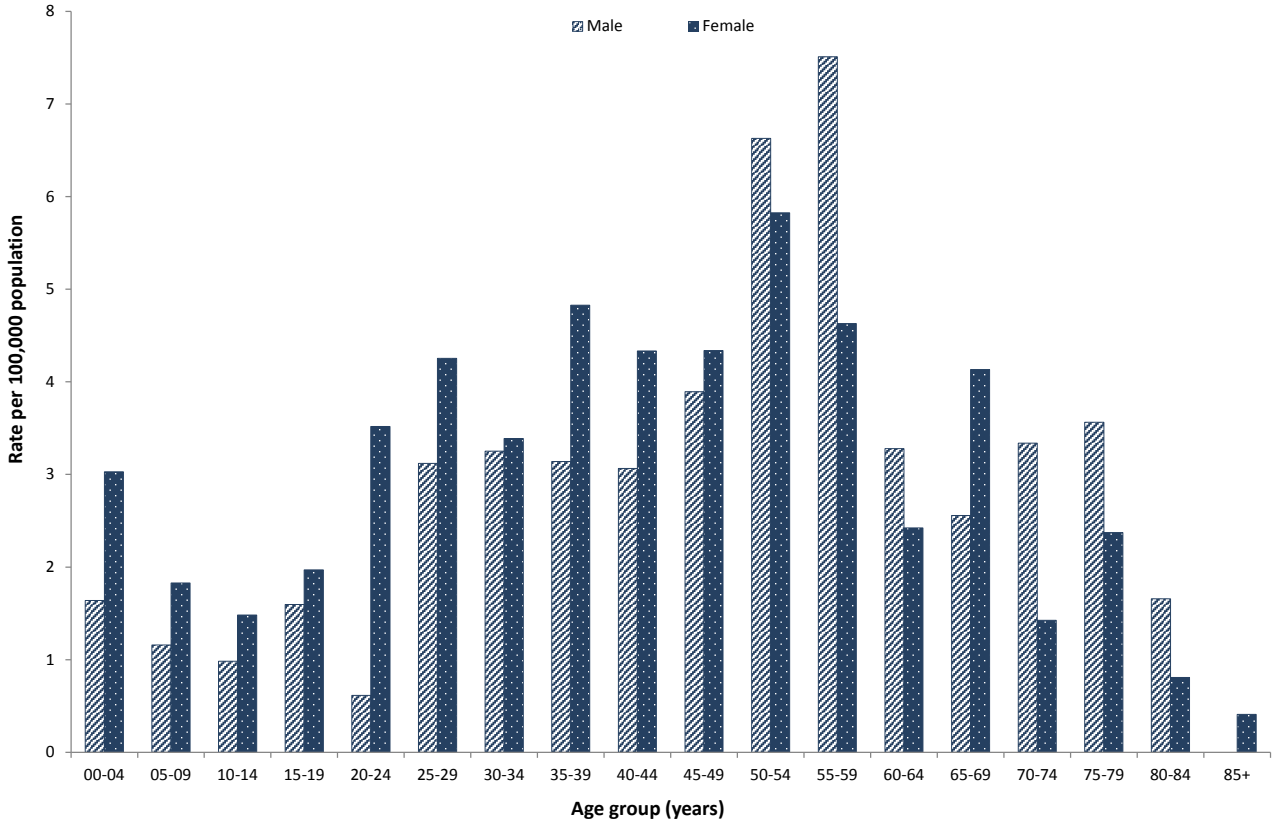
Cases admitted to intensive-care units (ICUs) represented 686 of the 5,085 (13.5%) hospitalised cases with confirmed influenza A(H1N1) pdm09 that were reported through NetEpi and Queensland's EpiLog system. However, Victoria and the ACT are not represented in the NetEpi dataset as these jurisdictions 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

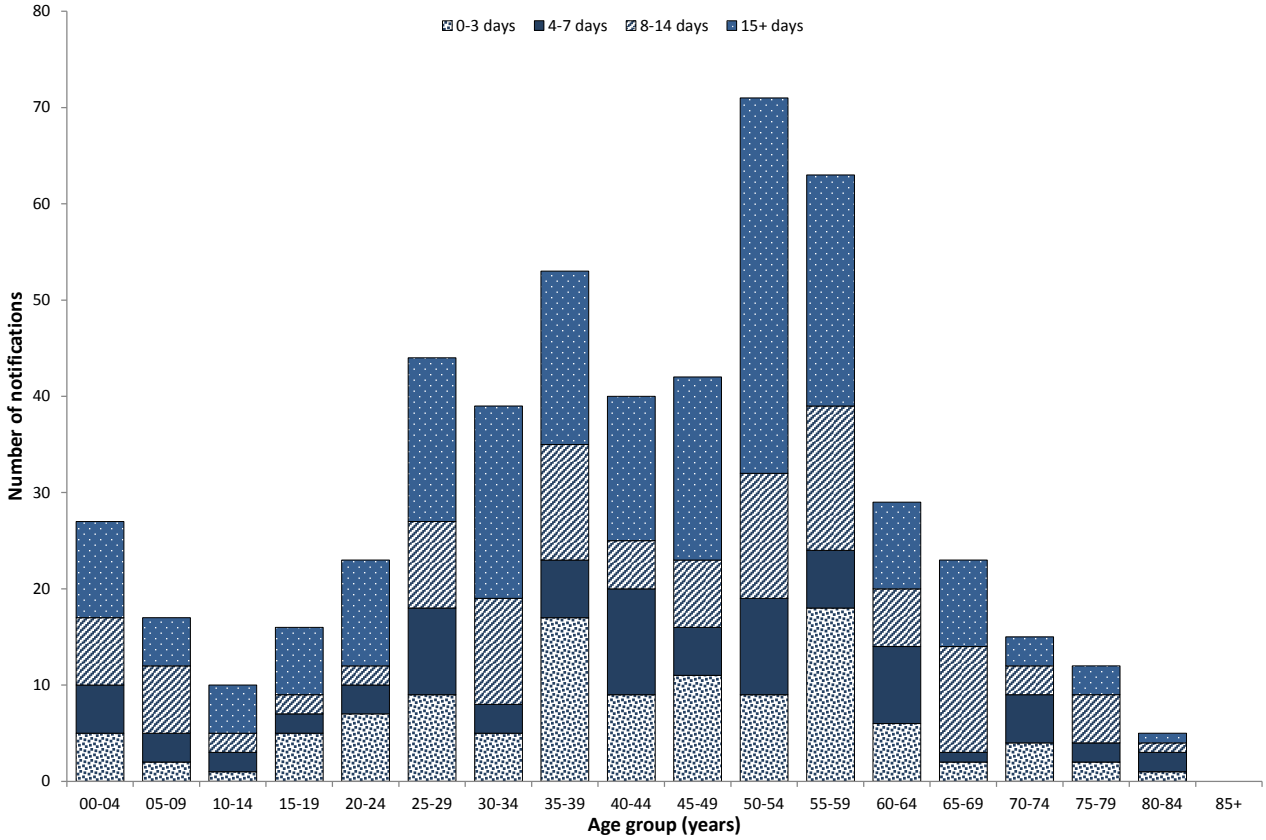
Information on the duration of hospitalisation for cases admitted to an ICU at some point during their hospitalisation was available for 84% (577/686) cases. It should be noted that the calculation of duration of stay could incorporate periods of care where the case was not in an ICU, but was still hospitalised. 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)

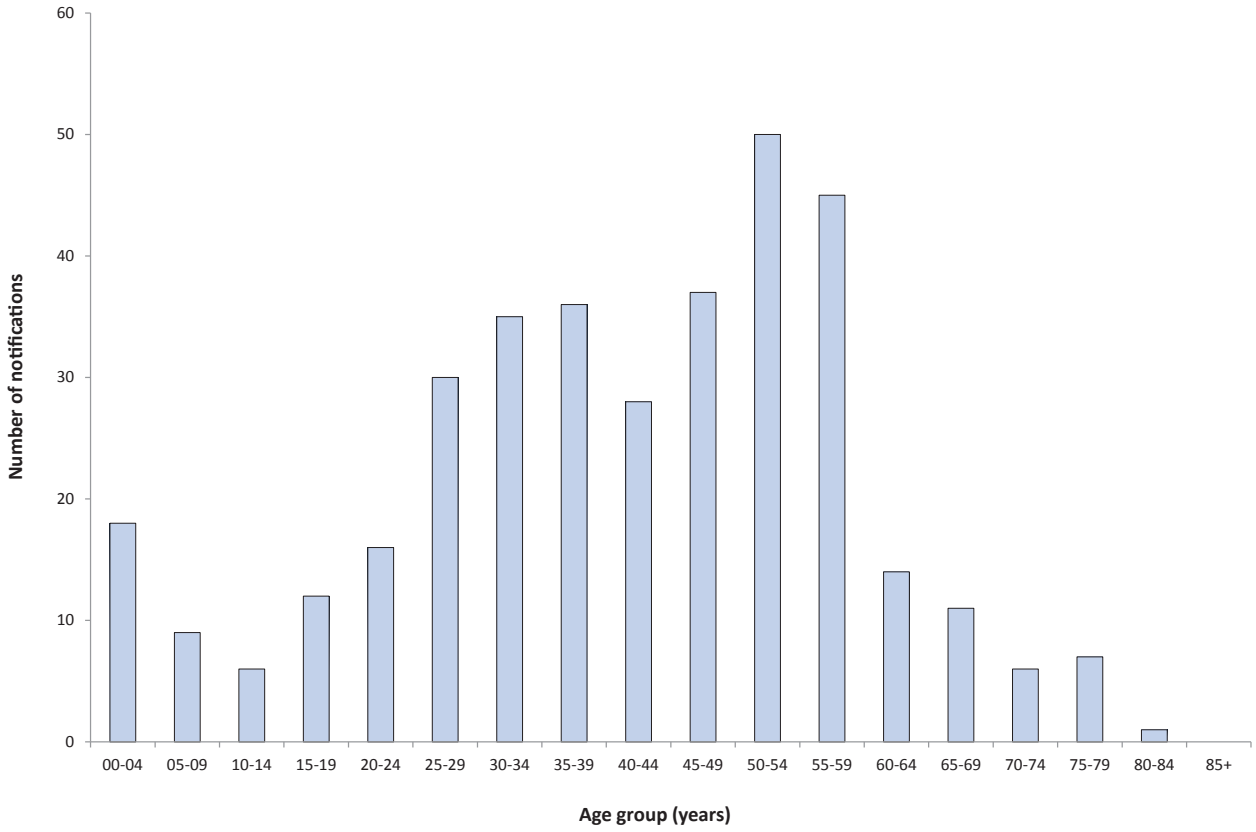
Data on influenza A(H1N1)pdm09 patients admitted to Australian ICUs were also col-

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-

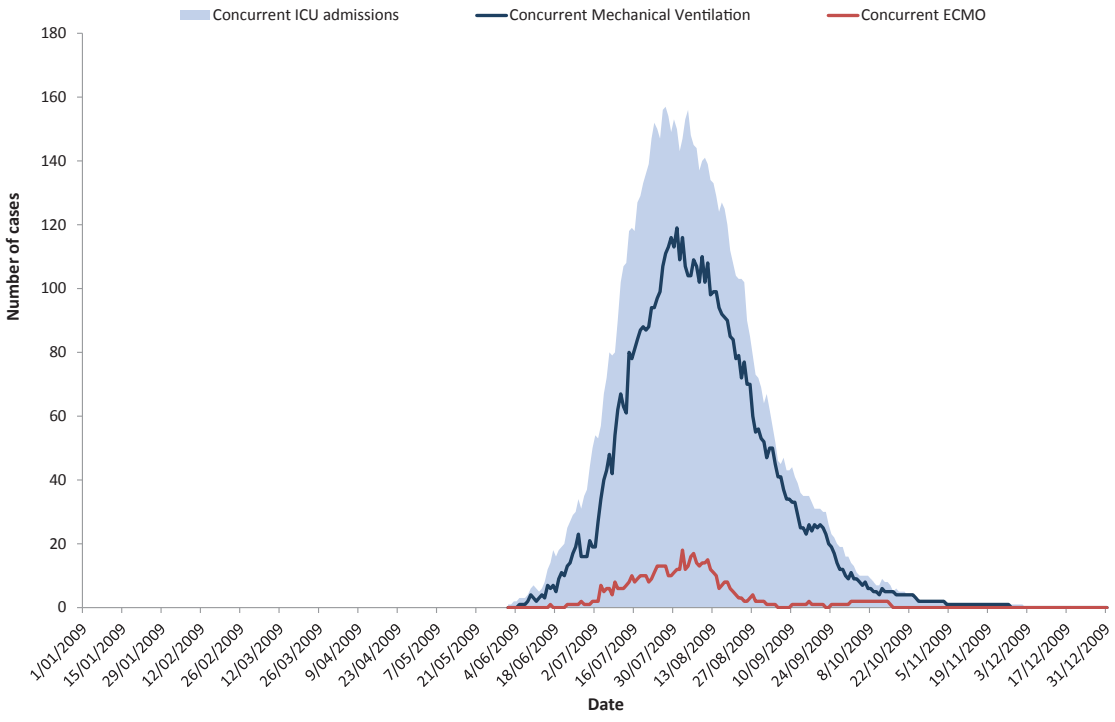
Table 9: Characteristics of cases admitted to an intensive care unit with confirmed influenza A(H1N1)pdm09 (n=718), Australia, 2009

	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.

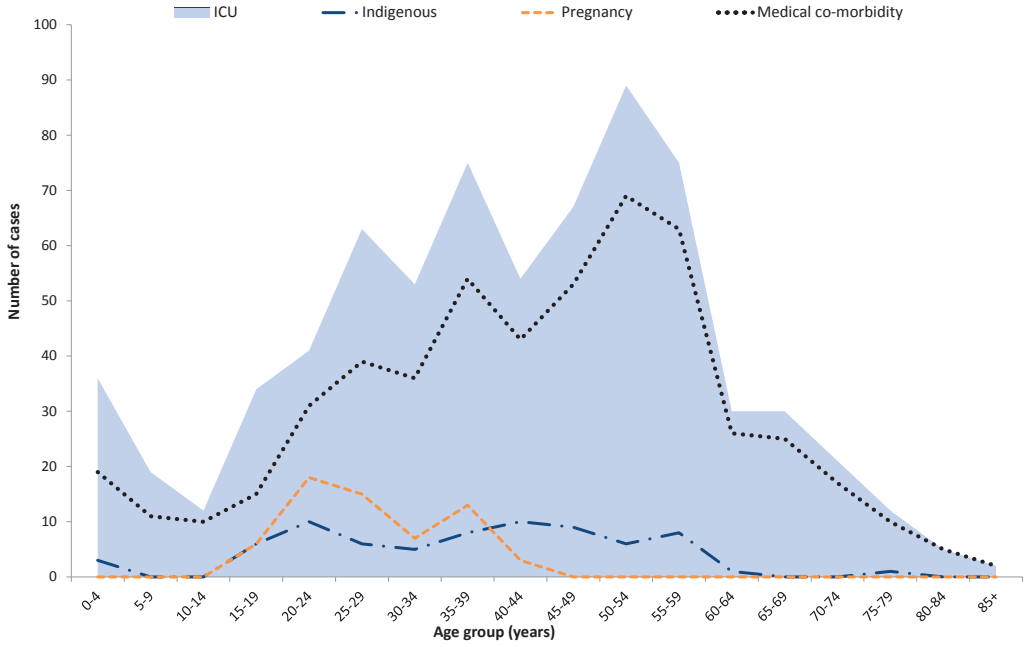
Figure 34: Number of confirmed influenza A(H1N1)pdm09 cases concurrently admitted to an ICU and either mechanically ventilated or receiving extracorporeal membrane oxygenation, Australia, 2009*, by date



Source: Australia and New Zealand Intensive Care Society

*ANZICS data collection commenced 1 June 2009

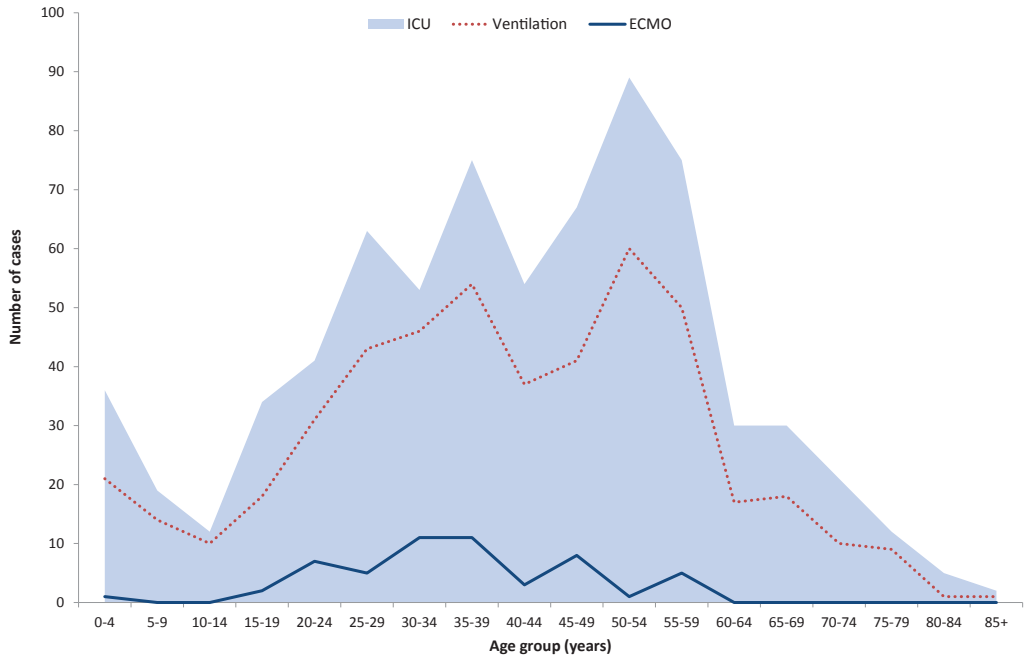
Figure 35: Number of confirmed influenza A(H1N1)pdm09 cases admitted to an ICU, Australia, 2009, by age group and risk factor*



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.

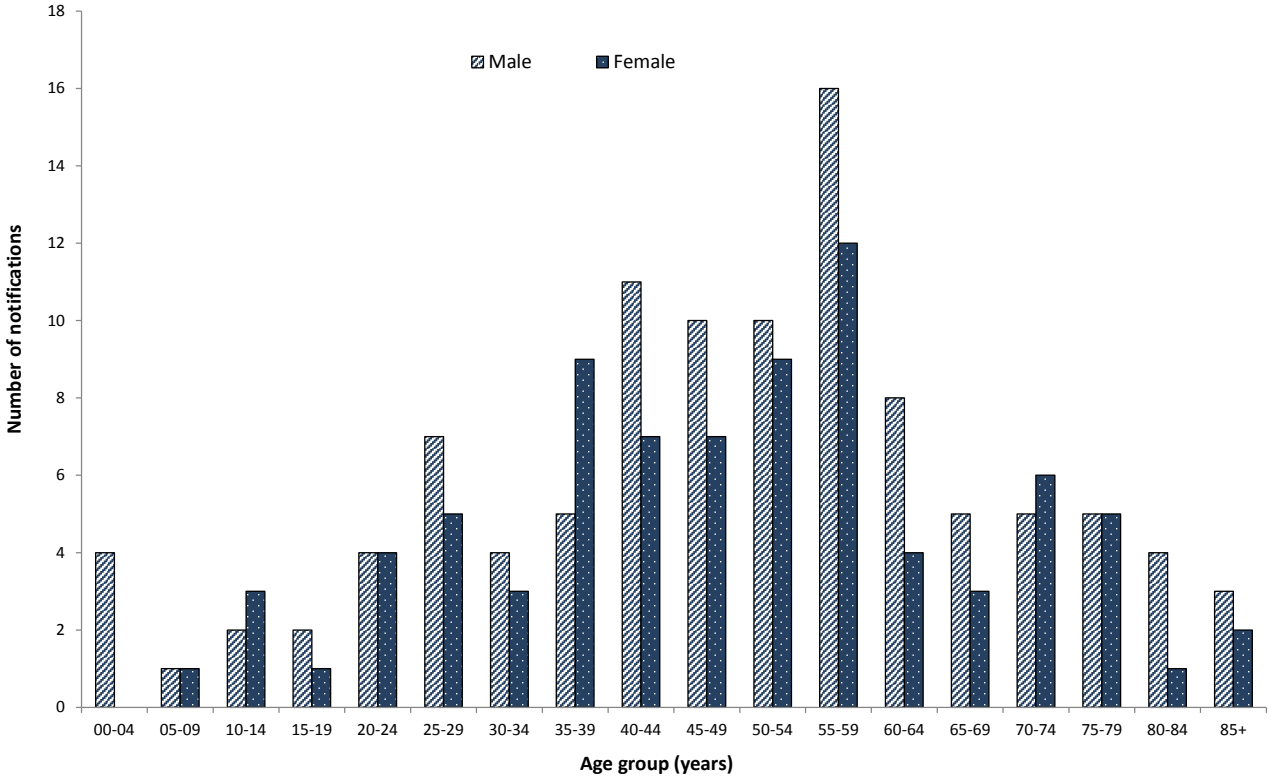
Of the cases who died, 82% (155/188) had been reported as being hospitalised, with the remainder reported as dying in another setting, such as palliative care services or at home.

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]

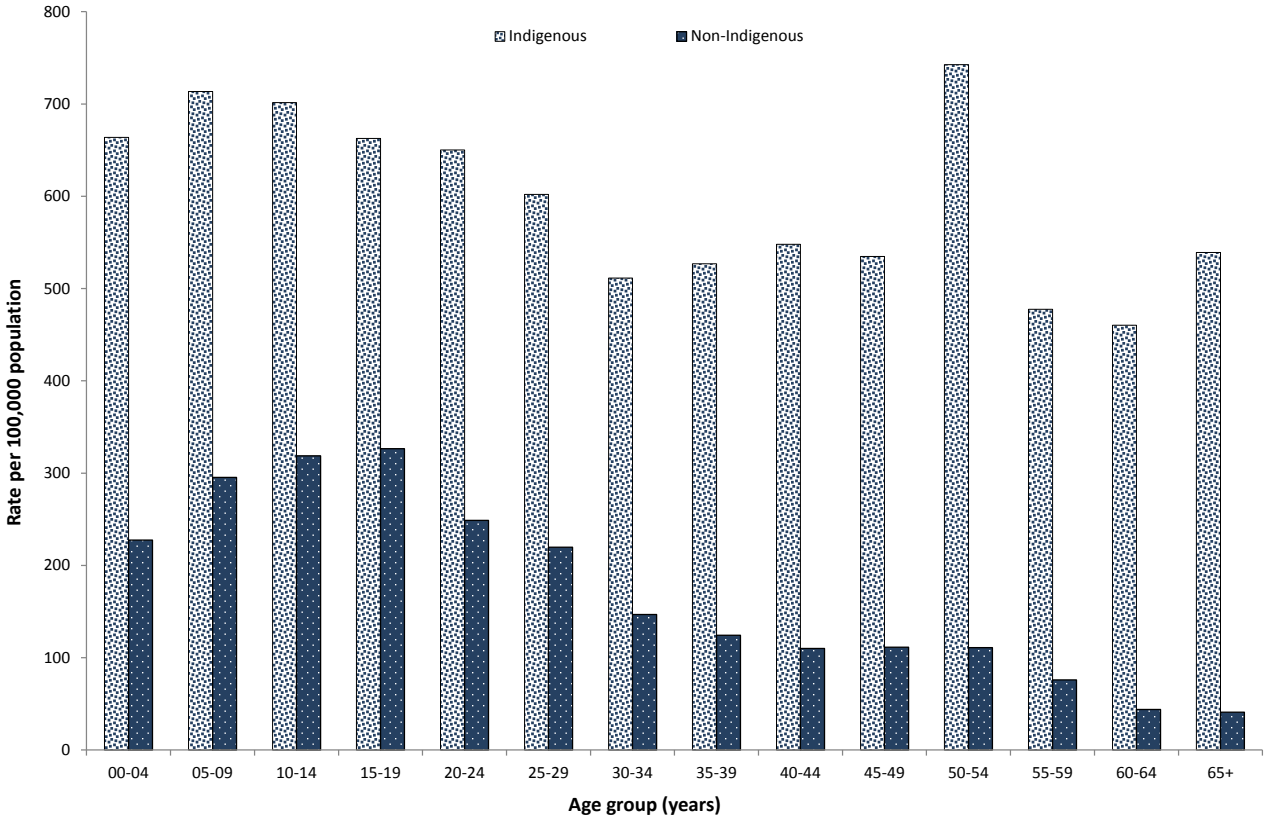
Specific risk group analysis

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

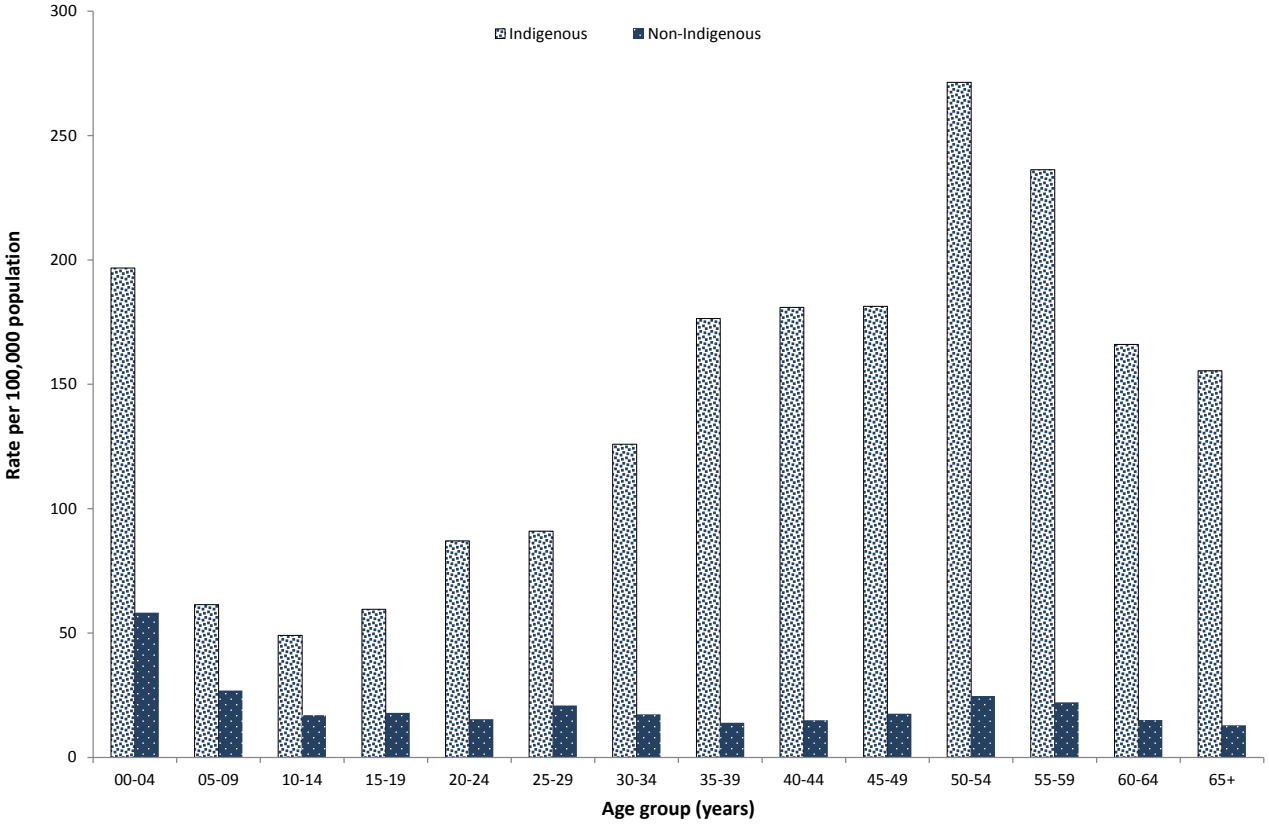
Of the 188 deaths reported to be associated with influenza A(H1N1)pdm09 in 2009, 23 (12.2%) were reported as Indigenous Australians. The age standardised mortality ratio for Indigenous Australians was 7.6 (Table 11).

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



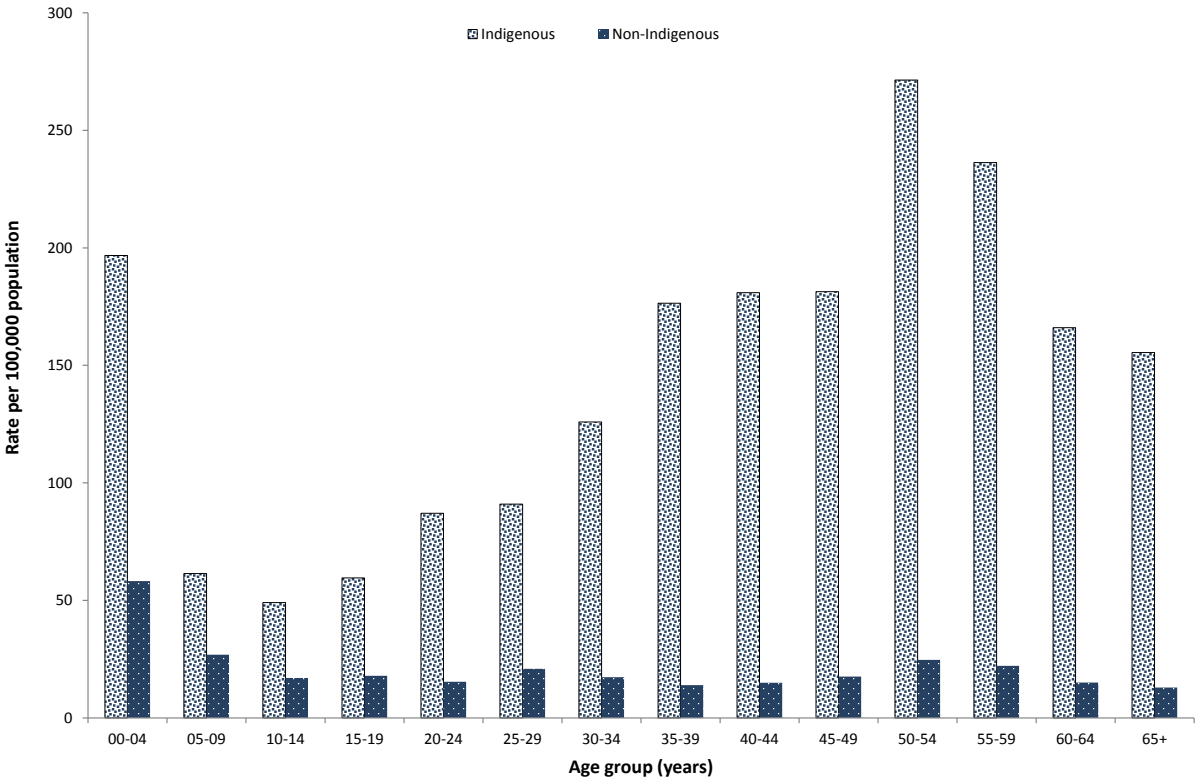
Source: NetEpi and EpiLog

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.

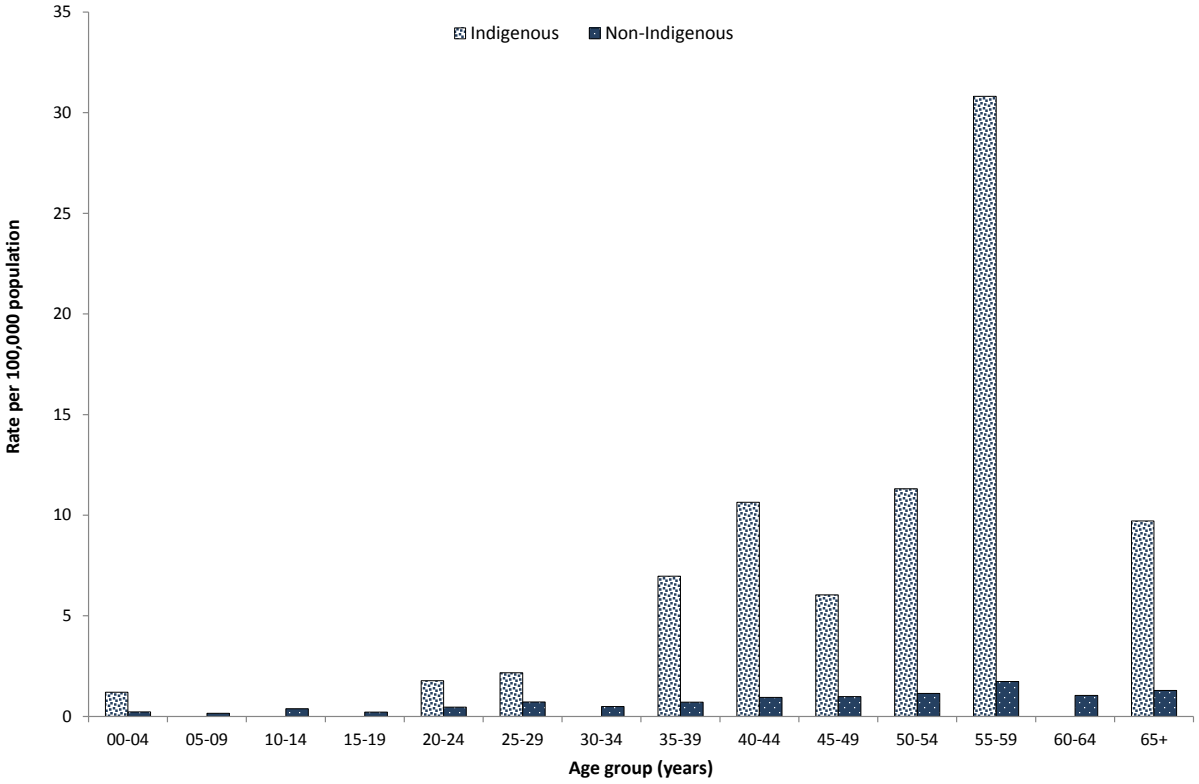
Figure 40: Rates of laboratory confirmed influenza A(H1N1)pdm09 among Indigenous Australians admitted to an ICU*, Australia, 2009, by age group and Indigenous status



Source: NetEpi and EpiLog

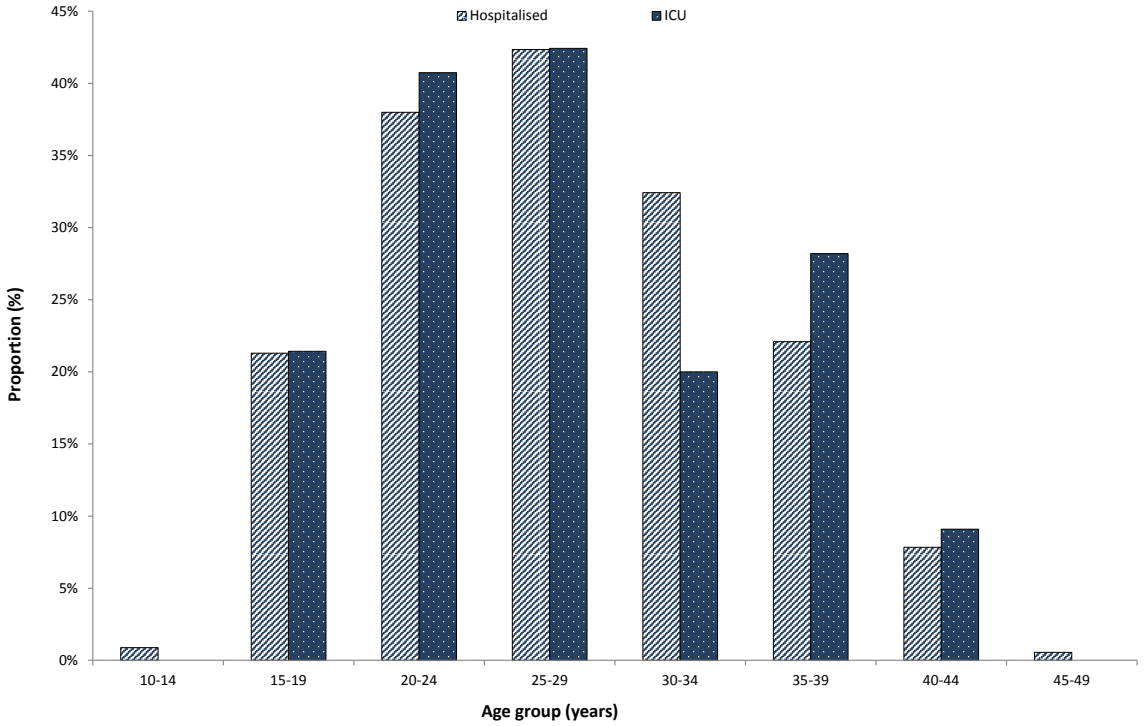
*Victoria and ACT ICU admissions are not represented in these data

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



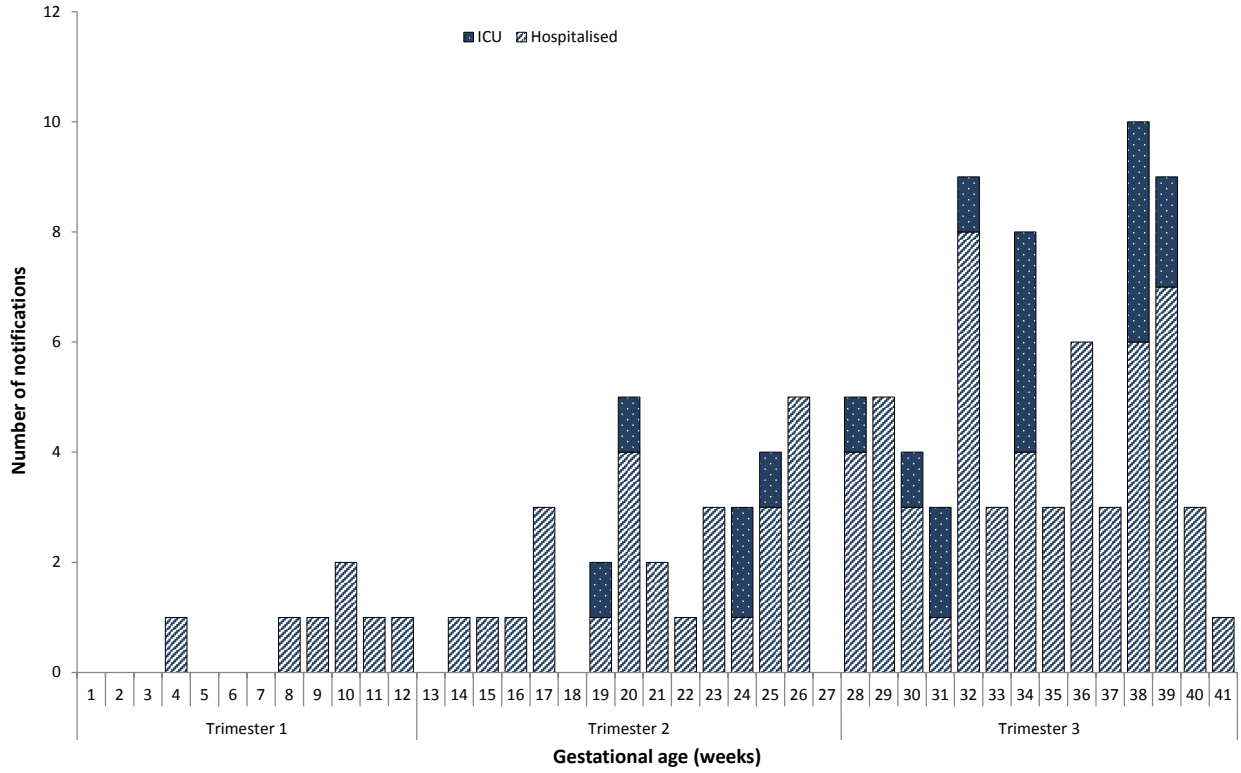
Source: NetEpi, EpiLog and NNDSS

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. Suspected and confirmed cases treated with antivirals and isolated. If a suspected case tested negative these measures were ceased. All contacts of suspected cases traced and if meeting the national contact definition were provided with antiviral prophylaxis and quarantined. 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. Suspected and confirmed cases treated with antivirals and isolated. If a suspected case tested negative these measures were ceased. 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. Antiviral treatment provided to people with acute febrile respiratory illness and immediate household contacts. Confirmed cases requested to isolate themselves for 3 days following commencement of antiviral treatment. No quarantine required for household contacts. Contact tracing in high risk settings intensified to protect those at greater risk of severe complications. 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. Sentinel testing also continued at hospital and community level for surveillance purposes and to monitor virus behaviour. Clinical cases offered antiviral treatment through consultation with healthcare professionals with an emphasis on treatment for persons in higher risk groups Contacts of cases not offered prophylaxis. 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: a person with acute febrile respiratory illness† with onset: 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 within 7 days of travel to Mexico, USA or Canada (countries to be updated where evidence of local transmission). 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: 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 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: 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 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: PCR (Matrix or other conserved region), OR an influenza rapid antigen, OR other antigen test e.g. immunofluorescence assay (IFA]). A suspected case with an influenza A positive test result is excluded where the sample tests: positive for human influenza A H1 and negative for H1N1Influenza 09 (human swine influenza) OR 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: PCR (Matrix or other conserved region), OR an influenza rapid antigen, OR other antigen test e.g. immunofluorescence assay (IFA]). A suspected case with an influenza A positive test result is excluded where the sample tests: positive for human influenza A H1 and negative for H1N1Influenza 09 (human swine influenza) OR 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: has an acute respiratory illness§, with or without fever, for which no other cause is identified, but 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: viral sequencing real-time RT-PCR 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: viral sequencing human swine influenza (H1N1) specific-PCR 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: viral sequencing human swine influenza (H1N1) specific-PCR 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: viral sequencing human swine influenza (H1N1) specific-PCR 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°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°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) 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: have travelled to an area with community transmission (anywhere overseas or to an Australian area of high prevalence) in the previous 7 days, OR are at risk of severe complications following human swine flu infection (pregnant women, people with diabetes or other chronic underlying illnesses, morbidly obese). Any Influenza virus A positive specimen should be subtyped and tested for H1N1 Influenza 09 (human swine influenza). **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: are at risk of severe complications following human swine flu infection (pregnant women, people with diabetes or other chronic underlying illnesses, morbidly obese). 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 NT Qld SA Tas 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 NSW[#] 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	NetEpi (until 6 July) NNDSS	n/a	NNDSS	NetEpi NNDSS
NT	n/a	NNDSS	NNDSS	n/a	NNDSS	NNDSS
All cases - Enhanced data[&]						
ACT NSW[#] 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)[*]	n/a	-*	NetEpi (until 6 July)
NT[*]	n/a	-	-	n/a	-	-
Hospitalised cases+ – Demographic data[&]						
ACT NSW[#] NT SA Tas 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) EpiLog (public) MS Excel Spreadsheet (private)	n/a	EpiLog (public) MS Excel Spreadsheet (private)	NetEpi EpiLog (public) MS Excel Spreadsheet (private)
Hospitalised cases+ – Enhanced data[&]						
ACT NSW[#] NT SA Tas 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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Annual report

Australian Rotavirus Surveillance Program: Annual Report, 2016

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Abstract

This report from the Australian Rotavirus Surveillance Program (ARSP) and collaborating laboratories Australia-wide, describes the rotavirus genotypes identified in children and adults with acute gastroenteritis during the period 1 January to 31 December 2016. During this period, 949 faecal specimens were referred for rotavirus G and P genotype analysis, of which 230 were confirmed as positive for wildtype rotavirus, and 184 were identified as rotavirus vaccine-like. Genotype analysis of the 230 samples from both children and adults revealed that G2P[4] was the dominant genotype in this reporting period nationally, identified in 29% of samples, followed by equine-like G3P[8] and G12P[8] (19% and 15% respectively). Genotype distribution remained distinct between States using RotaTeq® and Rotarix® vaccines. In RotaTeq® States, G12P[8] strains were more common, while G2P[4] and equine-like G3P[8] genotypes were more common in Rotarix® States and Territories. This report highlights the continued dominance of G12P[8] strains in RotaTeq® States and co-dominance of G2P[4] and equine-like G3P[8] in States and Territories using Rotarix®.

Keywords: rotavirus, gastroenteritis, genotypes, disease surveillance, Australia, vaccine, RotaTeq®, Rotarix®

Introduction

Rotaviruses, from the Reoviridae family, are triple layered dsRNA viruses that contain a segmented genome, consisting of 11 gene segments that encode 6 structural proteins and 6 non-structural proteins.[1] The segmented nature of rotavirus has been attributed as one of the major processes by which the virus can evolve, since it allows for reassortment both within and between human and animal strains, leading to the occurrence of unusual and novel rotavirus strains.[2] Rotaviruses are the most common cause of severe diarrhoea in young children worldwide, estimated to have caused 215,000 deaths in 2013 worldwide.[3] The latest figures are significantly lower than previous estimates of 611,000 deaths per annum[4], primarily due to the introduction of rotavirus vaccines, such as Rotarix® [GlaxoSmithKline] and RotaTeq® [Merck]. These two live attenuated oral rotavirus vaccines have been shown to be safe and highly effective in the prevention of severe diarrhoea due to rotavirus infection[5,6], leading to both vaccines being licensed in over 125 countries and included in the national vaccination schedules of 63 predominantly high and middle-income countries worldwide.[7] Since 1 July 2007, rotavirus vaccines have been included in the Australian National Immunisation Program (NIP), with excellent uptake in subsequent years across the nation. RotaTeq® is administered in Queensland, South Australia, Victoria, and Western Australia, while Rotarix® is administered in the Australian Capital Territory, New South Wales, the Northern Territory, and Tasmania.[8]

Before the introduction of rotavirus vaccines in Australia, rotavirus had accounted for ~10,000 childhood hospitalisations for diarrhoea each year.[9] A significant impact on acute gastroenteritis disease burden has been observed since vaccine introduction, with studies showing a

78% decline across Australia in both rotavirus coded and non-rotavirus coded hospitalisations in children under 5 years of age.[8,10,11]

The ARSP has characterised and reported the G- and P- genotypes of rotavirus strains causing severe disease in Australian children since 1999. Surveillance data generated by the ARSP has shown that strain diversity, as well as temporal and geographic changes occur each year.[12] Ongoing characterisation of circulating rotavirus genotypes will provide insight into whether vaccine introduction has impacted on virus epidemiology, altered circulating strains, or caused vaccine escape strains, which could have ongoing consequences for the success of current and future vaccination programs.

This report describes the G- and P- genotype distribution of rotavirus strains causing severe gastroenteritis in Australia for the period 1 January to 31 December 2016.

Methods

Rotavirus positive specimens detected by quantitative Reverse Transcription Polymerase Chain Reaction (RT-PCR), enzyme immunoassay (EIA), or latex agglutination in collaborating laboratories across Australia were collected, stored frozen and forwarded to the Australian National Rotavirus Reference Centre Melbourne, together with relevant age and gender details. The laboratories contributing samples for 2016 were:

- Microbiology Department, Canberra Hospital, Australian Capital Territory.
- The Virology Division, South Eastern Area Laboratory Services, Prince of Wales Hospital, New South Wales.
- Virology Department, The Children's Hospital at Westmead, New South Wales.
- Centre for Infectious Diseases and Microbiology, Westmead, New South Wales.
- The Microbiology Department, John Hunter Hospital, Newcastle, New South Wales.
- The Microbiology Department, Central Coast, Gosford, New South Wales.
- Douglas Hanly Moir Pathology, New South Wales.
- Royal North Shore Hospital, St. Leonards, New South Wales.
- The Microbiology Department, Royal Darwin Hospital, Casuarina, Northern Territory.
- The Microbiology Department, Alice Springs Hospital, Alice Springs, Northern Territory,
- Forensic and Scientific Services, Queensland Health, Herston, Queensland.
- Microbiology division, Pathology Queensland, Herston, Queensland.
- The Queensland Paediatric Infectious Diseases laboratory, Royal Children's Hospital, Brisbane, Queensland.
- Queensland Health laboratory, Townsville, Queensland.
- Microbiology and Infectious diseases laboratory, SA Pathology, Adelaide, South Australia.
- Molecular Medicine, Pathology Services, Royal Hobart Hospital, Hobart, Tasmania.
- The Serology Department, Royal Children's Hospital, Parkville, Victoria.
- QEII Microbiology Department, PathWest Laboratory Medicine WA, Perth, Nedlands, Western Australia.

Viral RNA was extracted from 10%–20% faecal extracts using the QIAamp Viral RNA mini extraction kit (Qiagen) according to the manufacturer's instructions. Rotavirus G- and P-genotypes were determined using an in-house hemi-nested multiplex RT-PCR assay. The first

round RT-PCR reactions were performed using the Qiagen one step RT-PCR kit, using VP7 conserved primers VP7F and VP7R, or VP4 conserved primers VP4F and VP4R. The second round genotyping PCR reactions were conducted using specific oligonucleotide primers for G types 1, 2, 3, 4, 8, 9, and 12, or P types [4], [6], [8], [9], [10], and [11].[13-17] The G- and P- genotype of each sample was assigned using agarose gel analysis of second round PCR products.

First round amplicons for VP7 were also purified for sequencing by using Wizard SV Gel for PCR Clean-Up System (Promega), according to the manufacturer's protocol. Purified DNA together with oligonucleotide primers (VP7F/R) were sent to the Australian Genome Research Facility, Melbourne, and sequenced using an ABI PRISM BigDye Terminator Cycle Sequencing Reaction Kit (Applied Biosystems, Foster City, CA, USA) in an Applied Biosystems 3730xl DNA Analyzer (Applied Biosystems, Foster City, CA, USA). Sequences were edited with Sequencher v.4.10.1. The genotype assignment was accomplished using BLAST (http://blast.ncbi.nlm.nih.gov/Blast.cgi) and RotaC v2.0 (http://rotac.regatools.be).[18]

Samples sent or identified as vaccine-like were confirmed for vaccine by amplifying a portion of the inner capsid VP6 gene, using human Rot3/Rot5 primers and Superscript III One-Step RT-PCR System with Platinum Taq DNA Polymerase (Invitrogen), as previously described.[19,20]

Any samples that provided a discordant result between the initial antigen detection and genotype assay were further tested using the commercial rotavirus ELISA assay ProSpecT (Thermo Fisher, Aus.), as per manufacturer's instructions, to confirm the presence of rotavirus antigen.

Results

Number of isolates

A total of 949 faecal specimens were collected during the period 1 January to 31 December 2016 for analysis from 18 collaborating centres across Australia, located in the Australian Capital Territory (ACT), New South Wales (NSW), Northern Territory (NT) Queensland (Qld), South Australia (SA), Tasmania (Tas), Victoria (Vic) and Western Australia (WA).

In 2016, 414 rotavirus positive samples from patients clinically diagnosed with acute gastroenteritis were identified. For analysis, these samples were divided based on whether a sample had no vaccine component identified (described herein as "wildtype rotavirus") or had a vaccine component identified based on VP6 or VP7 sequence analysis ("vaccine-like"). A total of 230 samples were confirmed as wildtype rotavirus positive by EIA (ProSpecT, OXOID) or RT-PCR analysis. Of these, 107 were collected from children under 5 years of age, and 123 were from older children and adults. An additional 535 specimens contained either insufficient specimen for genotyping (n=10), were duplicates of samples already analysed (n=43) or the specimen was not confirmed to be positive for rotavirus (n=482) and were thus not analysed further.

In addition, 184 samples were identified as rotavirus vaccine-like by VP6 and/or VP7 sequencing. The majority of these samples (n=162) were received from South Australia, where a duplex rapid real time qRT-PCR assay that could differentiate between wildtype and RotaTeq® NSP3 was used.[21] Of these, 101 were sent from hospital settings, while the remaining 61 were from general practices across South Australia. These samples were already identified as positive by the collaborator's rotavirus and RotaTeq®-specific qRT-PCR assays, and were consequently included in this surveillance report. Other RotaTeq® samples were identified from Qld Regional (n=1), Townsville Pathology, Qld (n=1), Royal Children's Hospital, Vic (n=3), and PathWest, WA (n=6). Rotarix® vaccine was identified in 11 cases by VP7 sequence. These samples were provided by Westmead, NSW (n=8), John Hunter Hospital, NSW (n=1) and Royal Hobart Hospital, Tas (n=1).

Wildtype rotavirus specimens:

Age distribution for wildtype rotavirus infections

From 1 January to 31 December 2016, 46.5% of rotavirus positive samples were obtained from children under 5 years of age (Table 1). A total of 14.3% of wildtype rotavirus positive samples were from children 13-24 months of age, and 35.2% of samples were from individuals older than 20 years of age.

In the samples from children under 5 years of age, almost a third of all samples (30.8%) were identified in children 13-24 months old, while the next most common age group was 7-12 months where 19.6% of cases were found.

Wildtype rotavirus genotype distribution

Genotype analysis was performed on the 230 confirmed rotavirus positive cases from children and adults (Table 2). G2P[4] was the most common genotype identified nationally, representing 29% of all specimens analysed. This genotype was identified as the dominant genotype in NSW and WA, representing 41% and 33% of strains respectively. G2P[4] was also the only strain detected in ACT, however only 3 rotavirus positive samples were received from ACT for this reporting period.

A previously described equine-like G3P[8] strain[22,23] was the second most common genotype found in Australia, representing 19% of all strains nationally (Table 2). The majority of these equine-like G3P[8] samples were found in NSW, representing 33% of all strains identified within the State. G12P[8] strains were the third most common genotype identified nationally, representing 15% of all specimens. G12P[8] strains were dominant in Qld, SA and Vic, representing 37%, 29% and 38% of each state total, respectively. Other common genotypes identified nationally in 2016 included G9P[8] (10%), G1P[8] (7%), and G3P[8] (6%).

Twenty-nine (13% of rotavirus positive) specimens did not fall into a common genotype category (Table 3). Whilst two samples were of mixed genotype (G1/G3P[8] and equine-like G3 P[4]/P[8]), the remaining 27 samples represented 12 uncommon rotavirus strains. Six of these strains included unusual combinations, such as G1P[6], G2P[8], G3P[4], G9P[4], G9P[6], and G12P[6]. The remaining 6 were represented by strains that contained an animal VP7 and/or VP4 component. Feline/canine-like G3P[3] were identified in 2 samples from the NT, while bovine-like strains such as G6P[14] (n=4), G8P[8] (n=3), G8P[14] (n=2), G10P[5] (n=1), and G10P[14] (n=1) were identified in multiple States across Australia.

A G- or P- genotype could not be assigned to 3 samples (Table 2). Two of these were G-non typeable samples from SA (G-non typeable P[4]) and Tas (G-non typeable P[8]). The third sample was an equine-like G3 P[non typeable] from Qld. The partially non typeable samples could be due to either low viral load, mutations in the primer annealing regions, or inhibitors within

Table 1: Age distribution of rotavirus wildtype gastroenteritis cases

Age (months)	Age (years)	n=	% of total	% under 5 years
0-6		13	5.7	12.1
7-12	≤1	21	9.1	19.6
13-24	1-2	33	14.3	30.8
25-36	2-3	20	8.7	18.7
37-48	3-4	12	5.2	11.2
49-60	4-5	8	3.5	7.5
Subtotal		107	46.5	-
61-120	5-10	23	10.0	
121-240	10-20	19	8.3	
241-960	20-80	58	25.2	
961+	>80	23	10.0	
Total		230	-	

Table 2: Rotavirus G and P genotype distribution in infants, children and adults, 1 January to 31 December 2016

Centre	Type total	G1P[8]		G2P[4]		G3P[8]		G3P[8]*		G9P[8]		G12P[8]		Other†		Non-type‡		Neg
		%	n	%	n	%	n	%	n	%	n	%	n	%	n	%	n	n
Australian Capital Territory																		
ACT	3	-	0	100	3	-	0	-	0	-	0	-	0	-	0	-	0	0
New South Wales																		
Prince of Wales Hospital	23	-	0	74	17	-	0	22	5	4	1	-	0	-	0	-	0	0
Westmead	19	11	2	42	8	-	0	16	3	16	3	-	0	16	3	-	0	22
Westmead - CIDM	14	-	0	29	4	7	1	7	1	36	5	7	1	14	2	-	0	6
John Hunter	15	-	0	13	2	-	0	80	12	7	1	-	0	-	0	-	0	5
Other §	11	-	0	27	3	9	1	55	6	-	0	-	0	-	0	9	1	4
NSW subtotal:	82	2	2	41	34	2	2	33	27	12	10	1	1	6	5	1	1	37
Northern Territory																		
Alice Springs	1	-	0	100	1	-	0	-	0	-	0	-	0	-	0	-	0	4
Royal Darwin Hospital	0	-	0	-	0	-	0	-	0	-	0	-	0	-	0	-	0	1
Other §	2	-	0	-	0	-	0	-	0	-	0	-	0	100	2	-	0	2
Northern Territory subtotal:	3	-	0	33	1	-	0	-	0	-	0	-	0	67	2	-	0	7
Queensland																		
Pathology Brisbane	3	33	1	-	0	-	0	33	1	-	0	33	1	-	0	-	0	4
Qld Regional	15	-	0	27	4	33	5	-	0	-	0	33	5	7	1	-	0	186
Pathology Townsville	1	-	0	-	0	-	0	-	0	-	0	100	1	-	0	-	0	3
Queensland subtotal:	19	5	1	21	4	26	5	5	1	-	0	37	7	5	1	-	0	193
South Australia																		
Adelaide	78	12	9	17	13	6	5	9	7	9	7	29	23	17	13	1	1	142
Tasmania																		
Hobart	4	-	0	-	0	-	0	50	2	25	1	-	0	-	0	25	1	2
Victoria																		
Royal Children's Hospital	8	-	0	-	0	-	0	13	1	13	1	38	3	38	3	-	0	26
Western Australia																		
PathWest	33	12	4	33	11	6	2	18	6	12	4	3	1	15	5	-	0	75
TOTAL	**230**	**7**	**16**	**29**	**66**	**6**	**14**	**19**	**44**	**10**	**23**	**15**	**35**	**13**	**29**	**1**	**3**	**482**

* Equine-like G3P[8]
† See Table 3
‡ A specimen where G and/or P genotype was not determined
§ NSW: Faecal specimens which were received from Pathology North, Central Coast Gosford, NSW; Douglas Hanly Moir Pathology, NSW; Royal North Shore Hospital, Sydney, NSW; Pathology Brisbane, Qld; Qld Regional, Qld; Adelaide, SA
§ NT: Faecal specimens which were received with SA/Pathwest WA samples

Table 3: Mixed and unusual G and P genotypes identified in infants, children and adults, 1 January to 31 December 2016

Genotype	RotaTeq®				Rotarix®		Total
	Qld	SA	Vic	WA	NSW	NT	
G1P[6]	-	1	-	-	-	-	1
G2P[8]	-	1	-	-	-	-	1
Feline/canine G3P[3]	-	-	-	-	-	2	2
G3P[4]	-	2	1	-	-	-	3
G6P[14]	-	2	1	-	1	-	4
G8P[8]	-	-	-	3	-	-	3
G8P[14]	-	1	-	-	1	-	2
G9P[4]	1	1	1	1	3	-	7
G9P[6]	-	1	-	-	-	-	1
G10P[5]	-	1	-	-	-	-	1
G10P[14]	-	1	-	-	-	-	1
G12P[6]	-	1	-	-	-	-	1
Mixed G1/G3P[8]	-	1	-		-	-	1
Equine G3 mixed P[4]/P[8]	-		-	1	-	-	1
Total:							**29**

the extracted RNA, which could have prevented the function of the enzymes used in the RT and/or PCR steps.

Genotypes identified in samples from children less than 5 years of age

107 wildtype rotavirus samples in total were collected from children under 5 years of age (Table 4). Within this cohort, G2P[4] was the most common genotype identified, found in 28% of all samples. Equine-like G3P[8] was the second most common genotype (19%), and G12P[8] strains were the third most common genotype (17%). G9P[8] and G1P[8] strains represented minor genotypes of children in this cohort, identified in 8% and 7% of samples respectively (Table 4).

Table 4: Rotavirus G and P genotype distribution in infants and children under 5 years of age, 1 January to 31 December 2016

Centre	Type total	G1P[8]		G2P[4]		G3P[8]		G3P[8]*		G9P[8]		G12P[8]		Other†		Non-type‡		Neg
		%	n	%	n	%	n	%	n	%	n	%	n	%	n	%	n	n
Australian Capital Territory																		
ACT	2	-	0	100	2	-	0	-	0	-	0	-	0	-	0	-	0	0
New South Wales																		
Prince of Wales Hospital	11	-	0	73	8	-	0	27	3	-	0	-	0	-	0	-	0	0
Westmead	15	7	1	33	5	-	0	20	3	20	3	-	0	20	3	-	0	22
Westmead - CIDM	12	-	0	33	4	8	1	8	1	42	5	-	0	8	1	-	0	4
John Hunter	8	-	0	-	0	-	0	100	8	-	0	-	0	-	0	-	0	3
Other §	4	-	0	25	1	25	1	50	2	-	0	-	0	-	0	-	0	1
NSW subtotal:	50	2	1	36	18	4	2	34	17	16	8	-	0	8	4	-	0	30
Northern Territory																		
Alice Springs	1	-	0	100	1	-	0	-	0	-	0	-	0	-	0	-	0	4
Royal Darwin Hospital	0	-	0	-	0	-	0	-	0	-	0	-	0	-	0	-	0	1
Other §	2	-	0	-	0	-	0	-	0	-	0	-	0	100	2	-	0	2
Northern Territory subtotal:	3	-	0	33	1	-	0	-	0	-	0	-	0	67	2	-	0	7
Queensland																		
Pathology Brisbane	1	100	1	-	0	-	0	-	0	-	0	-	0	-	0	-	0	0
Qld Regional	12	-	0	17	2	33	4	-	0	-	0	42	5	8	1	-	0	114
Pathology Townsville	1	-	0	-	0	-	0	-	0	-	0	100	1	-	0	-	0	3
Queensland subtotal:	14	7	1	14	2	29	4	-	0	-	0	43	6	7	1	-	0	117
South Australia																		
Adelaide	20	10	2	20	4	5	1	5	1	5	1	50	10	5	1	-	0	90
Tasmania																		
Hobart	2	-	0	-	0	-	0	50	1	-	0	-	0	-	0	50	1	2
Victoria																		
Royal Children's Hospital	4	-	0	-	0	-	0	25	1	-	0	25	1	50	2	-	0	12
Western Australia																		
PathWest	12	33	4	25	3	17	2	-	0	-	0	8	1	17	2	-	0	71
TOTAL	**107**	**7**	**8**	**28**	**30**	**8**	**9**	**19**	**20**	**8**	**9**	**17**	**18**	**11**	**12**	**1**	**1**	**329**

* Equine-like G3P[8]
† See Table 3
‡ A specimen where G and/or P genotype was not determined
§ NSW: Faecal specimens which were received from Pathology North, Central Coast Gosford, NSW; Douglas Hanly Moir Pathology, NSW; Royal North Shore Hospital, Sydney, NSW; Pathology Brisbane, Qld; Qld Regional, Qld; Adelaide, SA

§ NT: Faecal specimens which were received with SA/Pathwest WA samples

Genotypes identified in samples from individuals greater than 5 years of age

A total of 123 rotavirus samples were collected from children over the age of 5 years and adults (Table 5). This cohort was similar to the under 5 years of age group, in that G2P[4] was the main genotype identified (29%), followed by equine-like G3P[8] (20%) and G12P[8] (14%). G9P[8] was more prominent, found in 11% of all samples within this cohort.

Distribution of genotypes according to vaccine type in children less than 5 years of age

G- and P- genotypes of the 107 rotavirus positive samples were divided according to vaccine use (Figure). In states where RotaTeq® is in use, G12P[8] strains were the dominant genotype in children less than 5 years, identified in 36% of samples, compared to no observations in Rotarix® States. G2P[4] was the second most common genotype identified in RotaTeq® States (18%). Genotypes G1P[8] and G3P[8] were the third most common genotypes representing 14% of all samples individually. In locations using Rotarix®, G2P[4] strains were dominant, identified in 37% of strains, followed by equine-like G3P[8], identified in 32% of samples. By comparison, equine-like G3P[8] was only detected in 4% of all samples from States administering RotaTeq®.

Vaccine-like rotavirus specimens:

Age distribution for rotavirus vaccine cases

During the 2016 reporting period, 184 samples were identified as rotavirus vaccine by VP6 and/or VP7 sequencing (Table 6). Of these, 90.2% were from 0-6 month old patients, while 16% were from 7-12 month old patients. Two outlying samples were collected from older children in SA and Qld, aged 20 months and 40 months old respectively.

Genotype distribution of specimens containing rotavirus vaccine component

Of the 184 samples that had sequence confirmation of vaccine-like VP6 and/or VP7, 141 samples had been processed further for genotype analysis (Table 7). All samples identified with components of the Rotarix® vaccine (n=11) were genotyped as G1P[8], while samples containing RotaTeq® vaccine components (n=130) had more varied genotype combinations, due to the pentavalent nature of the vaccine. The most common combination identified by agarose gel electrophoresis was G1P[nt], identified in 48/130 samples, followed by G1P[8] (21/130). Single genotypes were identified in 17 samples, including G2, G4, and G6 with either a P[8] or P[non typeable]. Note, G6 samples had to be sequence confirmed, as primers for this bovine vaccine component are not included in the routine G-typing primer mix. G6 would present as G12 (~382bp band) in agarose gels if the G12 primer was included in the G-mix. Other combinations included various mixed G-types that contained two to four of all human virus components (G1, G2, G3 and G4) of the RotaTeq® vaccine, with either a P[8] or P[non typeable] type. Fully non typeable genotype results were attributed to 14 samples.

The majority of these P[nt] samples were most likely due to the bovine P[5] component of the RotaTeq® vaccine, for which a separate hemi-nested RT-PCR with specific bovine primers would have had to be used to identify the P[5] component. Due to time constraints, this was not performed for these samples.

Discussion

This 2016 ARSP report describes the distribution of rotavirus genotypes and geographic differences of rotavirus strains causing disease in Australia, for the period of 1 January to 31 December 2016. A reduction in confirmed rotavirus positive samples was observed during 2016, where only 230 samples were confirmed as positive with a wildtype rotavirus strain, and another 184 with a vaccine-like rotavirus strain. Of the 230 wildtype rotavirus specimens, 29% were

Table 5: Rotavirus G and P genotype distribution in children over 5 years of age and adults, 1 January to 31 December 2016

Centre	Type total	G1P[8]		G2P[4]		G3P[8]		G3P[8]*		G9P[8]		G12P[8]		Other†		Non-type‡		Neg
		%	n	%	n	%	n	%	n	%	n	%	n	%	n	%	n	n
Australian Capital Territory																		
ACT	1	-	0	100	1	-	0	-	0	-	0	-	0	-	0	-	0	0
New South Wales																		
Prince of Wales Hospital	12	-	0	75	9	-	0	17	2	8	1	-	0	-	0	-	0	0
Westmead	4	25	1	75	3	-	0	-	0	-	0	-	0	-	0	-	0	0
Westmead - CIDM	2	-	0	-	0	-	0	-	0	-	0	50	1	50	1	-	0	2
John Hunter	7	-	0	29	2	-	0	57	4	14	1	-	0	-	0	-	0	2
Other§	7	-	0	29	2	-	0	57	4	-	0	-	0	-	0	14	1	3
NSW subtotal:	32	3	1	50	16	0	0	31	10	6	2	3	1	3	1	3	1	7
Queensland																		
Pathology Brisbane	2	-	0	-	0	-	0	50	1	-	0	50	1	-	0	-	0	4
Qld Regional	3	-	0	67	2	33	1	-	0	-	0	-	0	-	0	-	0	72
Queensland subtotal:	5	-	0	40	2	20	1	20	1	-	0	20	1	-	0	-	0	76
South Australia																		
Adelaide	58	12	7	16	9	7	4	10	6	10	6	22	13	21	12	2	1	52
Tasmania																		
Hobart	2	-	0	-	0	-	0	50	1	50	1	-	0	-	0	-	0	0
Victoria																		
Royal Children's Hospital	4	-	0	-	0	-	0	-	0	25	1	50	2	-	1	-	0	14
Western Australia																		
PathWest	21	-	0	38	8	-	0	29	6	19	4	-	0	14	3	-	0	4
TOTAL	123	7	8	29	36	4	5	20	24	11	14	14	17	14	17	2	2	153

* Equine-like G3P[8]
† See Table 3
‡ A specimen where G and/or P genotype was not determined
§ NSW: Faecal specimens which were received from Douglas Hanly Moir Pathology, NSW & Pathology Brisbane, Qld

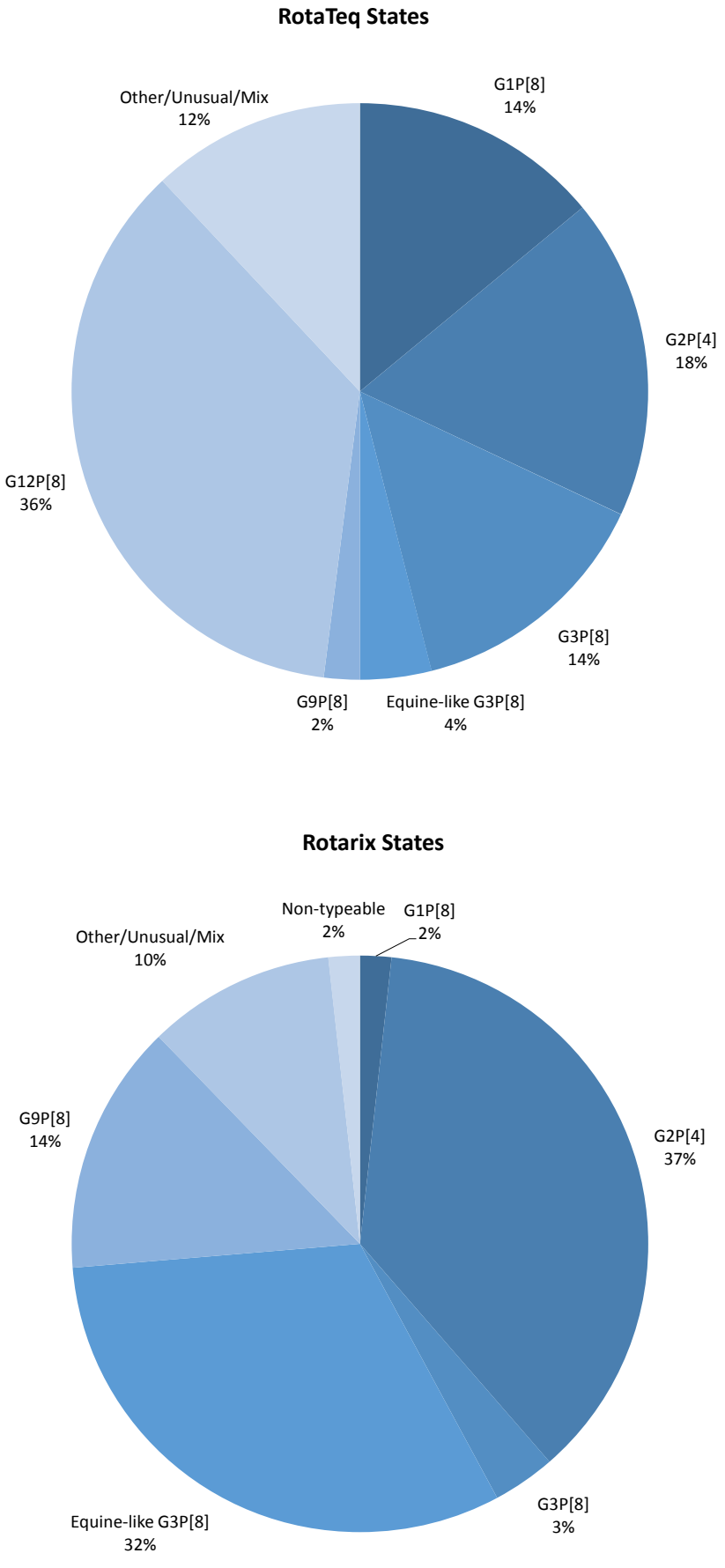

Figure: Overall distribution of wildtype rotavirus G- and P- genotypes identified in Australian children under 5 years of age, based on vaccine usage, Australia, 1 January to 31 December 2016

Table 6: Age distribution of gastroenteritis cases, where a rotavirus vaccine component was identified:

Age (months)	Age (years)	n=	% of total
0-6		166	90.2
7-12	≤1	16	8.7
13-24	1-2	1	0.5
37-48	3-4	1	0.5
Total		184	-

genotyped as G2P[4], 19% as equine-like G3P[8], and 15% as G12P[8], ending the four-year dominance of G12P[8] nationally.[23] However, distinct differences in genotype distribution based on vaccine usage continued to occur. As previously reported, G12P[8] was more common in States administering RotaTeq® compared to Rotarix®, whereas G2P[4] and equine-like G3P[8] was more prominent in locations using Rotarix®.[23] This ongoing distinction in circulating rotavirus genotypes between vaccine groups has not been reported previously in other countries that have had both vaccines added to their NIP, most likely due to the lack of geographical segregation of vaccine use. Only one vaccine-related trend has been described in the literature, where G2P[4] rotavirus gastroenteritis was associated with countries that use the Rotarix® vaccine, including Austria, Australia, Belgium, and countries within Latin America.[24-29] However, there still is a lack of evidence that could distinguish whether these observations are due to a temporal coincidence, since G2P[4] was found indiscriminately in both vaccinated and unvaccinated countries, or vaccine-induced immunological pressure, since the Rotarix® G1P[8] vaccine is less efficient against the heterotypic G2P[4] strain.[29,30]

Of the 184 samples identified as vaccine-strain, 166 were primarily from infants aged 0-6 months (90.2%), where subclinical shedding of rotavirus vaccine is expected.[31] A change in diagnostic methods at collaborating laboratories from less-sensitive ELISA or latex agglutination assays (conventional), to highly sensitive real time qRT-PCR, is the most likely cause for such an increase in rotavirus vaccine positive stool specimens, since conventional methods generally would not detect low level vaccine shedding.[32] This would also help explain the unusually high amount of negative or unconfirmed rotavirus cases reported for this year (n=482/949). It is plausible that the acute gastroenteritis for these patients was instead caused by another agent, however the ARSP does not have access to patient comorbidity records to comment further. On the other hand, two patients aged 20 months and 40 months were found to have vaccine-like rotavirus strain present in their stool specimens. It is possible that the vaccine strain was present in these two patients after horizontal transmission from a recently vaccinated sibling, a vaccine/wildtype reassortment strain infection, or the patient was immunocompromised and therefore unable to clear the vaccine efficiently.[19,33] Full genome characterisation of these two strains would help elucidate the cause of vaccine components being present in these older patients.

Despite a marked drop in overall rotavirus positive samples from 1,031 in 2015, to 230 in 2016, the number of specimens in which an unusual genotype was identified was similar to the number detected in 2015.[23] Furthermore, the genotypes identified within both surveillance periods included strains such as feline/canine G3 (P[3] or P[9]), G8 (P[4], P[8], or P[14]), G10P[14] and G12 (P[4] or P[6]).[23] These observations are unusual, in that a particular uncommon strain may occur sporadically within a year as a single case or a sporadic localised outbreak, rather than persisting across multiple years in different States across Australia. An example of this is the feline/canine-like G3P[3], which was detected in Australian States: NT (n=4), SA (n=1), and WA (n=1) in 2015, and in the NT (n=2) in 2016. This strain (with either P[3] or P[9]) has also been reported in multiple countries such as South Korea, China, Brazil, and the USA, but only as 1-3 cases in total across multiple years.[34-38] Similarly, bovine-human strains such as G6P[14] and G8P[14], were considered uncommon in Australia; identified in 6 individual cases over an 11-year surveillance period when combined (1997-2007).[12] In 2016 alone, 4 G6P[14] and 2

Table 7: Rotavirus G and P genotypes identified in rotavirus vaccine-like cases:

		P[8]	P[nt]
Rotarix®	G1 (Rix)	11	-
	G1 (Teq)	21	48
	G2	1	3
	G4	3	7
RotaTeq®*	G6 (VP7 seq)	3	-
	Mixed G1/G2/ G3/G4	7	3
***130/173 genotyped**	Mixed G1/G2/G4	1	-
	Mixed G1/G3	1	2
	Mixed G1/G3/G4	2	1
	Mixed G1/G4	3	8
	G-non typeable	2	14

G8P[14] strains have been identified, together with other uncommon human-animal reassortant rotavirus strains, such as G10P[14].

Animal rotavirus strains are considered to be attenuated to humans, however, multiple reassortment events between human and animal strains can lead to chimeric viruses that have more human segments, increasing their ability to infect and replicate within a human host.[39,40] This improved adaptation to the human host, together with a lack of pre-existing population immunity against such new strains, creates a niche for novel human-animal reassortant rotaviruses that have the potential to spread globally and persist within the human population, as seen with G9 and G12 strains.[41,42] These two strains were considered to be the product of multiple reassortment events between human and swine rotavirus.[40] The factors behind this increase in animal-human reassortant strains need to be elucidated, as these novel strains have the potential to become epidemiologically important. The recent global emergence of an equine-human reassortment G3P[8] strain, predominantly in vaccinated countries, raised the question of whether the increase in zoonotic strains was due to immunological pressures from the vaccine itself. Indeed, the prolonged differences in genotype diversity between vaccine groups described here suggests that the vaccines may be inducing selective pressures that favour certain genotypes. However, the introduction of vaccines cannot be the sole reason for the observed increase in zoonotic strain prevalence, as such events occurred prior to vaccine introduction, as shown with G9 and G12 strains.[40] Nevertheless, continued surveillance is vital for understanding how vaccines can affect rotavirus evolution and genotype diversity. Furthermore, continued epidemiological surveillance will gain insight into how these changes in rotavirus diversity can alter vaccine effectiveness in children.

Despite the continuous changes in circulating rotavirus genotypes, the introduction of both RotaTeq® and Rotarix® to the Australian NIP has substantially impacted on the rotavirus burden in Australia. It is estimated that for the 6 years post vaccine implementation, ~77,000 hospitalisations and ~3 deaths were prevented; 90% of which were for children under 5 years of age.[11] Such an impact was less noticeable in other healthcare outcomes, suggesting that there has been a shift in the severity of symptoms from severe to less critical outcomes.[11] This report supports the idea that vaccine implementation to the Australian NIP has been effective on reducing the burden caused by rotavirus infections, as shown by the decrease in rotavirus positive samples received for 2016. This decrease in sample number appears to be a true depiction of rotavirus epidemiology in Australia, when compared to available State-specific notifiable disease reports. For 2016, Western Australia reported an annual crude rate of 6.6/100,000 rotavirus infections per population, compared to an average of 16.9/100,000 over the preceding four years.[43]

In this 2016 annual report, an overall reduction in rotavirus positive samples was described. G2P[4], equine-like G3P[8], and G12P[8] continue to cause significant disease in Australia, however G12P[8] impacted more in RotaTeq® States, while equine-like G3P[8] and G2P[4] were associated with States administering Rotarix®. The continued dominance of G12 in RotaTeq® states only, and the increase in occurrence of novel strains such as the various animal-like G3, G8, G10, P[3], P[5] and P[14] strains, demonstrate a highly evolving and hard to predict trend in circulating genotypes since vaccine introduction to the Australian NIP. The contin-

ued variations in the wildtype strain population will remain a challenge to vaccine effectiveness and will require continued monitoring.

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Quarterly report

Invasive Pneumococcal Disease Surveillance, 1 January to 31 March 2017

Kate Pennington and the Enhanced Invasive Pneumococcal Disease Surveillance Working Group, for the Communicable Diseases Network Australia

Summary

The number of notified cases of invasive pneumococcal disease (IPD) in the first quarter of 2017 was less than the previous quarter, but greater than the number of notified cases in the first quarter of 2016. Overall, the decline in disease due to the serotypes targeted by the 13-valent pneumococcal conjugate vaccine (13vPCV) has been maintained across all age groups since the 13vPCV replaced the 7-valent pneumococcal conjugate vaccine (7vPCV) in the childhood immunisation program from July 2011 (Figure 1).

Key points

In the first quarter of 2017, there were 247 cases of IPD reported to the National Notifiable Disease Surveillance System (NNDSS). This represented a 38% decrease compared to the fourth quarter of 2016 (n=395) and a 35% increase when compared with the same period in 2016 (n=183) (Table 3). In the first quarter of 2017 the most common pneumococcal serotypes causing IPD were 3 (11%), 19F (7%), 22F (6%) and 23B (6%) (Table 2).

In non-Indigenous Australians this quarter, the number of notified cases was highest in children aged less than 5 years and older adult age groups, especially those aged 60 years or older (Table 3). In Indigenous Australians, cases were highest in children aged less than 5 years, and the 40-44 and 50-54 years age groups. The proportion of cases reported as Indigenous Australians this quarter (11%; 28/247) was lower compared to the proportion observed in the previous quarter (18%; 45/395), and similar compared to the proportion reported in the first quarter of 2016 (11%; 26/183).

In children aged less than 5 years, there were 46 cases of IPD reported, representing 19% of all cases reported in this quarter. The proportion of cases notified in this age group was higher in this reporting period when compared with the previous quarter (14%; 54/395), and similar compared to the proportion reported in the first quarter of 2016 (19%; 34/183). Of those cases with a known serotype reported this quarter, 33% (11/33) were due to a serotype included in the 13vPCV compared with 56% (23/41) of cases in the previous and 35% (9/26) in the first quarter of 2016 (Figure 2). During this quarter there were a number of different serotypes affecting this age group with no clear dominance (Table 2). Serotypes 3, 19A and 23B continued to be the common serotypes reported amongst this age group.

In the first quarter of 2017, there were four cases reported in fully vaccinated children aged less than 5 years who were considered to be 13vPCV failures. These 13vPCV failures were due to serotypes 19A (n=2) and 19F (n=2) (Table 4).

Among Indigenous Australians aged 50 years and over, there were 10 cases of IPD reported this quarter. Of those cases with a reported serotype (n=9), only two were due to a serotype included in the 23-valent pneumococcal polysaccharide vaccine (23vPPV) and overall there was no particular serotype dominant (Figure 3). The number of notified cases of IPD in this

age group were less than the number of cases reported in both the previous quarter (n=14) and the first quarter of 2016 (n=12).

Among non-Indigenous Australians* aged 65 years and over there were 80 cases of IPD reported this quarter. The number of notified cases of IPD in this age group decreased by 43% when compared to the previous quarter (n=142) but was 40% higher than the number reported in the first quarter of 2016 (n=57). Of those cases with a reported serotype, 61% (46/76) were due to a serotype included in the 23vPPV (Figure 4), which was similar to the proportion in the previous quarter (63%; 86/137). For this quarter, serotypes 3 (n=9), 11A (n=7) and 19F (n=7) were the most common serotypes for this population group, noting that these three serotypes are included in the 23vPPV.

During this quarter there were 19 deaths attributed to a variety of IPD serotypes, with serotype 3 (n=5) predominant. Almost all of the reported deaths (18/19) occurred in non-Indigenous Australians*. The median age of those cases who died was 78 years (range 1 to 96 years).

Notes

The data in this report are provisional and subject to change as laboratory results and additional case information become available. More detailed data analysis of IPD in Australia and surveillance methodology are described in the IPD annual report series published in *Communicable Diseases Intelligence*.

In Australia, pneumococcal vaccination is recommended as part of routine immunisation for children, individuals with specific underlying conditions associated with increased risk of IPD and older Australians. More information on the scheduling of the pneumococcal vaccination can be found on the Immunise Australia Program website (www.immunise.health.gov.au).

In this report, a 'vaccine failure' is reported when a child aged less than 5 years is diagnosed with IPD due to a serotype found in the 13vPCV and they have received 3 primary scheduled doses of 13vPCV at least 2 weeks prior to disease onset with at least 28 days between doses of vaccine.

There are 3 pneumococcal vaccines available in Australia, each targeting multiple serotypes (Table 5). Note that in this report serotype analysis is generally grouped according to vaccine composition.

Follow-up of all notified cases of IPD is undertaken in all states and territories except New South Wales and Victoria who conduct targeted follow-up of notified cases aged under 5 years, and 50 years or over for enhanced data. Follow-up of notified cases of IPD in Queensland is undertaken in all areas except Metro South and Gold Coast Public Health Units who conduct targeted follow-up of notified cases for those aged under 5 years only. However, in these areas where targeted case follow-up is undertaken, some enhanced data may also be available outside these targeted age groups.

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Report prepared with the assistance of Mr Mark Trungove and Ms Rachael Corvisy on behalf of the Enhanced Invasive Pneumococcal Disease Surveillance Working Group.

Enhanced Invasive Pneumococcal Disease Surveillance Working Group contributors to this report include (in alphabetical order): Frank Beard (NCIRS), Heather Cook (NT and secretariat), Lucinda Franklin (Vic.), Carolien Giele (WA), Robin Gilmour (NSW), Michelle Harlock (Tas.), Ben Howden (Microbiological Diagnostic Unit, University of Melbourne), Sanjay Jayasinghe (NCIRS), Vicki Krause (Chair), Shahin Oftadeh (Centre for Infectious Diseases and Microbiology Laboratory Services, NSW Health Pathology), Sue Reid (ACT), Vitali Sintchenko (Centre for Infectious Diseases and Microbiology- Public Health, Westmead Hospital), Helen Smith (Queensland Health Forensic and Scientific Services), Janet Strachan (Vic.), Hannah Vogt (SA), Angela Wakefield (Qld).

* Non-Indigenous Australians includes cases reported with as non-Indigenous, not stated, blank or unknown.

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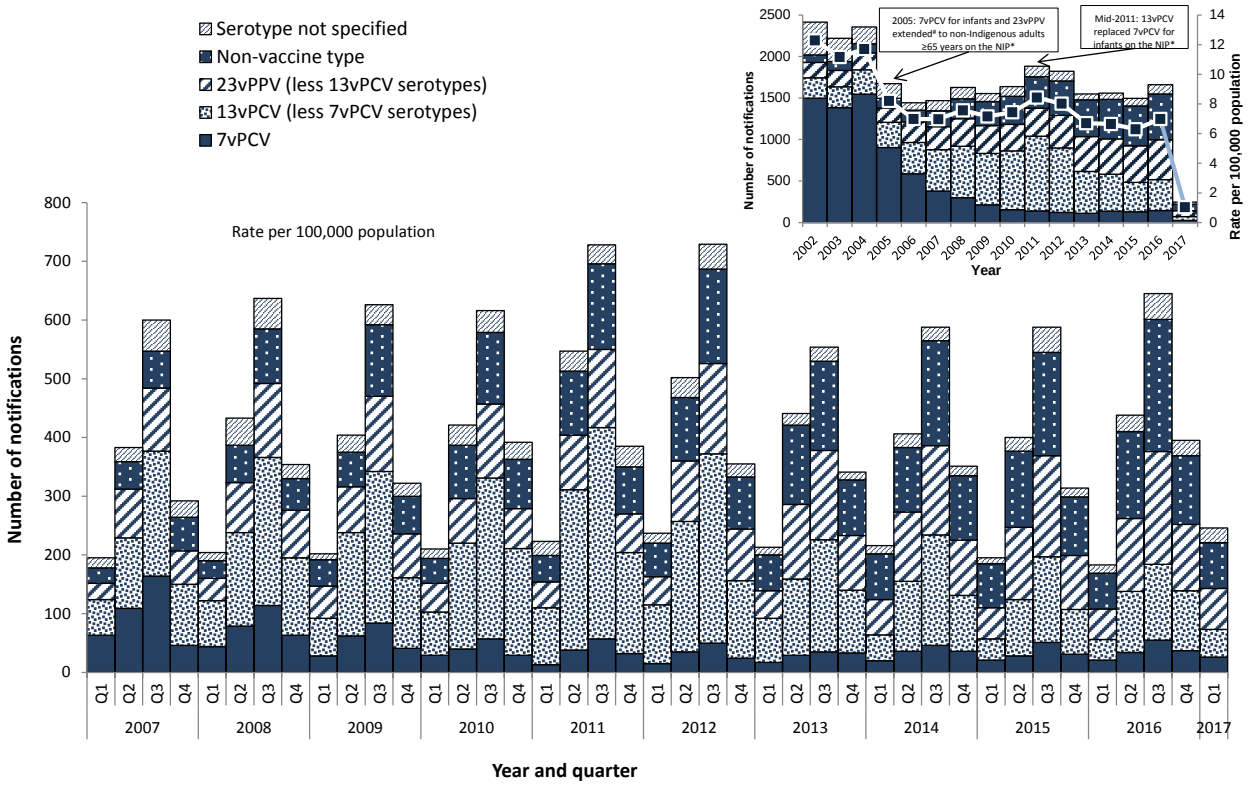
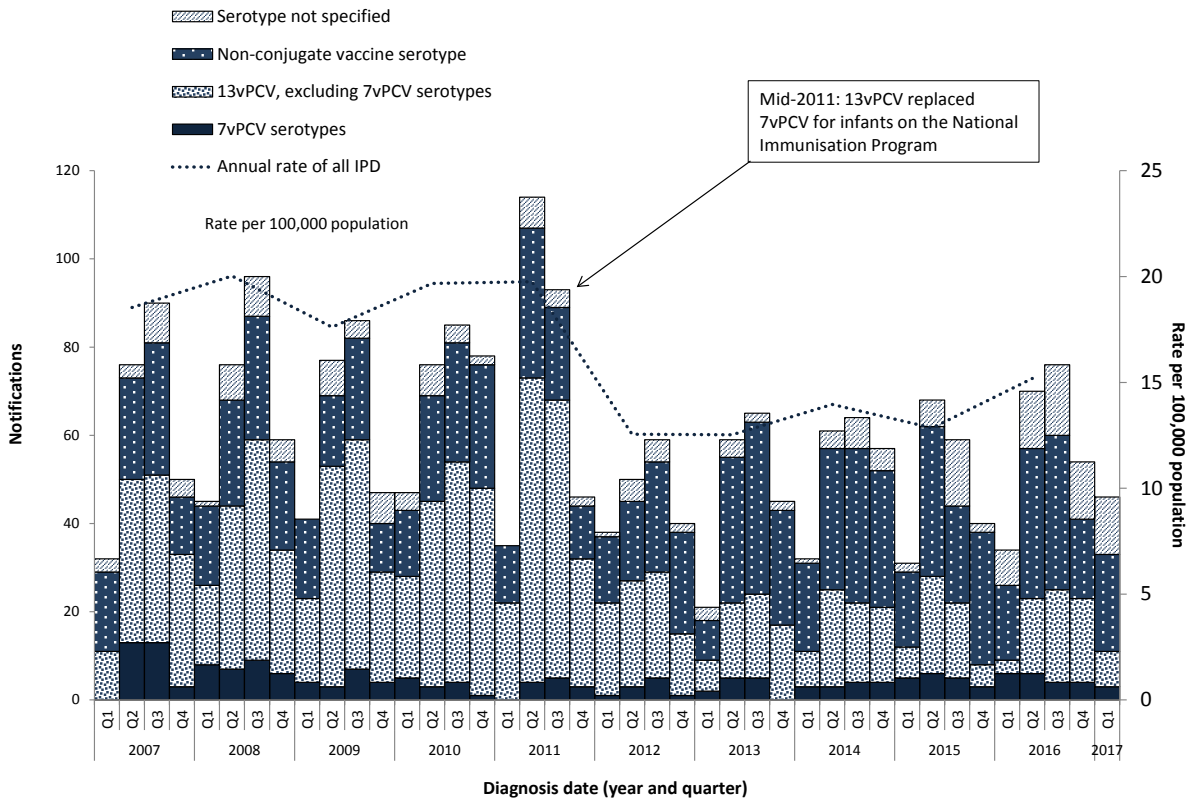
Figure 1: Notifications of invasive pneumococcal disease, Australia, 1 January 2002 to 31 March 2017, by vaccine serotype group, year and quarter



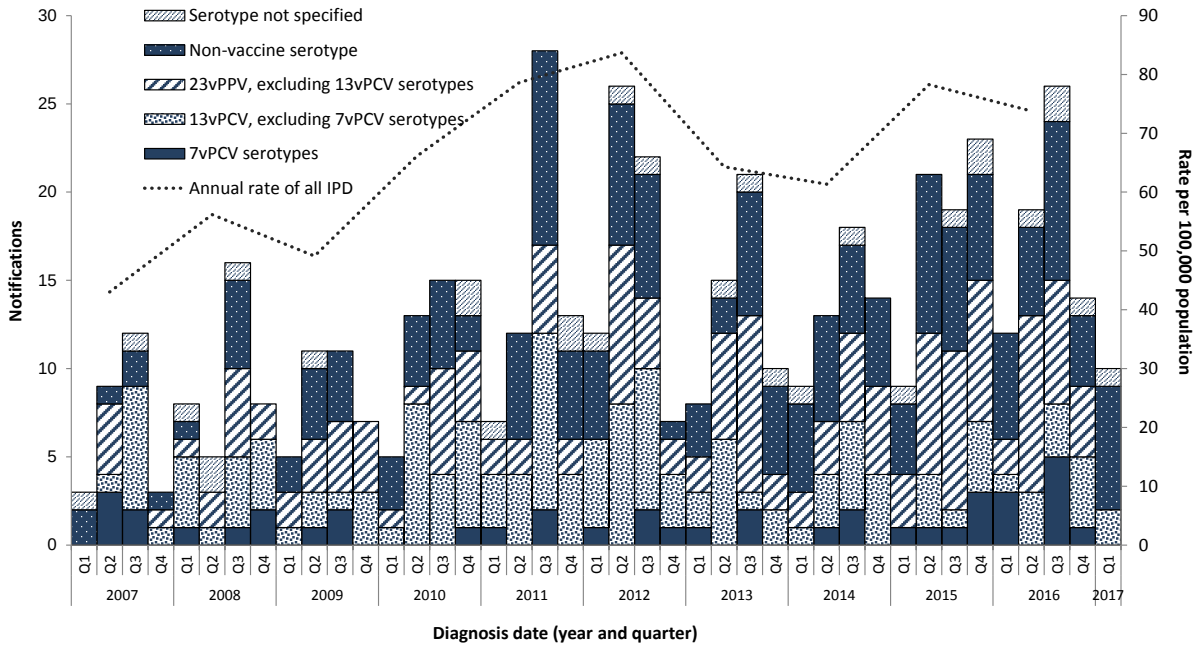
1999 - 23vPPV funded for all Indigenous Australians aged 50 years and over, as well as younger Indigenous Australian adults with risk factors.
* NIP - National Immunisation Program.

Figure 2: Notifications and annual rates* of invasive pneumococcal disease in children aged less than 5 years, Australia, 1 January 2007 to 31 March 2017, by vaccine serotype group



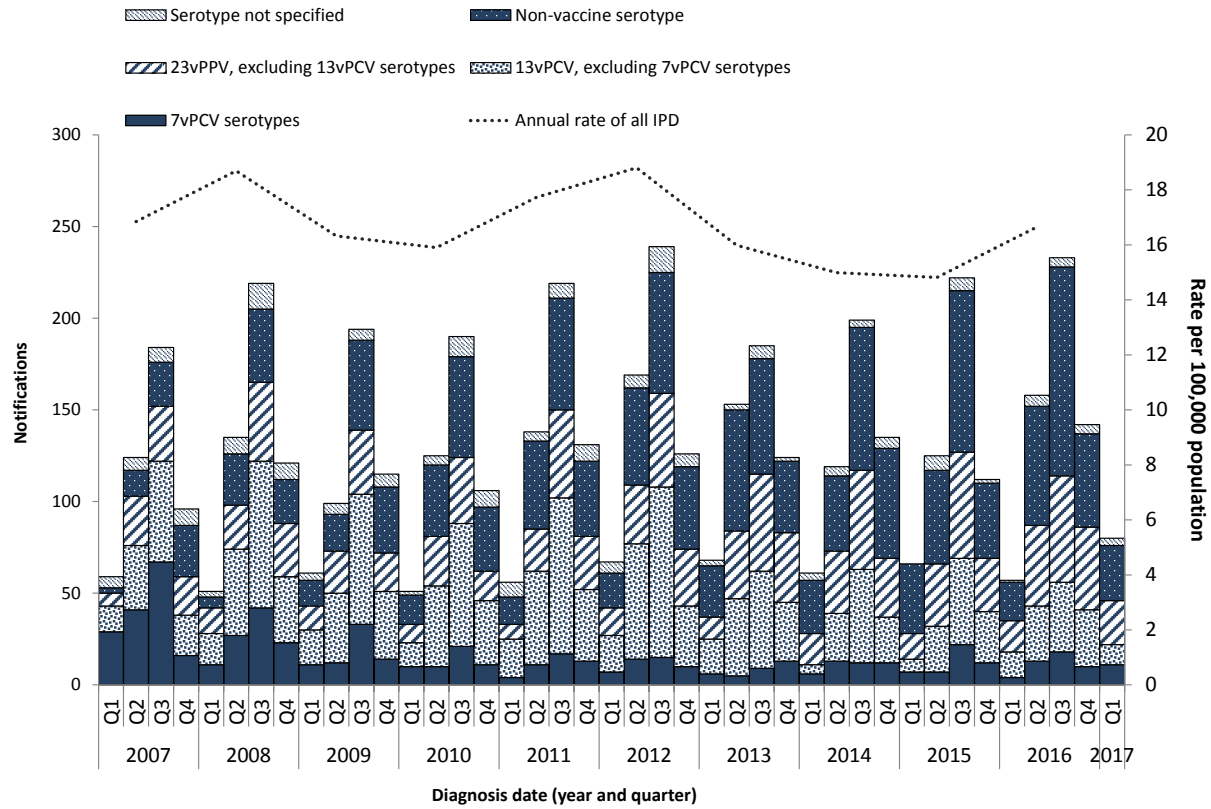
* Annual rates are shown on quarter 2, excluding 2017.

Figure 3: Notifications and annual rates* of all invasive pneumococcal disease in Indigenous Australians aged 50 years or over, Australia, 1 January 2007 to 31 March 2017, by vaccine serotype group



* Annual rates are shown on quarter 2, excluding 2017.

Figure 4: Notifications and annual rates* of all invasive pneumococcal disease in non-indigenous Australians# aged 65 years or over, Australia, 1 January 2007 to 31 March 2017, by vaccine serotype group



* Annual rates are shown on quarter 2, excluding 2017.
# Non-Indigenous Australians includes cases reported with as non-Indigenous, not stated, blank or unknown.

Table 1: Notified cases of invasive pneumococcal disease, Australia, 1 January to 31 March 2017, by Indigenous status, serotype completeness and state or territory

Indigenous status	ACT	NSW	NT	Qld	SA	Tas	Vic	WA	Total 1st qtr 2017	Total 4th qtr 2016	Total 1st qtr 2016	Year to date 2017
Indigenous	0	8	4	6	2	0	1	7	28	45	26	28
Non-Indigenous	2	59	1	35	25	7	37	20	186	308	144	186
Not stated / Unknown	0	12	0	0	0	0	20	1	33	42	13	33
Total	**2**	**79**	**5**	**41**	**27**	**7**	**58**	**28**	**247**	**395**	**183**	**247**
Indigenous status completeness* (%)	100	85	100	100	100	100	66	96	87	89	93	87
Indigenous status completeness in targeted groups *† (%)	100	87	100	100	100	100	90	94	93	96	99	93
Serotype completeness ‡ (%)	100	90	100	98	52	86	98	93	89	93	92	89

* Indigenous status completeness is defined as the reporting of a known Indigenous status, excluding the reporting of not stated or unknown Indigenous status.
† Targeted groups for followup by almost all jurisdictions and public health units are cases aged less than 5 years and 50 years and over.
‡ Serotype completeness is the proportion of all cases of invasive pneumococcal disease that were reported with a serotype or reported as non-typable. Incomplete serotype data can occur in cases when (i) no isolate was available as diagnosis was by polymerase chain reaction and no molecular typing was attempted or was not possible due to insufficient genetic material; (ii) the isolate was not referred to the reference laboratory or was not viable; (iii) typing was pending at the time of reporting, or no serotype was reported by the notifying jurisdiction to the National Notifiable Diseases Surveillance System.

Table 2: Distribution of serotypes causing invasive pneumococcal disease in notified cases, Australia, 1 January to 31 March 2017, by age group

Serotype	Vaccine type	Age groups			Serotype total
		Under 5 years	5-64 years	Over 65 years	
3	13vPCV non-7vPCV	4	13	9	26
19F	7vPCV	3	8	7	18
22F	23vPPV non-13vPCV	2	8	5	15
23B	Non-vaccine type	4	6	4	14
19A	13vPCV non-7vPCV	4	5	3	12
9N	23vPPV non-13vPCV	-	9	2	11
11A	23vPPV non-13vPCV	-	2	7	9
23A	Non-vaccine type	1	4	4	9
15A	Non-vaccine type	3	1	4	8
7F	13vPCV non-7vPCV	-	8	-	8
8	23vPPV non-13vPCV	-	6	2	8
15B	23vPPV non-13vPCV	4	1	1	6
16F	Non-vaccine type	-	4	2	6
33F	23vPPV non-13vPCV	-	5	1	6
6C	Non-vaccine type	1	2	3	6
10A	23vPPV non-13vPCV	1	1	3	5
12F	23vPPV non-13vPCV	1	2	2	5
15C	Non-vaccine type	2	2	1	5
35B	Non-vaccine type	-	2	3	5
Other	-	3	21	15	39
Unknown	-	13	9	4	26
Total		**46**	**119**	**82**	**247**

* Serotypes that only occur in less than 5 cases per quarter are grouped as 'Other' and include 'non-typable' isolates this quarter.
† 'Serotype unknown' includes those serotypes reported as 'no isolate', 'not referred', 'not viable', 'typing pending' and 'untyped'.

Table 3: Notified cases of invasive pneumococcal disease, Australia, 1 January to 31 March 2017, by Indigenous status and age group

Age group	Indigenous status			Total
	Indigenous	Non-Indigenous	Not reported*	
00-04	4	40	2	46
05-09	0	4	1	5
10-14	1	1	0	2
15-19	1	1	1	3
20-24	1	3	1	5
25-29	1	2	3	6
30-34	3	4	2	9
35-39	1	8	1	10
40-44	5	3	5	13
45-49	1	6	6	13
50-54	6	13	0	19
55-59	1	12	0	13
60-64	1	17	3	21
65-69	0	10	1	11
70-74	1	15	3	19
75-79	1	20	1	22
80-84	0	5	1	6
85+	0	22	2	24
Total	28	186	33	247

* Not reported is defined as not stated, blank or unknown Indigenous status.

Table 4: Characteristics of 13vPCV failures in children aged less than 5 years, Australia, 1 January to 31 March 2017

Age	Indigenous status	Serotype	Clinical category	Risk factor/s
2 years	Non-Indigenous	19A	Pneumonia	Other
3 years	Non-Indigenous	19F	Pneumonia	No risk factor identified
3 years	Non-Indigenous	19F	Pneumonia	Childcare attendee
3 years	Non-Indigenous	19A	Bacteraemia	Childcare attendee

Table 5: Streptococcus pneumoniae serotypes targeted by pneumococcal vaccines

Serotypes	7-valent pneumococcal conjugate vaccine (7vPCV)	10-valent pneumococcal conjugate vaccine (10vPCV)	13-valent pneumococcal conjugate vaccine (13vPCV)	23-valent pneumococcal polysaccharide vaccine (23vPPV)
1		✓	✓	✓
2				✓
3			✓	✓
4	✓	✓	✓	✓
5		✓	✓	✓
6A			✓	
6B	✓	✓	✓	✓
7F		✓	✓	✓
8				✓
9N				✓
9V	✓	✓	✓	✓
10A				✓
11A				✓
12F				✓
14	✓	✓	✓	✓
15B				✓
17F				✓
18C	✓	✓	✓	✓
19A			✓	✓
19F	✓	✓	✓	✓
20				✓
22F				✓
23F	✓	✓	✓	✓
33F				✓

Quarterly report

Invasive Pneumococcal Disease Surveillance, 1 April to 30 June 2017

Kate Pennington and the Enhanced Invasive Pneumococcal Disease Surveillance Working Group, for the Communicable Diseases Network Australia

Summary

The number of notified cases of invasive pneumococcal disease (IPD) in the second quarter of 2017 was greater than the previous quarter and also the second quarter of 2016. Following the July 2011 replacement of the 7-valent pneumococcal conjugate vaccine (7vPCV) in the childhood immunisation program with the 13-valent pneumococcal conjugate vaccine (13vPCV), there was an initial relatively rapid decline in disease due to the additional six serotypes covered by the 13vPCV across all age groups, however more recently this rate of decline has slowed. Additionally, over this period the number of cases due to the eleven serotypes additionally covered by the 23-valent pneumococcal polysaccharide vaccine (23vPPV) and also those serotypes not covered by any available vaccine has been increasing steadily across all age groups

Key points

In the second quarter of 2017, there were 495 cases of IPD reported to the National Notifiable Disease Surveillance System (NNDSS). This represented an almost doubling in cases (n=250) compared to the number of cases notified in the previous quarter, however, compared to the same quarter in 2016 there was only a 14% increase in the number of cases (n=436) (Table 1). This increase tended to be consistent with the seasonal increase in cases observed in quarters two and three each year (Figure 1). In the second quarter of 2017, the most common pneumococcal serotypes causing IPD were 3 (14%), 22F (8%) and 19A (6%) (Table 2).

Among non-Indigenous Australians this quarter, the number of notified cases continued to be highest in children aged less than 5 years and older adult age groups, particularly those aged 60 years or older (Table 3). Among Indigenous Australians, cases were highest in children aged less than 5 years, and the 45-59 years age groups. The proportion of cases reported as Indigenous Australians this quarter (8%; 41/495) was lower compared to the proportion observed in the previous quarter (12%; 29/250), and similar compared to the proportion reported in the second quarter of 2016 (7%; 32/436).

In children aged less than 5 years, there were 86 cases of IPD reported, representing 17% of all cases reported in this quarter. The proportion of cases notified in this age group was lower in this reporting period when compared with the previous quarter (19%; 47/250), and similar compared to the proportion reported in the second quarter of 2016 (16%; 70/436). Of those cases with a known serotype reported this quarter, 49% (25/51) were due to a serotype included in the 13vPCV, compared with 33% (11/33) of cases in the previous quarter and 40% (23/57) in the second quarter of 2016 (Figure 2). During this quarter the main serotypes affecting this age group were 3 (27%; 14/51), followed by 19A (10%; 5/51) and 19F (10%; 5/51) (Table 2). All of these serotypes are included in the 13vPCV.

In the first quarter of 2017, there were 21 cases reported in fully vaccinated children aged less than 5 years who were considered to be 13vPCV

failures. These 13vPCV failures were due to serotypes 3 (n=13), 19A (n=5), 19F (n=2) and 18C (n=1) (Table 4).

Among Indigenous Australians aged 50 years and over, there were 18 cases of IPD reported this quarter. Of those cases with a reported serotype (n=15), eight (53%) were due to a serotype included in the 23vPPV and overall there was no particular serotype dominant (Figure 3). The number of notified cases of IPD in this age group was less than the number of cases reported in the previous quarter (n=11), but similar to the number reported in the second quarter of 2016 (n=19).

Among non-Indigenous Australians* aged 65 years and over there were 184 cases of IPD reported this quarter. The number of notified cases of IPD in this age group was more than two-times the number of cases reported in the previous quarter (n=80) and 16% higher than the number reported in the second quarter of 2016 (n=158). Of those cases with a reported serotype (n=169), almost two-thirds (62%; 105/169) were due to a serotype included in the 23vPPV (Figure 4), which was similar to the proportion in the previous quarter (60%; 47/78). For this quarter, serotypes 3 (n=25), 22F (n=22) and 23A (n=16) were the most common serotypes for this population group, noting that only serotypes 3 and 22F are included in the 23vPPV.

During this quarter there were 35 deaths attributed to a variety of IPD serotypes, with serotypes 3 (n=7) and 11A (n=4) the most common. Almost all of the reported deaths (91%; n=32) occurred in non-Indigenous Australians*. The median age of those cases who died was 74 years (range 1 to 94 years).

Notes

The data in this report are provisional and subject to change as laboratory results and additional case information become available. More detailed data analysis of IPD in Australia and surveillance methodology are described in the IPD annual report series published in *Communicable Diseases Intelligence*.

In Australia, pneumococcal vaccination is recommended as part of routine immunisation for children, individuals with specific underlying conditions associated with increased risk of IPD and older Australians. More information on the scheduling of the pneumococcal vaccination can be found on the Immunise Australia Program website (www.immunise.health.gov.au).

In this report, a 'vaccine failure' is reported when a child aged less than 5 years is diagnosed with IPD due to a serotype found in the 13vPCV and they have received 3 primary scheduled doses of 13vPCV at least 2 weeks prior to disease onset with at least 28 days between doses of vaccine.

There are 3 pneumococcal vaccines available in Australia, each targeting multiple serotypes (Table 5). Note that in this report serotype analysis is generally grouped according to vaccine composition.

Follow-up of all notified cases of IPD is undertaken in all states and territories except New South Wales and Victoria who conduct targeted follow-up of notified cases aged under 5 years, and 50 years or over for enhanced data. Follow-up of notified cases of IPD in Queensland is undertaken in all areas except Metro South and Gold Coast Public Health Units who conduct targeted follow-up of notified cases for those aged under 5 years only. However, in these areas where targeted case follow-up is undertaken, some enhanced data may also be available outside these targeted age groups.

Acknowledgements

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Enhanced Invasive Pneumococcal Disease Surveillance Working Group contributors to this report include (in alphabetical order): Frank

* Non-Indigenous Australians includes cases reported with as non-Indigenous, not stated, blank or unknown.

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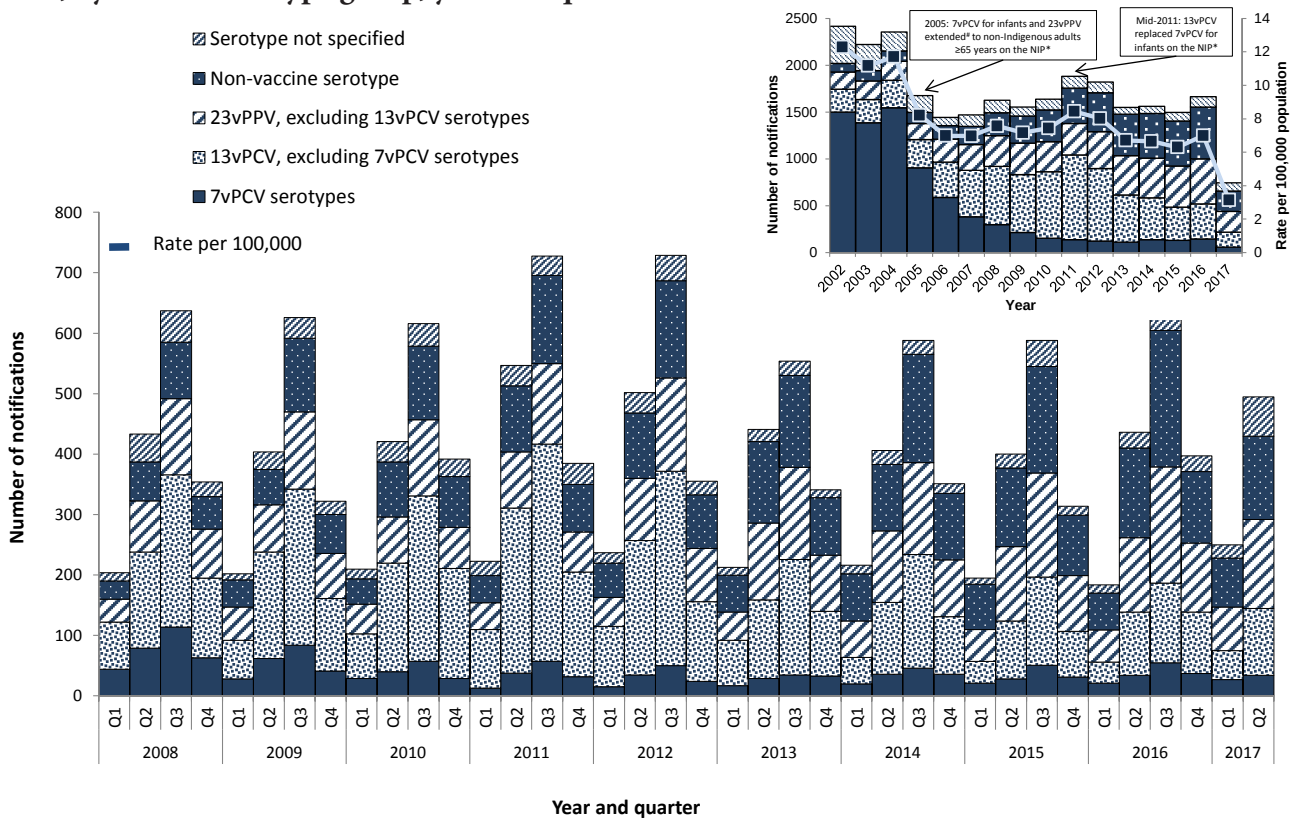
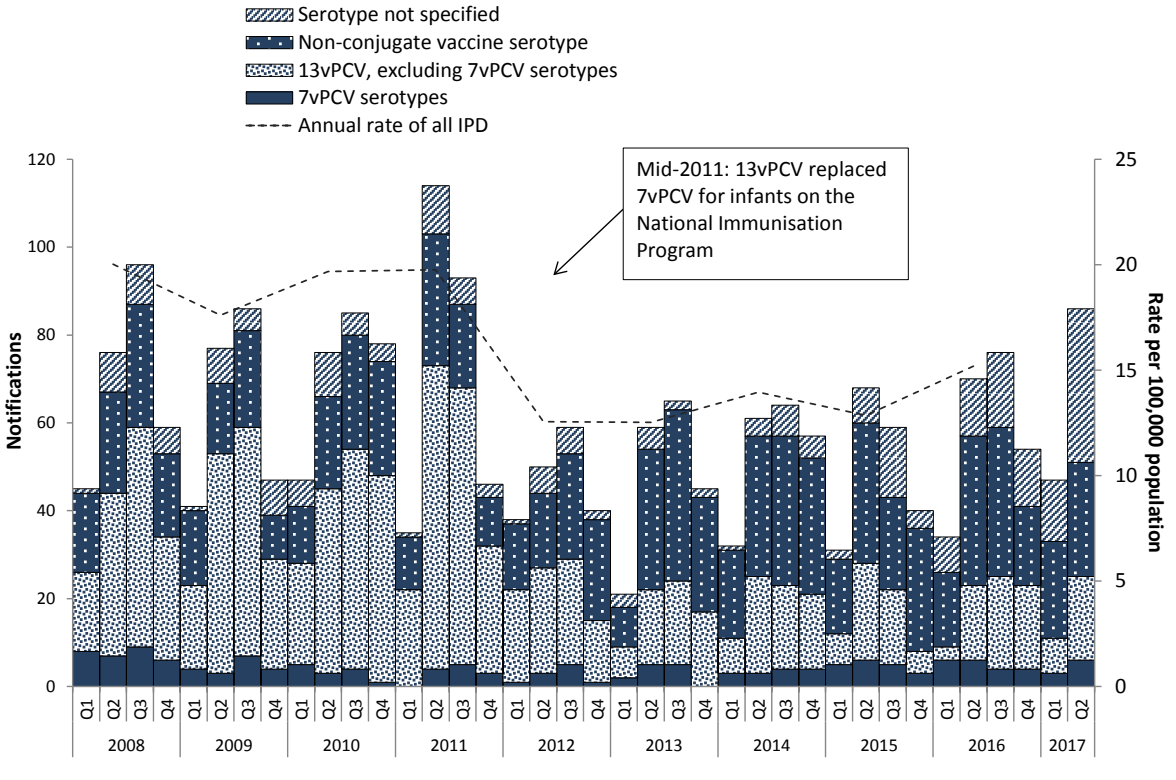
Figure 1: Notifications of invasive pneumococcal disease, Australia, 1 January 2002 to 30 June 2017, by vaccine serotype group, year and quarter



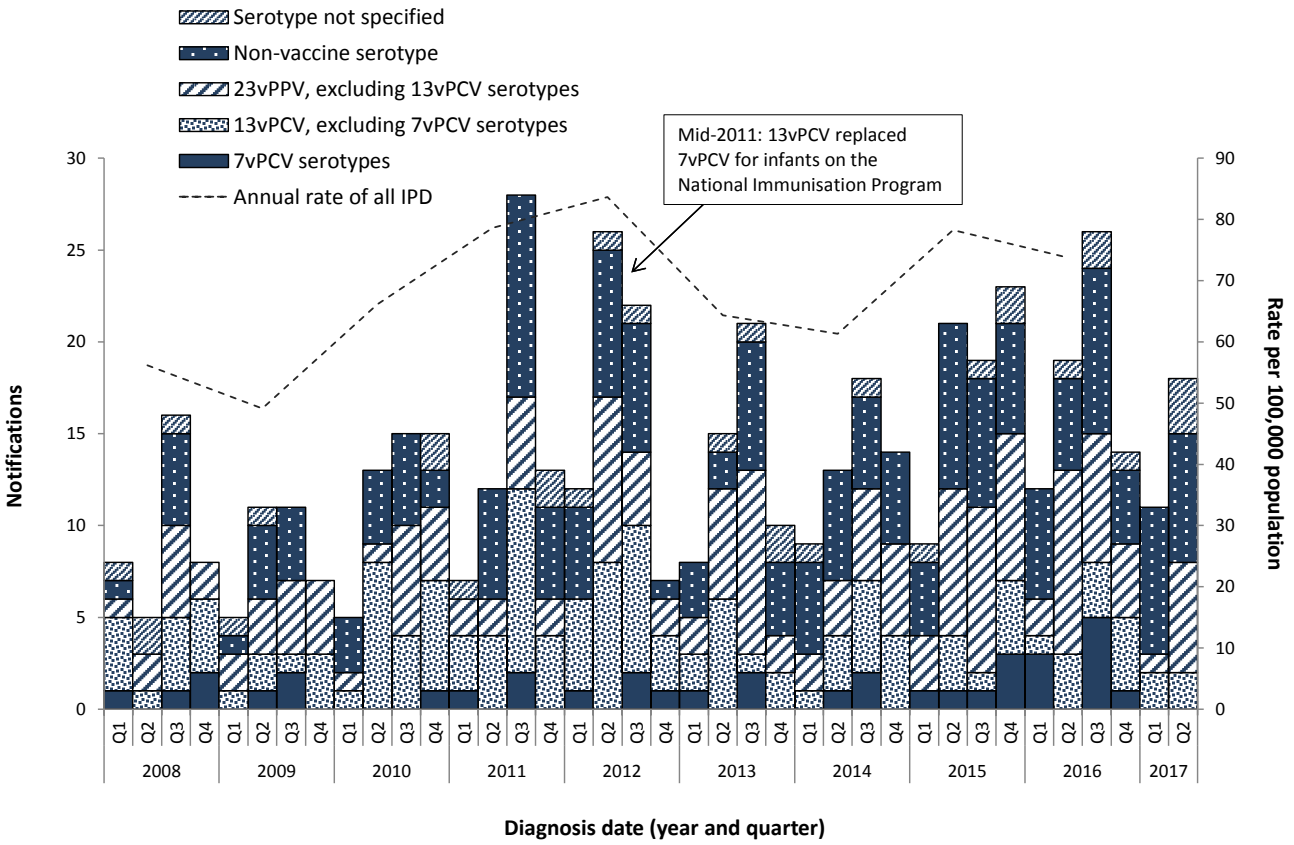
In 1999 the 23vPPV funded for all Indigenous Australians aged 50 years and over, as well as younger Indigenous Australian adults with risk factors.
* NIP - National Immunisation Program.

Figure 2: Notifications and annual rates* of invasive pneumococcal disease in children aged less than 5 years, Australia, 1 January 2008 to 30 June 2017, by vaccine serotype group



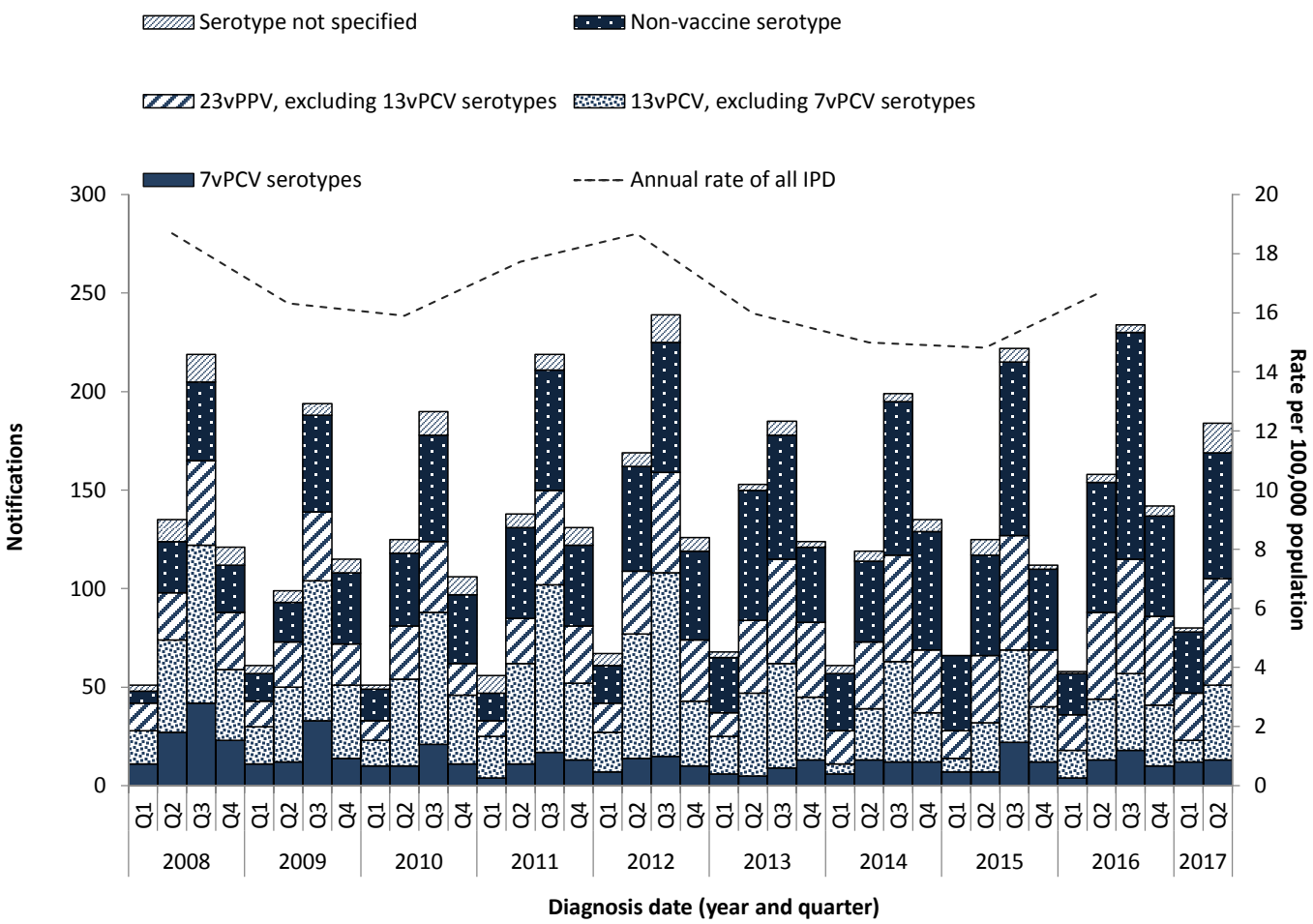
* Annual rates are shown on quarter 2, excluding 2017.

Figure 3: Notifications and annual rates* of all invasive pneumococcal disease in Indigenous Australians aged 50 years or over, Australia, 1 January 2008 to 30 June 2017, by vaccine serotype group



* Annual rates are shown on quarter 2, excluding 2017.

Figure 4: Notifications and annual rates* of all invasive pneumococcal disease in non-indigenous Australians# aged 65 years or over, Australia, 1 January 2008 to 30 June 2017, by vaccine serotype group



* Annual rates are shown on quarter 2, excluding 2017.

Non-Indigenous Australians includes cases reported with as non-Indigenous, not stated, blank or unknown.

Table 1: Notified cases of invasive pneumococcal disease, Australia, 1 April to 30 June 2017, by Indigenous status, serotype completeness and state or territory

Indigenous status	ACT	NSW	NT	Qld	SA	Tas	Vic	WA	Total 2nd qtr 2017	Total 1st qtr 2017	Total 2nd qtr 2016	Year to date 2017
Indigenous	0	3	11	10	6	0	2	9	41	29	32	70
Non-Indigenous	6	137	4	61	53	6	69	40	376	188	367	564
Not stated / Unknown	0	24	0	0	0	1	52	1	78	33	37	111
Total	6	164	15	71	59	7	123	50	495	250	436	745
Indigenous status completeness* (%)	100	85	100	100	100	86	58	98	84	87	92	85
Indigenous status completeness in targeted groups *† (%)	100	91	100	100	100	83	70	97	89	93	99	91
Serotype completeness ‡ (%)	100	82	93	96	64	86	98	92	87	92	96	89

* Indigenous status completeness is defined as the reporting of a known Indigenous status, excluding the reporting of not stated or unknown Indigenous status.
† Targeted groups for followup by almost all jurisdictions and public health units are cases aged less than 5 years and 50 years and over.
‡ Serotype completeness is the proportion of all cases of invasive pneumococcal disease that were reported with a serotype or reported as non-typable. Incomplete serotype data can occur in cases when (i) no isolate was available as diagnosis was by polymerase chain reaction and no molecular typing was attempted or was not possible due to insufficient genetic material; (ii) the isolate was not referred to the reference laboratory or was not viable; (iii) typing was pending at the time of reporting, or no serotype was reported by the notifying jurisdiction to the National Notifiable Diseases Surveillance System.

Table 2: Distribution of serotypes causing invasive pneumococcal disease in notified cases, Australia, 1 April to 30 June 2017, by age group

Serotype	Vaccine type	Age groups			Serotype total
		Under 5 years	5-64 years	Over 65 years	
3	13vPCV non-7vPCV	14	29	25	68
22F	23vPPV non-13vPCV	3	14	22	39
19A	13vPCV non-7vPCV	5	15	10	30
9N	23vPPV non-13vPCV	1	19	7	27
19F	7vPCV	5	8	10	23
23A	Non-vaccine type	-	6	16	22
23B	Non-vaccine type	3	11	7	21
11A	23vPPV non-13vPCV	1	12	7	20
15A	Non-vaccine type	1	7	11	19
8	23vPPV non-13vPCV	-	15	3	18
35B	Non-vaccine type	3	4	7	14
6C	Non-vaccine type	2	6	6	14
16F	Non-vaccine type	-	7	6	13
7F	13vPCV non-7vPCV	-	10	2	12
33F	23vPPV non-13vPCV	1	6	4	11
15B	23vPPV non-13vPCV	3	2	5	10
10A	23vPPV non-13vPCV	1	7	1	9
17F	23vPPV non-13vPCV	2	2	4	8
35F	Non-vaccine type	-	3	4	7
Other	-	8	23	16	39
Unknown	-	33	14	16	26
Total		**86**	**220**	**189**	**495**

* Serotypes that only occur in less than 5 cases per quarter are grouped as 'Other' and include 'non-typable' isolates this quarter.
† 'Serotype unknown' includes those serotypes reported as 'no isolate', 'not referred', 'not viable', 'typing pending' and 'untyped'.

Table 3: Notified cases of invasive pneumococcal disease, Australia, 1 April to 30 June 2017, by Indigenous status and age group

Age group	Indigenous status			Total
	Indigenous	Non-Indigenous	Not reported*	
00-04	7	76	3	86
05-09	0	11	2	13
10-14	1	1	0	2
15-19	1	2	1	4
20-24	0	2	4	6
25-29	3	3	5	11
30-34	2	6	3	11
35-39	3	6	3	12
40-44	1	7	7	15
45-49	5	10	10	25
50-54	6	25	7	38
55-59	4	24	4	32
60-64	3	42	6	51
65-69	1	41	3	45
70-74	2	24	1	27
75-79	0	32	4	36
80-84	1	24	7	32
85+	1	40	8	49
Total	41	376	78	495

* Not reported is defined as not stated, blank or unknown Indigenous status.

Table 4: Characteristics of 13vPCV failures in children aged less than 5 years, Australia, 1 April to 30 June 2017

Age	Indigenous status	Serotype	Clinical category	Risk factor/s
8 months	Indigenous	19A	Pneumonia	Other
1 year	Non-Indigenous	19A	Bacteraemia	No data available
1 year	Non-Indigenous	3	Pneumonia and other (pleural effusion)	No risk factor identified
1 year	Non-Indigenous	3	Pneumonia and other (pleural effusion)	Premature (<37 weeks gestation)
1 year	Non-Indigenous	3	Pneumonia and other (pleural effusion)	No risk factor identified
1 year	Non-Indigenous	19A	Bacteraemia	No data available
1 year	Non-Indigenous	19A	Pneumonia	Childcare attendee
1 year	Non-Indigenous	3	Pneumonia and other (pleural effusion)	No data available
2 years	Non-Indigenous	3	Pneumonia and other (pleural effusion)	No data available
2 years	Non-Indigenous	19A	Bacteraemia	No risk factor identified
2 years	Non-Indigenous	3	Pneumonia and other (pleural effusion)	Childcare attendee
2 years	Non-Indigenous	3	Pneumonia	No data available
2 years	Non-Indigenous	3	Pneumonia and other (pleural effusion)	No data available
3 years	Non-Indigenous	3	Pneumonia	Childcare attendee
3 years	Non-Indigenous	3	Pneumonia	Other
3 years	Non-Indigenous	3	Pneumonia	No data available
3 years	Non-Indigenous	19F	No data provided	Other
3 years	Non-Indigenous	19F	Bacteraemia	No data available
3 years	Non-Indigenous	3	Pneumonia and other (pleural effusion)	No data available
3 years	Non-Indigenous	18C	Bacteraemia	No data available
4 years	Non-Indigenous	3	Pneumonia and other (pleural empyema)	No risk factor identified

Table 5: *Streptococcus pneumoniae* serotypes targeted by pneumococcal vaccines

Serotypes	7-valent pneumococcal conjugate vaccine (7vPCV)	10-valent pneumococcal conjugate vaccine (10vPCV)	13-valent pneumococcal conjugate vaccine (13vPCV)	23-valent pneumococcal polysaccharide vaccine (23vPPV)
1		✓	✓	✓
2				✓
3			✓	✓
4	✓	✓	✓	✓
5		✓	✓	✓
6A			✓	
6B	✓	✓	✓	✓
7F		✓	✓	✓
8				✓
9N				✓
9V	✓	✓	✓	✓
10A				✓
11A				✓
12F				✓
14	✓	✓	✓	✓
15B				✓
17F				✓
18C	✓	✓	✓	✓
19A			✓	✓
19F	✓	✓	✓	✓
20				✓
22F				✓
23F	✓	✓	✓	✓
33F				✓

Quarterly report

The Australian Sentinel Practices Research Network, 1 January to 31 March 2017

Monique Chilver, Daniel Blakeley and Nigel Stocks

Introduction

The Australian Sentinel Practices Research Network (ASPREN) is a national influenza and infectious diseases surveillance system that is funded by the Australian Government Department of Health. ASPREN was established by the Royal Australian College of General Practitioners in 1991 and is currently directed through the Discipline of General Practice at the University of Adelaide.

The network consists of general practitioners and nurse practitioners, Australia wide, who report syndromic presentations on a number of defined medical conditions each week. ASPREN was established in 1991 to provide a rapid monitoring scheme for infectious diseases that can inform public health officials of the epidemiology of pandemic threats in the early stages of a pandemic, as well as play a role in the evaluation of public health campaigns and research of conditions commonly seen in general practice. Reporters currently submit data via automated data extraction from patient records, web-based data collection or paper forms.

The list of conditions reported for syndromic surveillance is reviewed annually by the ASPREN management committee. In 2017, 4 conditions were being monitored. They were influenza-like illness (ILI), gastroenteritis and two varicella infections (chickenpox and shingles). Definitions of these conditions are described in surveillance systems reported in CDI, published in *Commun Dis Intell* 2016;40(1):11.

In 2010, virological surveillance was established allowing ASPREN practitioners to collect nasal swab samples for laboratory viral testing a systematic sample of ILI patients for a range of respiratory viruses including influenza A and influenza B.

Results

Sentinel practices contributing to ASPREN were located in all 8 states and territories in Australia. A total of 203 general practitioners regularly contributed data to ASPREN in the 1st quarter of 2017. Each week an average of 173 general practitioners provided information to ASPREN with an average of 13,912 (range 11,597 to 14,935) consultations per week and an average of 97 (range 75 to 123) notifications per week (all conditions).

ILI rates reported from 1 January to 31 March 2017 averaged 2.5 cases per 1,000 consultations (range 1.2 to 4.5 cases per 1,000 consultations). This was higher than the rates in the same reporting period in 2016, which averaged 1.8 cases per 1,000 consultations (range 0.8 to 3.5 cases per 1,000 consultations, Figure 1).

The ASPREN ILI swab testing program continued in 2017 with 106 tests being undertaken from 1 January to 31 March. The most commonly reported virus during this reporting period was rhinovirus (26% of all swabs performed), with the second most common virus being influenza A (19% of all swabs performed,

Figure 2). This was higher than seen in the same reporting period in 2016 where rhinovirus and influenza accounted for 10% and 7% of all swabs performed, respectively.

From the beginning of 2017 to the end of week 13, there were 25 cases of influenza detected, with 20 of these typed as influenza A (19% of all swabs performed) and the remaining 5 being influenza B (5% of all swabs performed) (Figure 2).

During this reporting period, consultation rates for gastroenteritis averaged 3.8 cases per 1,000 consultations (range 2.4 to 5.6 cases per 1,000, Figure 3). This was lower than the rates in the same reporting period in 2016 where the average was 4.6 cases per 1,000 consultations (range 3.5 to 6.0 cases per 1,000).

Varicella infections were reported at a similar rate for the 1st quarter of 2017 compared with the same period in 2016. From 1 January to 31 March 2017, recorded rates for chickenpox averaged 0.1 cases per 1,000 consultations (range 0.0 to 0.8 cases per 1,000 consultations, Figure 4).

In the 1st quarter of 2017, reported rates for shingles averaged 1 case per 1,000 consultations (range 0.3 to 1.8 cases per 1,000 consultations, Figure 5). This was similar to the rates in the same reporting period in 2016 where the average shingles rate was 1.1 cases per 1,000 consultations (range 0.6 to 2.3 cases per 1,000 consultations).

Figure 1: Consultation rates for influenza-like illness, ASPREN, 2016 and 1 January to 31 March 2017, by week of report

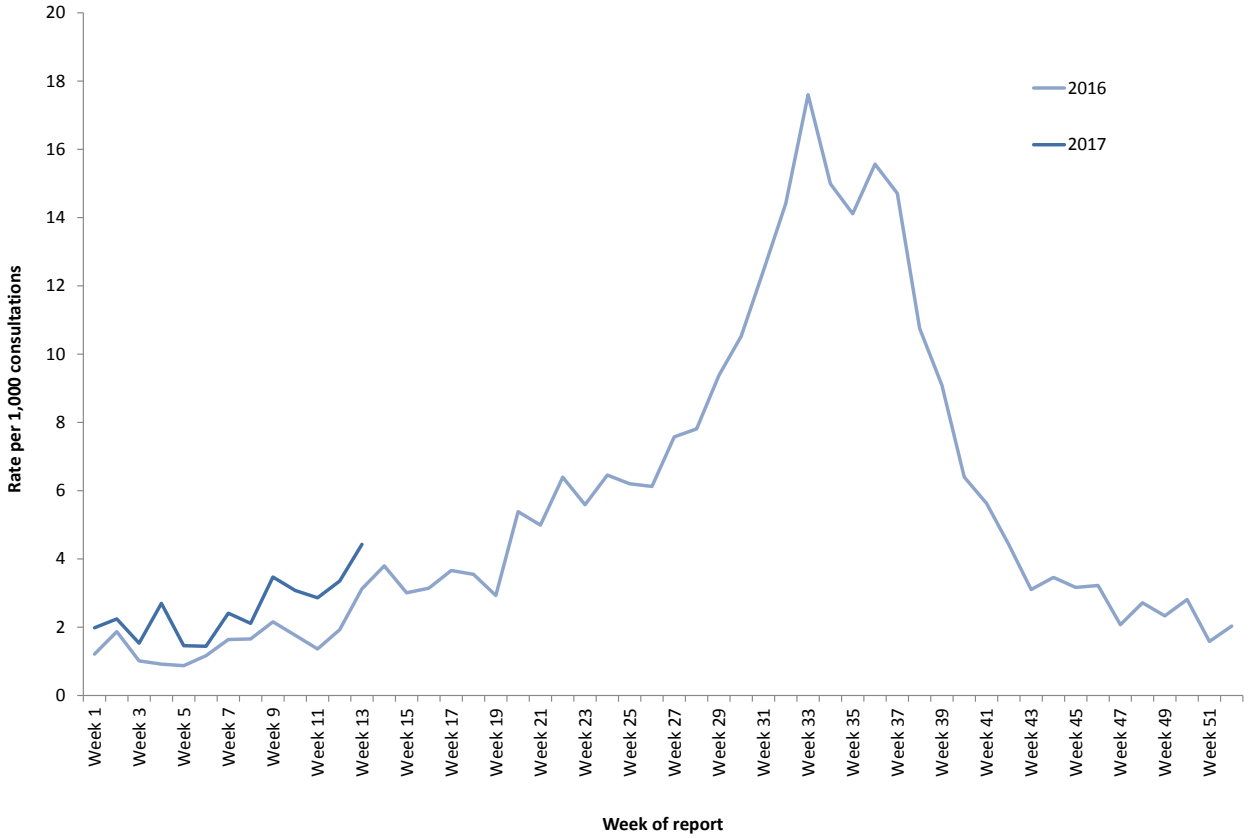


Figure 2: Influenza-like illness swab testing results, ASPREN, 1 January to 31 March 2017, by week of report

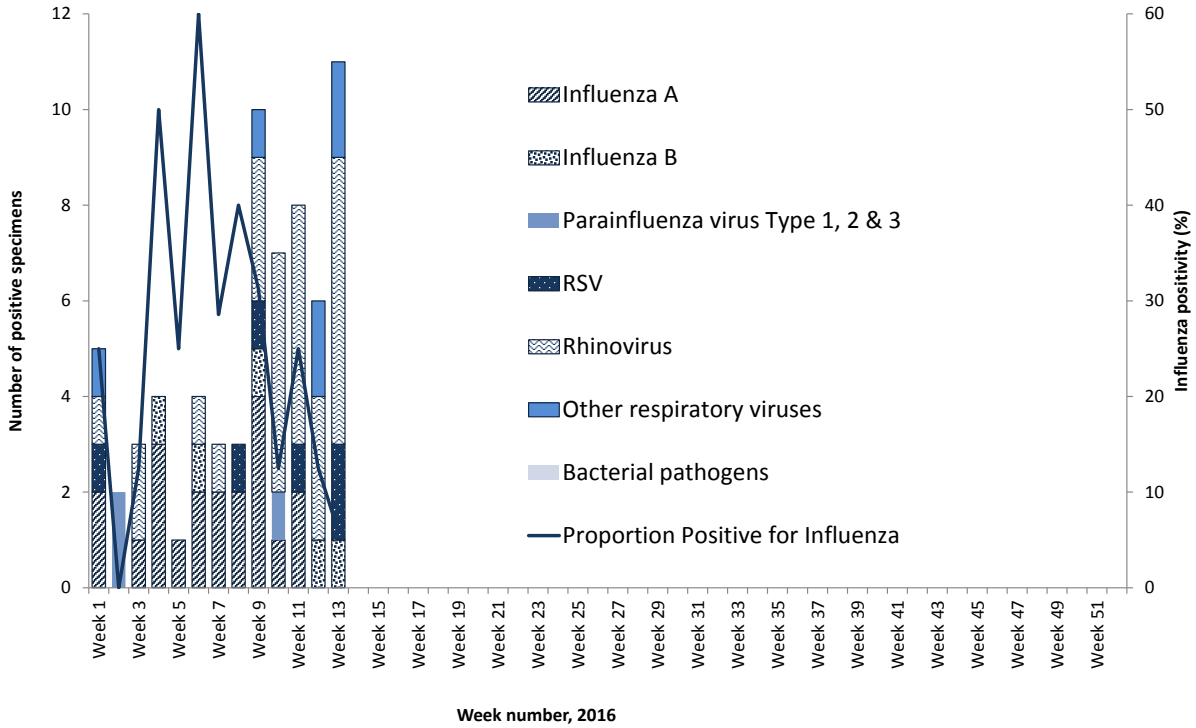


Figure 3: Consultation rates for gastroenteritis, ASPREN, 2016 and 1 January to 31 March 2017, by week of report

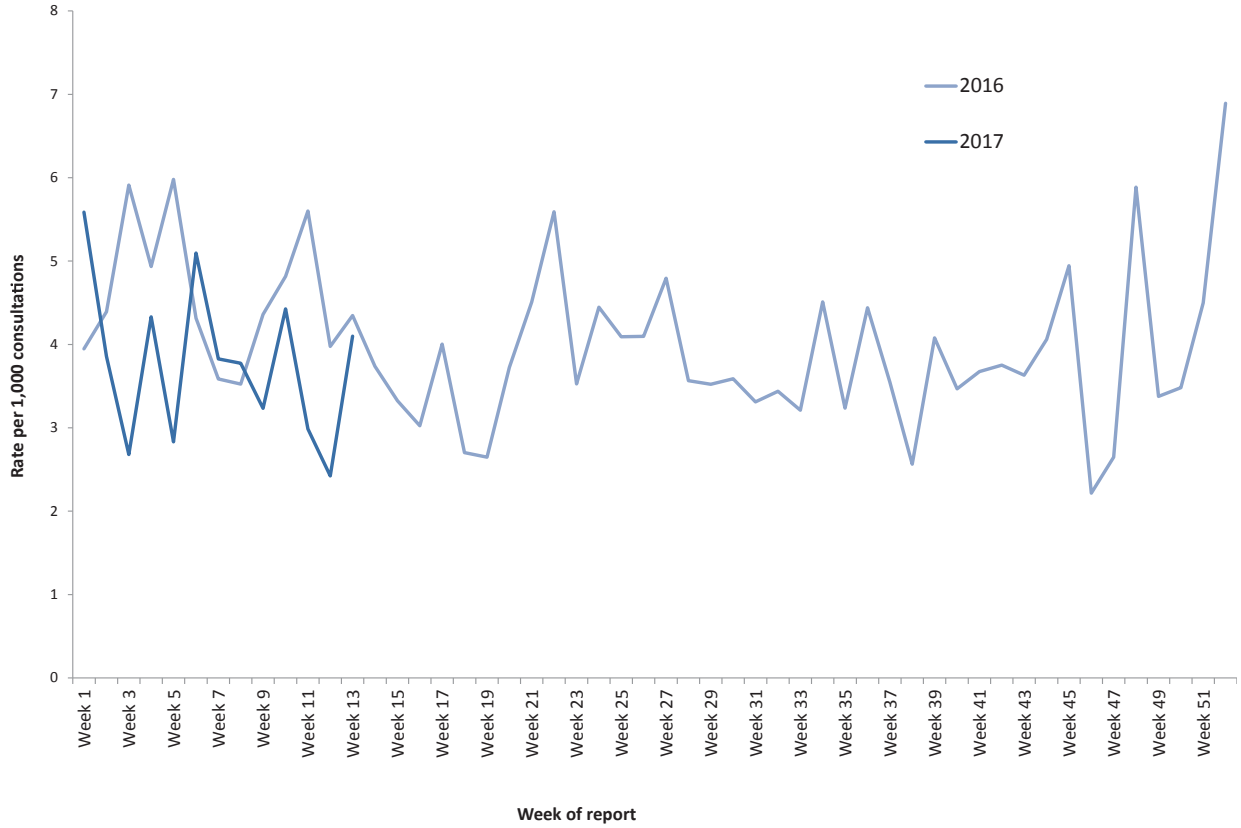


Figure 4: Consultation rates for chickenpox, ASPREN, 2016 and 1 January to 31 March 2017, by week of report

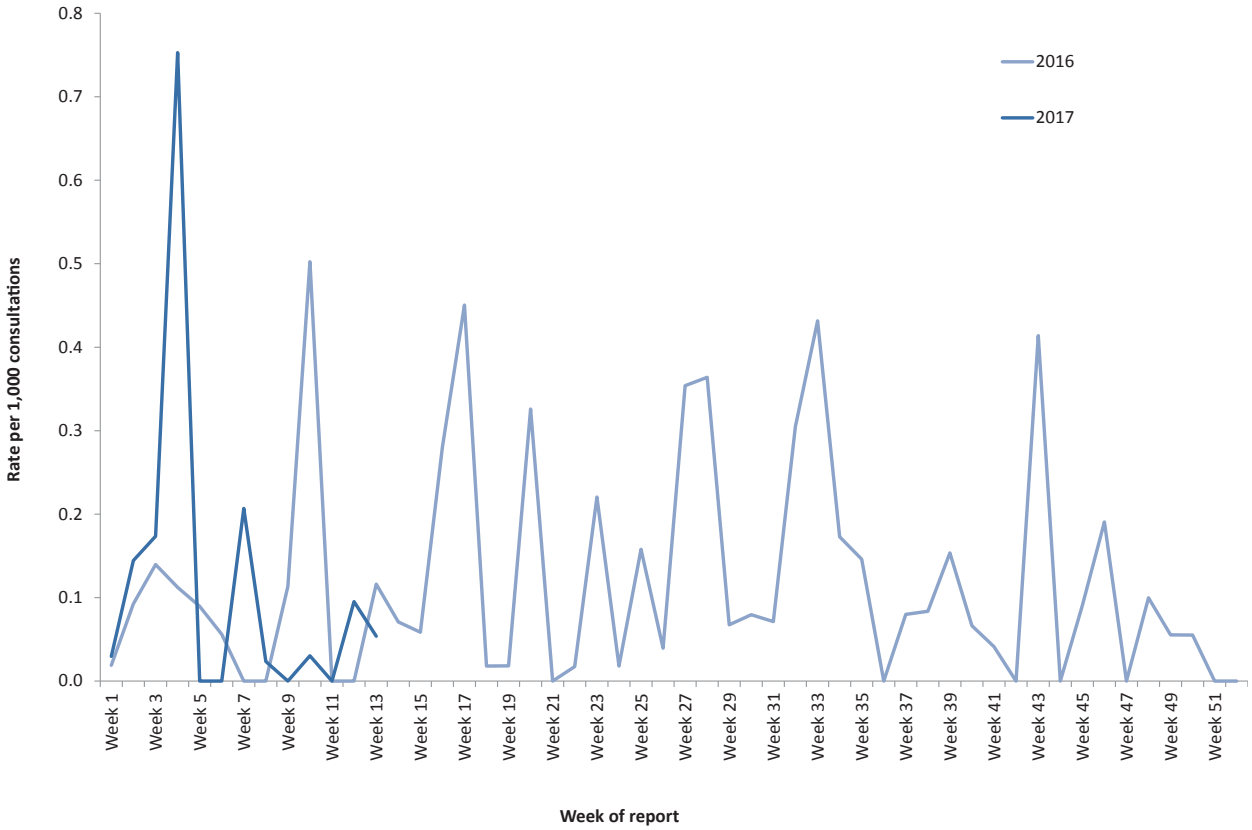
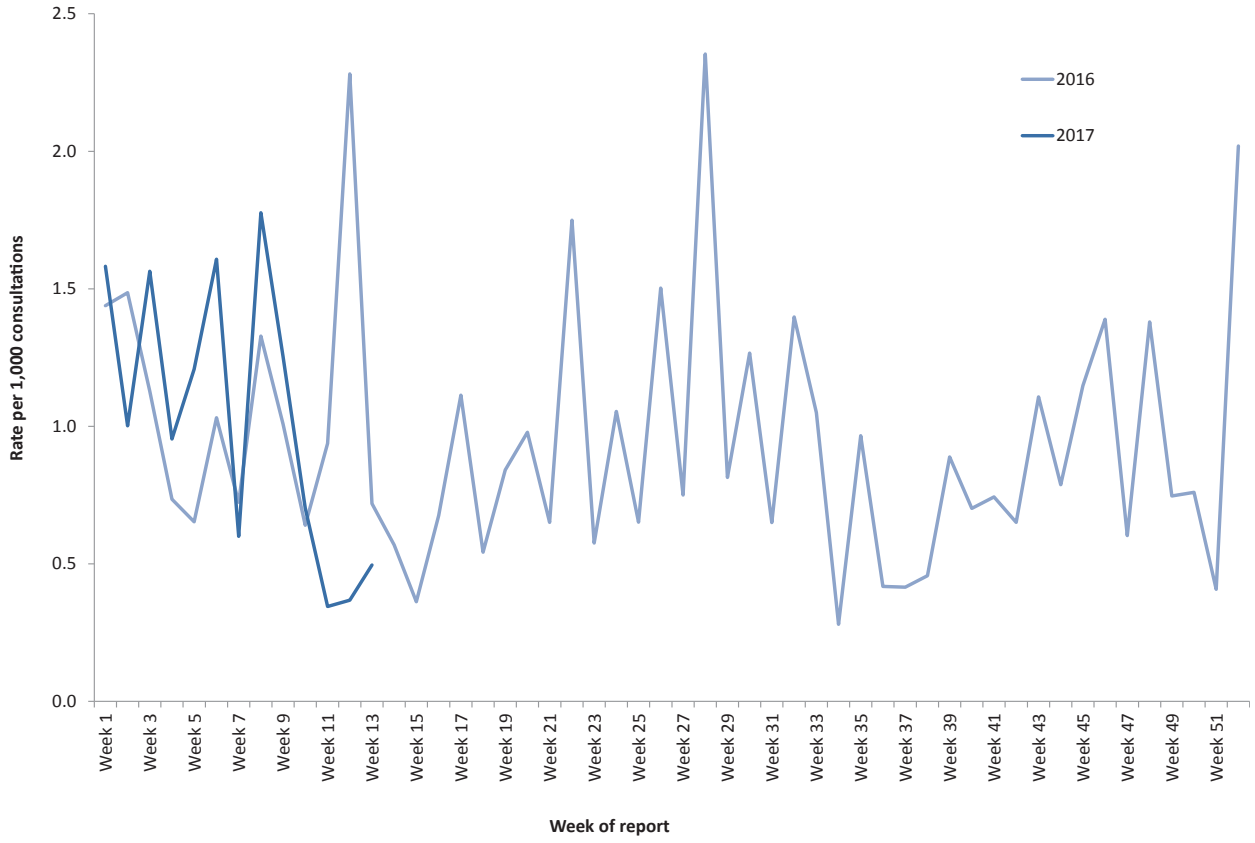


Figure 5: Consultation rates for shingles, ASPREN, 2016 and 1 January to 31 March 2017, by week of report



Quarterly report

OzFoodNet quarterly report, 1 April to 30 June 2015

The OzFoodNet Working Group

Introduction

The Australian Government Department of Health established the OzFoodNet network in 2000 to collaborate nationally to investigate foodborne disease. In each Australian state and territory, OzFoodNet epidemiologists investigate outbreaks of enteric infection. In addition, OzFoodNet conducts studies on the burden of illness and coordinates national investigations into outbreaks of foodborne disease. This quarterly report documents investigations of outbreaks of gastrointestinal illness and clusters of disease potentially related to food, which commenced in Australia between 1 April and 30 June 2015.

Data were received from OzFoodNet epidemiologists in all Australian states and territories. The data in this report are provisional and subject to change.

During the 2nd quarter of 2015 (1 April to 30 June), OzFoodNet sites reported 352 outbreaks of enteric illness, including those transmitted by contaminated food or water. Outbreaks of gastroenteritis are often not reported to health authorities, which results in current figures under-representing the true burden of enteric disease outbreaks within Australia. There were 5,214 people affected in these outbreaks and 192 hospitalisations. There were 11 deaths reported during these outbreaks. This represents a decrease in the number of people affected compared with the 5-year average from 2010 to 2014 for the 2nd quarter (8,191). The majority of reported outbreaks of gastrointestinal illness in Australia are due to person-to-person transmission. In this quarter, 72% (255/352) of outbreaks were transmitted via this route (see Table 1). This percentage was similar to the same quarter in 2014 (73%, 305/419) but the total number is lower than the 5-year average (2nd quarter, 2010-2014) of 360 outbreaks transmitted person-to-person. Of the person-to-person outbreaks in the 2nd quarter of 2015, 47% (119/255) occurred in child care facilities and 40% (102/255) occurred in aged care facilities.

Table 1: Outbreaks and clusters of gastrointestinal illness and number ill reported by OzFoodNet, Australia, 1 April to 30 June 2015, by mode of transmission.

Transmission mode	Number of outbreaks and clusters	Per cent of total (%)*	Number ill
Foodborne and suspected foodborne	33	10	363
Person-to-person	255	72	4,061
Suspected waterborne	4	1	14
Unknown	52	15	578
Unknown (*Salmonella* cluster)	7	2	182
Unknown (other pathogen cluster)	1	<1	16
Total	352	100	5,214

* May not add up to 100% due to rounding.

Foodborne and suspected foodborne disease outbreaks

There were 33 outbreaks during this quarter where consumption of contaminated food was suspected or confirmed as being the primary mode of transmission (Appendix 1). These outbreaks affected 363 people, of which 177 were laboratory confirmed cases, and resulted in 44 hospitalisations. There were no deaths reported during these outbreaks.

There were fewer foodborne outbreaks than were reported in the 1st quarter of 2015 (47) but similar to the 5-year average (2010-2014) for the 2nd quarter (34 outbreaks). The data within this report, provided by OzFoodNet sites, has associated limitations, including the potential variation in categorisation of features of outbreaks, depending on varied circumstances and investigator interpretation. Changes in the number of foodborne disease outbreaks should be interpreted with caution due to the small number each quarter.

Salmonella Typhimurium was identified as the aetiological agent in 52% (17/33) of foodborne or suspected foodborne outbreaks during this quarter (Appendix 1); a lower total and proportion than for the same quarter in 2014 (62%, 24/39). The aetiological agents for the remaining outbreaks included *Clostridium perfringens* (3 outbreaks), and one outbreak each due to: *Campylobacter jejuni*; ciguatoxin; *S.* Agona; *S.* Hvittingfoss; norovirus; *S.* subsp I ser 4, 5, 12: i:-, and *S.* Virchow. For 6 outbreaks the aetiological agent was unknown.

Fourteen outbreaks (42% of all foodborne or suspected foodborne outbreaks) reported in this quarter were associated with food prepared in restaurants (Table 2). This is similar to the 5 year average for the 2nd quarter (2014-2014) of 13 outbreaks.

To investigate these outbreaks, OzFoodNet sites conducted 4 cohort studies, 1 case control study and collected descriptive case series data for 19 investigations. For 9 outbreaks no individual patient data were collected. The evidence used to implicate food vehicles included analytical and microbiological evidence in 1 outbreak, analytical evidence in 3 outbreaks, microbiological evidence in 8 outbreaks, and descriptive evidence in 21 outbreaks.

The following jurisdictional summaries describe key outbreaks and public health actions that occurred during the quarter.

Australian Capital Territory

There were 2 outbreaks of foodborne or suspected foodborne illness reported in the Australian Capital Territory (ACT) in this quarter. The aetiological agents identified were *S.* Typhimurium phage type (PT) 135 and *C. perfringens*.

Table 2: Outbreaks of foodborne or suspected foodborne disease and number ill reported by OzFoodNet, Australia, 1 April to 30 June 2015, by food preparation setting.

Food preparation setting	Number of outbreaks	Per cent of foodborne outbreaks (%)*	Number ill	Number laboratory confirmed
Restaurant	14	42	136	69
Private residence	9	27	71	48
Commercial caterer	2	6	41	0
Take-away	2	6	10	4
Bakery	1	3	40	30
Institution – not otherwise specified	1	3	23	10
Other (home business)	1	3	17	2
Aged care facility	1	3	12	2
Camp	1	3	9	8
Child care facility	1	3	4	4
Total	33	100	363	177

* May not add up to 100% due to rounding.

Description of key outbreak

An outbreak was investigated in June after two people made a complaint of illness following a catered event attended by 2,600- 2,700 people. Active case finding was conducted on a subset of members of the public who had sent an RSVP for the event. Food histories were obtained for an additional 134 attendees and 11 (8%) of these reported diarrhoea. Interviews with staff members found 16/45 (36%) who reported consuming food at the event had diarrhoea. None of the cases visited a medical practitioner and no samples were collected. An analytical study conducted with catering staff found a statistically significant association with eating the butter chicken and becoming ill (adjusted risk ratio [aRR] 5.2; 95% confidence interval [95% CI] 1.1–24.9; $P<0.05$). The environmental investigation identified food handling and temperature control issues. Several food samples were taken and 170,000 colony forming units per gram (cfu/g) of *C. perfringens* was isolated from the butter chicken.

New South Wales

There were 10 outbreaks of foodborne or suspected foodborne illness reported in New South Wales (NSW) in this quarter. The aetiological agents identified were *S.* Typhimurium (for 2 outbreaks) and one outbreak each of *S.* Agona; *C. perfringens*; *Ca. jejuni;* and ciguatoxin. Four outbreaks were of unknown aetiology.

Description of key outbreak

An outbreak was investigated in June after routine surveillance identified an increase in *S.* Agona (9 cases) in Western Sydney. A total of 37 cases were notified in NSW between January and June 2015, with 13 of these cases notified in May and June 2015. The previous 5 year annual average in NSW for this serovar was 28 cases. Sixteen of the most recent cases were interviewed including all 9 located in Western Sydney. Six had consumed sushi from one of 2 sushi venues in the same shopping centre; 3 cases at sushi outlet A, 2 cases ate at sushi outlet B and 1 case ate at both outlet A and outlet B. No links were found between the other 10 cases. Both venues were inspected by the NSW Food Authority and were reported to have potential for cross contamination of ready to eat foods. It was reported no ingredients or staff were shared between shops and records were not available to confirm this. Samples were taken from both venues, with outlet A returning a positive *S.* Agona result from sushi rolls. Sushi outlet A was inspected another 2 times during the following 19 days. On all occasions the tuna mix for tuna sushi rolls was positive for *S.* Agona, even though the individual ingredients for this mix and the tools used to make this mix were all negative. The venue was prohibited from selling the tuna product until it showed evidence of *Salmonella* clearance. Whole genome sequencing showed *S.* Agona isolates from the 4 confirmed outbreak cases who reported eating at sushi outlet A, shared identical sequencing with 2 cases who reported eating just at sushi outlet B, and with isolates from 5 other cases from the same time period who either did not report eating at the sushi restaurant or were not interviewed. All of the *S.* Agona isolates from food samples at sushi outlet A were identical to the case isolates and very similar to 2 of the isolates from retail samples of chicken meat earlier in the year. This analysis suggests the source of the *S.* Agona in the cluster may have been cross contamination from raw chicken meat, with a common source of chicken for the 2 sushi venues likely at the time of the outbreak. This investigation was the first time NSW used whole genome sequencing for a *Salmonella* outbreak.

Northern Territory

There was 1 outbreak of foodborne or suspected foodborne illness reported in the Northern Territory (NT) in this quarter. The aetiological agent identified was *S.* Typhimurium PT 9.

Description of key outbreak

An outbreak was investigated in June after 23 people reported becoming ill after attending the same restaurant. Eight cases were laboratory confirmed with *S.* Typhimurium PT 9. There were 4 hospitalisations as a result of the

outbreak. Analysis of a cohort study involving 76/80 patrons and 3 staff found an association between consuming duck prosciutto and illness (relative risk [RR] undefined; OR 18.6; $P<0.05$). Duck prosciutto was eaten by all cases and had a food-specific attack rate (AR) of 27%. An environmental health inspection of the restaurant identified that the duck prosciutto was likely to have been cured for an inadequate time period and in an area where cross contamination could occur. Duck prosciutto was immediately removed from the menu. Samples of raw duck meat and duck prosciutto were collected. The raw duck meat tested negative for *Salmonella* spp. and coliforms. The duck prosciutto also tested negative for *Salmonella* spp. but contained high levels of coliforms ($2x10^7$ cfu/g) with the increase in coliforns suggesting contamination of the prosciutto during the curing process.

Queensland

There were 5 outbreaks of foodborne or suspected foodborne illness reported in Queensland (Qld) in this quarter. The aetiological agents identified were *S.* Typhimurium (for 2 outbreaks), and *S.* Hvittingfoss, *S.* Virchow PT 8 and norovirus genogroup II (for 1 outbreak each).

Description of key outbreak

An outbreak was investigated in April after 9 cases of gastrointestinal illness were identified among 2 school groups that attended a camp facility. *S.* Virchow PT 8 was detected in 8/9 cases. No common food vehicle was identified; however, water samples collected from a rainwater tank, which supplied the kitchen facility, tested positive for *S.* Virchow PT 8. Whole genome sequencing indicated a close genetic relatedness between the isolates from the human specimens and the water samples. Investigations identified potential issues with regard to vermin, birds and leaf litter from trees surrounding the kitchen facility. The ultraviolet disinfection system connected to the rainwater tanks required re-calibration and sediment filters were in need of maintenance. All rainwater tanks were subsequently chlorinated.

South Australia

There were 5 outbreaks of foodborne or suspected foodborne illness reported in South Australia (SA) in this quarter. The aetiological agents identified were *S.* Typhimurium (for 4 outbreaks) and *S.* subsp 1 ser 4,5,12:i:- (for 1 outbreak).

Description of key outbreak

An outbreak was investigated in June after initial interviews identified 2 cases of *S.* Typhimurium PT 9, multi-locus variable number tandem repeat analysis (MLVA) profile 03-14-08-11-550 who had eaten at the same bakery in metropolitan Adelaide before becoming unwell. A total of 30 cases, 8 of whom were hospitalised, reported consuming Vietnamese rolls purchased from two bakeries owned by the same family. Ten additional people reported having gastroenteritis following eating at one of the two bakeries, but were not tested. The rolls were made with raw egg butter and an environmental investigation identified multiple poor practices in relation to handling the raw egg butter. An improvement notice was issued.

Tasmania

There were no outbreaks of foodborne or suspected foodborne illness reported in Tasmania in this quarter.

Victoria

There were 8 outbreaks of foodborne or suspected foodborne illness reported in Victoria (Vic.) in this quarter. The aetiological agents identified were *S.* Typhimurium (for 5 outbreaks) and *C. perfringens* for 1 outbreak. Two outbreaks were of unknown aetiology.

Description of key outbreak

An outbreak associated with the consumption of food from a restaurant was investigated in June after a complaint was made to a local council. Seventy-five people attended a birthday dinner that comprised an Asian buffet style meal with

a range of desserts made at different premises. Sixteen of 28 attendees interviewed reported being ill with diarrhoea; the majority of whom also experienced abdominal pain and fever. One case was considered to have been a secondary case due to a delayed onset of symptoms. Twelve cases presented to a doctor and 2 were hospitalised. *S.* Typhimurium PT 135 MLVA 03-11-11/12-14-523 was isolated from 8 of 9 faecal specimens. One restaurant staff member had an onset of diarrhoea 48 hours after this group dined at the restaurant and submitted a faecal specimen which was polymerase chain reaction (PCR) positive for *Salmonella* but culture negative. A case-control study showed that cases were more likely to have eaten desserts when compared to those who were not ill (odds ratio [OR] 12; 95%CI 1.0-590.2; $P<0.05$). The desserts included tiramisu, cheesecake, custard cream cake and fruit. No leftover food from this function was available for testing, however samples of fish and raw eggs collected from the premises during the investigation tested negative for *Salmonella*.

Western Australia

There were 2 outbreaks of foodborne or suspected foodborne illness reported in Western Australia (WA) in this quarter. The aetiological agent identified was *S.* Typhimurium (for both outbreaks).

Description of key outbreak

An outbreak was investigated in April after cases of *S.* Typhimurium pulsed-field gel electrophoresis (PFGE) type 0001 reported independently eating at the same café. In total there were 9 confirmed cases and 1 suspected case. Most cases (8/10) had eaten breakfast meals containing eggs, while the remaining 2 cases had consumed fruit smoothies. Cases reported that the eggs were undercooked. One sample from the implicated egg brand tested positive for *S.* Typhimurium PFGE 0001. Environmental samples (eggs and faecal material) from the implicated egg farm were negative for *Salmonella*.

Multi-jurisdictional investigations

In the first half of 2015, OzFoodNet investigated a multi-jurisdictional outbreak of hepatitis A associated with the consumption of a particular imported frozen mixed berry product. Consumer level recalls of the implicated product and related products were conducted in February 2015. Case finding was conducted in every jurisdiction which included all cases of hepatitis A notified from 1 October 2014 to take into account the long incubation period of the virus and the time period that the implicated frozen berries were in the marketplace. A total of 35 laboratory-confirmed cases of hepatitis A with genotype IA and an identical genetic sequence were associated with this outbreak; 15 in Qld, 13 in NSW, 4 in Vic., and 1 each in WA, the ACT, and SA. Of the 35 confirmed cases, 28 recalled consumption of the implicated brand of imported frozen mixed berries during their acquisition period. Three cases were secondary infections (of confirmed outbreak cases), 2 cases had consumed frozen berries during their acquisition period but couldn't recall the brands, and 2 cases could not recall eating any frozen berries and had no other risk factors.

Hepatitis A RNA was detected in 1/3 opened packets of the implicated berry product that were obtained from cases' homes and from 1/15 sealed packets obtained from retail premises that were removed from sale during the recall. The RNA in the sample from the open packet was amplified and confirmed to be genotype IA with the outbreak genetic sequence, however, the RNA in the sealed packet was present at very low levels and unable to be amplified to enable genotyping and sequencing to be conducted.

This outbreak involved a multi-jurisdictional response involving state and territory health departments and food safety agencies, OzFoodNet, public health reference laboratories, the Australian Department of Agriculture and Food Standards Australia New Zealand. The Chief Medical Officer of Australia also activated the National Incident Room at the Australian Department of Health in the initial stages of the investigation to assist in the coor-

dination of communication between the various national, state and territory agencies involved in the response.

A prospective case-control study was conducted with 23 cases of confirmed hepatitis A (genotype IA with the outbreak genetic sequence) and 47 *Salmonella* cases which were used as controls and enrolled from the respective jurisdictional notifiable disease databases where the cases were notified. Univariate analyses revealed statistically significant results for consuming the implicated frozen mixed berry product (odds ratio [OR] 440; 95% confidence interval [CI] 32-18,531; $P<0.05$), consuming any frozen mixed berries (OR 88; 95% CI 10.5-3727; $P<0.05$) and consuming any frozen berries (OR 49; 95% CI 6.2-2073; $P<0.05$).

Cluster investigations

During this quarter, OzFoodNet sites conducted investigations into 15 clusters of infection for which no common food vehicle or source of infection could be identified. Aetiological agents for these clusters included *S.* Typhimurium (3 clusters), *S.* Virchow (3 clusters), and one cluster each of: *S.* Mississipi (ampicillin resistant); *S.* Victoria; *S.* Zanzibar; *S.* Mbandaka; *S.* Newport; *S.* Chester; *Yersinia enterocolitica*, *Campylobacter spp.*; and *Cryptosporidium spp.*

Comments

This quarter marks OzFoodNet's first use of whole genome sequencing (WGS) during foodborne disease outbreak investigations (1 NSW and 1 Qld). WGS provides unparalleled resolution of communicable disease pathogens. Clusters can be more accurately defined through this process offering a more targeted response to outbreaks of foodborne disease. The genomics of communicable disease pathogens can be analysed, interpreted and stored and then shared across national and international borders. This allows for rapid identification of multinational outbreaks, a process which without whole genome sequencing, can take months to years. WGS has already been applied to match an Australian human *Listeria monocytogenes* isolate to an outbreak in stone fruit in the United States of America. It was subsequently identified that some implicated stone fruit had been imported to Australia and the case had reported consuming some, leading to a recall in Australia.

The hepatitis A multi-jurisdictional outbreak investigation (MJOI) was a complex investigation involving unprecedented levels of inter-agency communication and media interest. The investigation ultimately led to a review of national communication protocols for foodborne incidents, and also to proposed legislative changes to the imported food scheme.

Acknowledgements

OzFoodNet thanks the investigators in the public health units and state and territory departments of health, as well as public health laboratories, local government environmental health officers and food safety agencies who provided the data used in this report. We would particularly like to thank the reference laboratories for conducting sub-typing of *Salmonella* species and other enteric pathogens and for their continuing work and advice during the quarter.

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Appendix 1: Outbreaks of foodborne or suspected foodborne disease reported by OzFoodNet sites*, 1 April to 30 June 2015 (n=33)

State or Territory	Month †	Setting Prepared	Agent responsible	Number affected ^	Number lab confirmed	Number hospitalised ^	Evidence	Responsible vehicles
ACT	Jun	Private residence	*S.* Typhimurium PT 135, MLVA 03-17-08-12-525	2	2	2	D	Smoothie containing raw egg
ACT	Jun	Commercial caterer	*Clostridium perfringens*	29	0	0	AM	Butter chicken
NSW	Apr	Private residence	Ciguatera fish poisoning	4	0	1	D	Spanish mackerel
NSW	Apr	Restaurant	*Clostridium perfringens*	4	2	0	D	Unknown
NSW	Apr	Restaurant	*S.* Typhimurium MLVA 03-12-12-09-523	11	8	0	D	Undercooked egg dishes
NSW	May	Restaurant	Unknown	7	0	0	D	Unknown
NSW	May	Restaurant	*Campylobacter jejuni*	2	2	1	D	Chicken liver pâté
NSW	May	Commercial caterer	Unknown	12	0	1	D	Unknown
NSW	Jun	Restaurant	Unknown	9	0	1	D	Unknown
NSW	Jun	Child care centre	*S.* Typhimurium MLVA 03-14-09-13-523	4	4	1	D	Unknown
NSW	Jun	Take-away	*S.* Agona	4	4	0	M	Tuna mix for sushi
NSW	Jun	Take-away	Unknown	6	0	0	D	Kebabs
NT	Jun	Restaurant	*S.* Typhimurium PT 9	23	8	4	A	Duck prosciutto
QLD	Apr	Other	Norovirus genogroup II	17	2	0	D	Birthday cake
QLD	Apr	Camp	*S.* Virchow PT 8	9	8	0	M	Water
QLD	May	Private residence	*S.* Hvittingfoss	23	21	Unknown	M	Unknown (mixed food)
QLD	May	Restaurant	*S.* Typhimurium MLVA 05-21-08-14-456	14	14	6	D	Unknown
QLD	May	Restaurant	*S.* Typhimurium MLVA 03-17-09-11-523	8	6	2	D	Unknown
SA	Apr	Restaurant	*S.* Typhimurium PT 9, MLVA 03-24-11-10-523	9	8	3	M	Eggs

State or Territory	Month †	Setting Prepared	Agent responsible	Number affected ^	Number lab confirmed	Number hospitalised ^	Evidence	Responsible vehicles
SA	May	Private residence	*S.* Typhimurium PT 170/108, MLVA 03-09-07-12-523	9	5	2	M	Veal and chicken schnitzel in egg batter
SA	Apr	Private residence	*S.* Typhimurium PT 135a, MLVA 03-11-12-14-523	4	4	2	D	Unknown
SA	Apr	Private residence	*Salmonella* subsp 1 ser 4, 5, 12: i:-, MLVA 04-15-11-00-490	6	3	1	D	Unknown
SA	Jun	Bakery	*S.* Typhimurium PT 9, MLVA 03-14-08-11-550	40	30	8	D	Vietnamese rolls with raw egg butter
VIC	Apr	Private residence	*S.* Typhimurium PT 44, MLVA 03-10-09-08-523	6	2	2	D	Temperature abuse of pasta made with raw egg
VIC	Apr	Private residence	*S.* Typhimurium PT 135a, MLVA 03-11-09-11-523	6	4	1	M	Chocolate mousse
VIC	Apr	Private residence	*S.* Typhimurium PT 9, MLVA 03-23-23-10-523	11	7	0	A	Pasta carbonara containing raw egg
VIC	Apr	Restaurant	Unknown (one confirmed case of *S.* Virchow PT8)	4	1	0	D	Unknown
VIC	May	Institution not otherwise specified	*S.* Typhimurium PT 135, MLVA 03-14-10-08-523	23	10	1	M	Chicken
VIC	Jun	Restaurant	*S.* Typhimurium PT 135, MLVA 03-11-11/12-14-523	16	8	2	A	Desserts including tiramisu, cheesecake & custard cream cake
VIC	Jun	Aged care facility	*Clostridium perfringens*	12	2	0	D	Temperature abuse of food served from a bain-marie
VIC	Jun	Restaurant	Unknown	14	0	0	D	Unknown
WA	Apr	Restaurant	*S.* Typhimurium PT 9, PFGE 0001	5	3	2	D	Semifreddo containing raw egg
WA	Apr	Restaurant	*S.* Typhimurium PT 9, PFGE 0001	10	9	1	M	Egg dishes
Total				363	177	44		

* *No foodborne outbreaks were reported in Tasmania during the quarter*
† *Month of outbreak is the month of onset of the first case or month of notification of the first case or month the investigation of the outbreak commenced.*
^ *The number of people affected and hospitalised relate to the findings of the outbreak investigation at the time of writing and not necessarily in the month specified or in this quarter. The number of people affected does not necessarily equal the number of laboratory-confirmed cases.*
A *Analytical epidemiological association between illness and one or more foods*
D *Descriptive evidence implicating the suspected vehicle or suggesting foodborne transmission*
M *Microbiological confirmation of aetiological agent in the suspected vehicle and cases*
MLVA *Multi-locus variable number tandem repeat analysis*
PFGE *Pulsed-field gel electrophoresis*
PT *Phage type*

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Quarterly report

OzFoodNet quarterly report, 1 July to 30 September 2015

The OzFoodNet Working Group

Introduction

The Australian Government Department of Health established the OzFoodNet network in 2000 to collaborate nationally to investigate foodborne disease. In each Australian state and territory, OzFoodNet epidemiologists investigate outbreaks of enteric infection. In addition, OzFoodNet conducts studies on the burden of illness and coordinates national investigations into outbreaks of foodborne disease. This quarterly report documents investigations of outbreaks of gastrointestinal illness and clusters of disease potentially related to food, which commenced in Australia between 1 July and 30 September 2015.

Data were received from OzFoodNet epidemiologists in all Australian states and territories. The data in this report are provisional and subject to change.

During the 3rd quarter of 2015 (1 July to 30 September), OzFoodNet sites reported 474 outbreaks of enteric illness, including those transmitted by contaminated food or water. Outbreaks of gastroenteritis are often not reported to health authorities, which results in current figures under-representing the true burden of enteric disease outbreaks within Australia. There were 8,561 people affected in these outbreaks and 246 hospitalisations. There were 25 deaths reported during these outbreaks. This represents a decrease in the number of people affected compared with the 5-year average from 2010 to 2014 for the 3rd quarter (n=10,047). The majority of reported outbreaks of gastrointestinal illness in Australia are due to person-to-person transmission. In this quarter, 85% (402/474) of outbreaks were transmitted via this route (see Table 1). This percentage was higher compared to the same quarter in 2014 (71%, 242/342) but the total number is lower than the 5-year average (3rd quarter, 2010-2014) of 447 outbreaks transmitted person-to-person. Of the person-to-person outbreaks in the 3rd quarter of 2015, 40% (159/402) occurred in child care facilities and 45% (181/402) occurred in aged care facilities.

Table 1: Outbreaks and clusters of gastrointestinal illness and number ill reported by OzFoodNet, Australia, 1 July to 30 September 2015, by mode of transmission

Transmission mode	Number of outbreaks and clusters	Per cent of total outbreaks and clusters (%)*	Number ill
Foodborne and suspected foodborne	29	6%	659
Person-to-person	402	85%	7,520
Salmonella cluster	4	1%	66
Suspected waterborne	2	<1%	32
Unknown	37	8%	284
Total	474	100	8,561

* May not add up to 100% due to rounding.

Foodborne and suspected foodborne disease outbreaks

There were 29 outbreaks during this quarter where consumption of contaminated food was suspected or confirmed as being the primary mode of transmission (Appendix 1). These outbreaks affected 659 people, of which 195 were laboratory confirmed cases, and resulted in 78 hospitalisations. There was one death reported during these outbreaks. This was a decrease on the number of foodborne outbreaks that were reported in the 2nd quarter of 2015 (33 outbreaks), but the number in this quarter was similar to the 5-year average for the 3rd quarter between 2010 and 2014 (30 outbreaks). The data within this report, provided by OzFoodNet sites, have associated limitations, including the potential variation in categorisation of features of outbreaks, depending on varied circumstances and investigator interpretation. Changes in the number of foodborne disease outbreaks should be interpreted with caution due to the small number each quarter.

Salmonella Typhimurium was identified as the aetiological agent in 34% (11/29) of foodborne or suspected foodborne outbreaks during this quarter (Appendix 1); a lower proportion than for the same quarter in 2014 (40%, 10/25). The aetiological agents for the remaining outbreaks included norovirus (for 4 outbreaks), *Campylobacter* (for 2 outbreaks), ciguatoxin, and *S.* subsp 1:4,5,12:i- (for 1 outbreak each). For 11 outbreaks the aetiological agent was unknown.

Twelve outbreaks (41% of all foodborne or suspected foodborne outbreaks) reported in this quarter were associated with food prepared in restaurants (Table 2). This is lower than the average number of foodborne or suspected foodborne outbreaks in restaurants in the 3rd quarter from 2010 to 2014 (13.4 outbreaks).

To investigate these outbreaks, sites conducted 3 cohort studies, 2 case control studies, 1 case series and cohort study, and collected descriptive case series data for 15 investigations. For 8 outbreaks no individual patient data were collected. The evidence used to implicate food vehicles included analytical evidence in 3 outbreaks, microbiological evidence in 5 outbreaks, both analytical and microbiological evidence in 1 outbreak, and descriptive evidence in 20 outbreaks.

The following jurisdictional summaries describe key outbreaks and public health actions that occurred during the quarter.

Table 2: Outbreaks of foodborne or suspected foodborne disease and number ill reported by OzFoodNet, Australia, 1 July to 30 September 2015, by food preparation setting

Food preparation setting	Number of outbreaks	Per cent of foodborne outbreaks (%)*	Number ill	Number laboratory confirmed
Restaurant	12	41%	324	113
Aged care facility	3	10%	155	14
Take-away	3	10%	26	14
Private residence	3	10%	12	7
Commercial caterer	2	7%	54	3
Bakery	1	3%	18	1
Overseas (returning military personnel)	1	3%	19	19
Child care facility	1	3%	2	2
Fair, festival, other temporary/mobile service	1	3%	4	3
Hospital	1	3%	37	16
School	1	3%	8	3
Total	29	100%	659	195

* May not add up to 100% due to rounding.

Australian Capital Territory

There was 1 outbreak of foodborne or suspected foodborne illness reported in the Australian Capital Territory (ACT) in this quarter. The aetiological agent identified was *S.* Typhimurium phage type (PT) 9.

Description of outbreak

The outbreak occurred in a private residence. Three people were affected, with onsets within two hours of each other. One of the three cases was admitted to hospital. Two cases were laboratory confirmed *S.* Typhimurium PT 9. A possible food vehicle was identified through routine interviews. A smoothie containing raw eggs was consumed by all three cases. No other family members were exposed or ill. The suspected source of infection was the raw eggs used in the smoothies.

New South Wales

There were 11 outbreaks of foodborne or suspected foodborne illness reported in New South Wales (NSW) in this quarter. The aetiological agents were identified as *S.* Typhimurium (for 2 outbreaks), norovirus and ciguatera fish poisoning (for 1 outbreak each). In 7 outbreaks, the aetiological agents were unknown. In three of the outbreaks, a suspected food vehicle could be implicated.

Description of key outbreak

Three separate complaints of food poisoning from three separate groups dining at the same Chinese restaurant were received by the NSW Food Authority. An investigation was initiated. A retrospective cohort study was conducted. Forty-four of 59 diners were interviewed, 40 reporting illness following dinner at the restaurant. Eleven stool samples were positive for *S.* Typhimurium multi-locus variable number tandem repeat analysis (MLVA) profile 03-12-11-14-523. Univariate analysis identified a statistically significant association between illness and those that consumed fried ice cream (odds ratio [OR] 6.89, 95% confidence interval [CI] 1.2-39.0, $P \leq 0.05$)

NSW Food Authority, in conjunction with the local council, conducted an inspection of the premises. General hygiene and food handling practices were found to be good; however fried ice cream was made using raw egg. Food and environmental samples were taken during the inspection. Samples of uncooked fried ice cream balls and fried ice cream crumbs both tested positive for *S.* Typhimurium MLVA 03-12-11-14-523. The NSW Food Authority issued the restaurant with a prohibition order on serving fried ice cream (made using a raw egg component).

A traceback on the egg supplier was conducted. The egg supplier was associated with four other outbreaks during 2014 and 2015. The egg farms associated with the supplier were inspected by the NSW Food Authority. Various *Salmonella* serovars (including *S.* Typhimurium MLVA 03-09-08-13-523, *S.* Agona, *S.* Bareilly, and *S.* Orion) were detected on the farm. No deficits in hygiene or processing were observed. The farm reported some bird illness in the last 18 months as well as new flock populations. It is possible stress events such as these could exacerbate the presence of *Salmonella* on otherwise hygienic and well run egg farms. [1]

Northern Territory

There were 3 outbreaks of foodborne or suspected foodborne illness investigated in the Northern Territory (NT) this quarter. The aetiological agents identified included *S.* Typhimurium and *Campylobacter* for 1 outbreak each, and for 1 outbreak the aetiological agent was unknown.

Description of key outbreak

An outbreak was investigated in Darwin after 4 reports of illness were made following a cultural festival. Of these reports, 3 were laboratory confirmed cases of *S.* Typhimurium (including 2 cases of *S.* Typhimurium PT 168a). Environmental health officers were notified but the festival was a one-night only event and no food remained for testing. A number of patrons who were interviewed as part of the outbreak investigation reported poor temperature control of cooked foods on the night in question.

Queensland

There were 2 outbreaks of foodborne or suspected foodborne illness reported in Queensland in this quarter. *S.* Typhimurium was identified as the aetiological agent for both outbreaks.

Description of key outbreak

An outbreak was investigated after gastrointestinal illness was reported in 2 separate groups that had consumed meals at the same Brisbane restaurant. A total of 76 reported cases had attended the restaurant during a one week period. Forty-two of the 76 cases were laboratory confirmed: 41 cases with *S.* Typhimurium MLVA 03-17-09-11-523 (1 had a co-infection with *S.* Hvittingfoss); and 1 case with *S.* Typhimurium MLVA 03-16-09-11-523. A case series investigation of 57 ill attendees indicated the majority of cases had consumed foods containing various mayonnaise-based sauces including aioli and tartare sauce. The environmental health investigation determined that a base mayonnaise sauce was used to create various different flavoured sauces. The base mayonnaise sauce was prepared each week in a 25 litre bucket using whole eggs, into which oil, vinegar and mustard were also added and the mixture emulsified using a stick blender. Numerous environmental samples were collected. *S.* Typhimurium (MLVA 03-17-09-11-523) was isolated from samples of chilli, mustard, aioli and base mayonnaise. Additionally, *Escherichia coli* and *Staphylococcus aureus* were detected on a sauce bottle and kitchen tap handle. Eggs that were collected during the investigation for microbial analysis were negative for *Salmonella*. Following the investigation, the restaurant management removed all raw egg-based sauces from the menu (replaced with commercially made mayonnaises) and replaced all chopping boards, sauce bottles and the stick blender.

South Australia

There were 2 outbreaks of foodborne or suspected foodborne illness reported in South Australia (SA) in this quarter. *S.* Typhimurium PT 9 was identified as the aetiological agent for both outbreaks.

Description of key outbreak

An outbreak was investigated after a report was received from a hospital about two patients who had tested positive for *Salmonella*. A total of 37 people who had contact with the hospital were reported as unwell, with 16 testing positive for *S.* Typhimurium PT 9 (MLVA 03-24-12-10-523) and 2 people tested positive for *Aeromonas*. The hospital kitchen was inspected and a range of food and environmental samples were collected. Imported frozen fish samples were positive for *S.* Matopeni and *S.* Weltevreden. Samples of the uncooked imported fish that had been coated with an egg-based crumb were positive for *S.* Typhimurium PT 9 (MLVA 03-24-12-10-523). Internal components from a stab mixer were also positive for *S.* Typhimurium PT 9 (MLVA 03-24-12-10-523). Both items were suspected to have been contaminated by eggs.

Tasmania

There was one outbreak of foodborne or suspected foodborne illness reported in Tasmania in this quarter. The aetiological agent was identified as norovirus.

Description of outbreak

An outbreak was investigated in an aged care facility after 84 residents and 40 staff reported symptoms of gastroenteritis. The overall attack rate was estimated to be 37%. Six of 8 specimens collected had norovirus detected. Five people presented to a medical practitioner, and 1 person was hospitalised. The epidemiology suggested a point source outbreak with a large number of cases becoming ill on the same day and at around the same time. Foodborne transmission was suspected, but the vehicle for the outbreak could not be definitively determined.

Victoria

There were 8 outbreaks of foodborne or suspected foodborne illness reported in Victoria in this quarter. The aetiological agents identified were norovirus (for 2 outbreaks), *S.* subsp.

1:4,5,12:i:- *S.* Typhimurium and *Campylobacter* (for 1 outbreak each). In 3 outbreaks the aetiological agent was unknown.

Description of key outbreak

An outbreak associated with a catered work function was investigated. Symptoms of diarrhoea were reported in 14 of 200 attendees. Foods served included a variety of roasted meats, gravy, vegetables, dips, quiche and falafels. The workplace provided a list of attendees with details of who had been ill. A random sample of 25 attendees taken from both ill and well groups was interviewed and odds ratios were calculated for each of the food exposures. Illness was associated with consumption of 3 different types of roasted meats. Univariate analysis identified a statistically significant association between illness and those that consumed roast pork (OR 10.8, 95% CI 0.84 - 550.59, P=0.026) roast beef (OR undefined, 95% CI 1.2 - undefined, p=0.037), ham (OR undefined; Lower 95% CI 3.5 – undefined, P=0.002). The number of attendees interviewed was too small for stratified analysis to be conducted. Illness duration and symptom profile was consistent with *Clostridium perfringens* enterotoxin. Three faecal specimens were collected, albeit between 3 and 4 days after symptoms resolved. All three specimens tested negative for viral and bacterial pathogens, including *C. perfringens* enterotoxin.

Western Australia

There was 1 outbreak of foodborne or suspected foodborne illness reported in Western Australia (WA) in this quarter. The aetiological agent was identified as *S.* Typhimurium.

Description of outbreak

An outbreak was investigated in an after school care setting following notification of two cases of *S.* Typhimurium pulsed-field gel electrophoresis (PFGE) type 0001, MLVA 03-10-15-11-496. Both cases consumed raw cake mixture and became ill within 48 hours. No other food was shared between the cases. Eggs used in the cake mixture were from home hens. The manager of the facility was advised not to use home hen eggs at the facility and not to offer students raw cake mixture to eat.

Multi-jurisdictional investigations

There were no multi-jurisdictional outbreak investigations in this quarter.

Cluster investigations

During this quarter, OzFoodNet sites conducted investigations into 7 clusters of infection for which no common food vehicle or source of infection could be identified. Aetiological agents that were able to be identified during the investigations included *S.* Typhimurium (for 4 clusters), and *S.* Bovismorbificans, *Salmonella* subsp I, and *S.* Saintpaul (for 1 cluster each).

Comments

OzFoodNet has performed enhanced surveillance on all notified cases of invasive listeriosis nationally since 2010 through its National Enhanced Listeriosis Surveillance System (NELSS). All *Listeria monocytogenes* isolates from these cases were subtyped using PFGE performed at the Microbiological Diagnostic Unit Public Health Laboratory (MDU) in Melbourne, Victoria. PFGE is a slow, technically demanding and resource intensive process,[2] so several less discriminatory but rapid subtyping methods (binary typing, molecular serotyping and MLVA) were performed at MDU and other enteric reference laboratories to assist with rapid cluster detection. At the start of this quarter, MDU and OzFoodNet began routinely performing whole genome sequencing in parallel with PFGE for subtyping and reporting of invasive listeriosis cases in Australia.

The Implementation Subcommittee for Food Regulation (ISFR) *Principles for the Inspection of Food Businesses* were endorsed by ISFR committee members this quarter. The document provides principles for the guidance of the inspection of food businesses. "Inspection of food premises is

an important activity to assess compliance with the *Australia New Zealand Food Standards Code* (the Code) and food act provisions.[3]

Acknowledgements

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Appendix 1: Outbreaks of foodborne or suspected foodborne disease reported by OzFoodNet sites, 1 July to 30 September 2015 (n=29)

State or Territory	Month*	Setting Prepared	Agent responsible	No. affected †	No. lab confirmed	No. hospitalised†	Evidence	Responsible vehicles
ACT	Jul	Private residence	*S.* Typhimurium PT 9	3	2	1	D	Smoothie containing raw egg
NSW	Jul	Restaurant	Unknown	10	0	0	D	Unknown
NSW	Jul	Take-away	Unknown	9	0	0	D	Unknown
NSW	Aug	Restaurant	Unknown	5	0	0	D	Unknown
NSW	Aug	Restaurant	Unknown	3	0	0	D	Unknown
NSW	Aug	Restaurant	Unknown	3	0	0	D	Unknown
NSW	Sep	Restaurant	Unknown	29	0	0	D	Unknown
NSW	Sep	Take-away	*S.* Typhimurium MLVA 03-16-09-11-523	12	12	9	M	Vietnamese pork rolls
NSW	Sep	Private residence	Ciguatera fish poisoning	4	0	0	M	Red throat emperor and purple rock cod
NSW	Sep	Restaurant	Unknown	5	0	0	D	Unknown
NSW	Sep	Restaurant	*S.* Typhimurium MLVA 03-12-11-14-523	40	11	0	M	Fried ice cream
NSW	Aug	Bakery	Norovirus	18	1	1	D	Unknown
NT	Aug	Fair, festival, other temporary/ mobile service	*S.* Typhimurium PT 168a	4	3	0	D	Unknown
NT	Aug	Restaurant	Unknown	3	0	0	D	Aioli
NT	Aug	Institution (overseas)	*Campylobacter*	19	19	19	D	Unknown
Qld	Aug	Aged Care Facility	*S.* Typhimurium MLVA 03-17-09-11-523	22	8	2	D	Unknown
Qld	Sep	Restaurant	*S.* Typhimurium MLVA 03-17-09-11-523 (41) & MLVA 03-16-09-11-523 (1)	76	42	16	M	Aioli and mayonnaise-based sauces
SA	Jul	Take-away	*S.* Typhimurium PT 9, MLVA 03-24-11-10-523	5	2	0	D	Egg based crêpes

State or Territory	Month*	Setting Prepared	Agent responsible	No. affected †	No. lab confirmed	No. hospitalised†	Evidence	Responsible vehicles
SA	Jul	Hospital	*S.* Typhimurium PT 9, MLVA 03-24-12-10-523	37	16	6	M	Egg based crumb and contaminated stab mixer
Tas.	Aug	Aged Care Facility	Norovirus	124	6	1	D	Unknown
Vic.	Jul	Restaurant	*S.* Typhimurium PT 170/108, MLVA 03-09-09-15-525	133	55	22	AM	Mayonnaise containing raw egg
Vic.	Jul	Aged Care Facility	Unknown	9	0	0	D	Unknown
Vic.	Jul	Commercial caterer	Unknown	14	0	0	A	Roast meats
Vic.	Sep	Restaurant	Unknown	8	3	0	D	Unknown
Vic.	Aug	School	*Campylobacter*	8	3	0	D	Unknown
Vic.	Sep	Commercial caterer	Norovirus	40	3	0	A	Any food from a platter
Vic.	Sep	Restaurant	Norovirus	9	2	0	A	Salad – young leaves with house dressing
Vic.	Sep	Private residence	*Salmonella* subsp. 1:4,5,12:i:- PT 9, MLVA 03-10-14-11-496	5	5	1	D	Raw chocolate chip cookie dough containing eggs
WA	Aug	Child care centre	*S.* Typhimurium PFGE 0001, MLVA 03-10-15-11-496	2	2	0	D	Cake mix containing raw eggs
Total				658	195	78		

** Month of outbreak is the month of onset of the first case or month of notification of the first case or month the investigation of the outbreak commenced.*
† The number of people affected and hospitalised relate to the findings of the outbreak investigation at the time of writing and not necessarily in the month specified or in this quarter. The number of people affected does not necessarily equal the number of laboratory-confirmed cases.
A Analytical epidemiological association between illness and one or more foods
D Descriptive evidence implicating the suspected vehicle or suggesting foodborne transmission
M Microbiological confirmation of aetiological agent in the suspected vehicle and cases
MLVA Multi-locus variable number tandem repeat analysis
PFGE Pulsed-field gel electrophoresis
PT Phage type

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Quarterly report

National Notifiable Diseases Surveillance System, 1 April to 30 June 2017

Office of Health Protection, Department of Health

A summary of diseases currently being reported by each jurisdiction is provided in Table 1. There were 75,935 notifications to the National Notifiable Diseases Surveillance System (NNDSS) between 1 April to 30 June 2017 (Table 2). The notification rate of diseases per 100,000 population for each state or territory is presented in Table 3.

Table 1: Reporting of notifiable diseases by jurisdiction

Disease	Data received from:
Bloodborne diseases	
Hepatitis (NEC)	All jurisdictions
Hepatitis B (newly acquired)	All jurisdictions
Hepatitis B (unspecified)	All jurisdictions
Hepatitis C (newly acquired)	All jurisdictions
Hepatitis C (unspecified)	All jurisdictions
Hepatitis D	All jurisdictions
Gastrointestinal diseases	
Botulism	All jurisdictions
Campylobacteriosis	All jurisdictions except New South Wales
Cryptosporidiosis	All jurisdictions
Haemolytic uraemic syndrome	All jurisdictions
Hepatitis A	All jurisdictions
Hepatitis E	All jurisdictions
Listeriosis	All jurisdictions
Paratyphoid	All jurisdictions
Shiga toxin/verotoxin-producing *Escherichia coli*	All jurisdictions
Salmonellosis	All jurisdictions
Shigellosis	All jurisdictions
Typhoid fever	All jurisdictions
Quarantinable diseases	
Avian influenza in humans	All jurisdictions
Cholera	All jurisdictions
Middle East respiratory syndrome coronavirus	All jurisdictions
Plague	All jurisdictions
Rabies	All jurisdictions
Severe acute respiratory syndrome	All jurisdictions
Smallpox	All jurisdictions
Viral haemorrhagic fever	All jurisdictions
Yellow fever	All jurisdictions
Sexually transmissible infections	
Chlamydial infection	All jurisdictions
Donovanosis	All jurisdictions

Disease	Data received from:
Gonococcal infection	All jurisdictions
Syphilis < 2 years duration	All jurisdictions
Syphilis > 2 years or unspecified duration	All jurisdictions
Syphilis - congenital	All jurisdictions
Vaccine preventable diseases	
Diphtheria	All jurisdictions
Haemophilus influenzae type b	All jurisdictions
Influenza (laboratory confirmed)	All jurisdictions
Measles	All jurisdictions
Mumps	All jurisdictions
Pertussis	All jurisdictions
Pneumococcal disease – invasive	All jurisdictions
Poliovirus infection	All jurisdictions
Rubella	All jurisdictions
Rubella - congenital	All jurisdictions
Tetanus	All jurisdictions
Varicella zoster (chickenpox)	All jurisdictions except New South Wales
Varicella zoster (shingles)	All jurisdictions except New South Wales
Varicella zoster (unspecified)	All jurisdictions except New South Wales
Vectorborne diseases	
Barmah Forest virus infection	All jurisdictions
Chikungunya virus infection	All jurisdictions except Australian Capital Territory
Dengue virus infection	All jurisdictions
Flavivirus infection (unspecified)	All jurisdictions
Japanese encephalitis virus infection	All jurisdictions
Kunjin virus infection	All jurisdictions
Malaria	All jurisdictions
Murray Valley encephalitis virus infection	All jurisdictions
Ross River virus infection	All jurisdictions
Zoonoses	
Anthrax	All jurisdictions
Australian bat lyssavirus infection	All jurisdictions
Brucellosis	All jurisdictions
Leptospirosis	All jurisdictions
Lyssavirus infection (NEC)	All jurisdictions
Ornithosis	All jurisdictions
Q fever	All jurisdictions
Tularaemia	All jurisdictions
Other bacterial infections	
Legionellosis	All jurisdictions
Leprosy	All jurisdictions
Meningococcal infection – invasive	All jurisdictions
Tuberculosis	All jurisdictions

NEC Not elsewhere classified.

Table 2: Notifications of diseases received by state and territory health authorities, 1 April to 30 June 2017, by date of diagnosis*

Disease	ACT	NSW	NT	QLD	SA	TAS	VIC	WA	Total current quarter	Total last quarter	Total same period previous year	Last 5 years mean current quarter	Ratio	Year to date	Last 5 years YTD mean
Bloodborne diseases															
Hepatitis B (newly acquired) †	0	1	3	12	4	2	9	6	37	39	43	41.2	0.9	76	88.8
Hepatitis B (unspecified) ‡	29	614	23	202	63	11	449	127	1,518	1,544	1,860	1,638	0.9	3,060	3,207.2
Hepatitis C (newly acquired) †	3	10	4	79	7	7	17	23	150	158	184	154.2	1	307	316.2
Hepatitis C (unspecified) ‡	37	996	39	492	91	48	494	275	2,472	2,638	2,945	2,486.2	1	5,096	4,968
Hepatitis D	0	3	0	2	0	0	3	0	8	14	15	13.8	0.6	22	27
Gastrointestinal diseases															
Botulism	0	0	0	0	0	0	0	0	0	0	0	0.6	0	0	1.2
Campylobacteriosis	90	NN	123	1,649	632	135	1,413	705	4,747	6,585	5,225	4,270	1.1	11,283	9,178
Cryptosporidiosis	26	266	34	328	128	11	397	89	1,279	2,282	1,499	1,035.6	1.2	3,553	2,605.8
Haemolytic uraemic syndrome (HUS)	0	1	0	1	0	0	0	1	3	7	1	3.6	0.8	10	9.2
Hepatitis A	0	5	0	2	4	0	7	2	20	51	26	35	0.6	71	103.2
Hepatitis E	0	6	0	2	0	0	2	2	12	18	5	9.8	1.2	30	22.4
Listeriosis	0	7	0	8	1	0	2	2	20	22	23	19.8	1	42	43.8
Paratyphoid	0	0	0	1	1	0	4	1	7	27	13	16.4	0.4	34	46.2
STEC §	0	16	1	6	52	0	2	7	84	153	57	35.2	2.4	233	73.8
Salmonellosis	68	830	171	1,043	335	47	734	622	3,850	6,324	4,505	3,631.2	1.1	10,145	8,857
Shigellosis	1	47	146	70	5	3	96	36	404	403	351	207.8	1.9	807	471.2
Typhoid Fever	0	12	0	3	1	0	6	5	27	58	21	24	1.1	85	73.8
Quarantinable diseases															
Avian influenza in humans (AIH)	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0
Cholera	0	0	0	0	0	0	0	0	0	0	0	1.4	0	0	1.6
Middle East respiratory syndrome coronavirus (MERS-CoV)	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0
Plague	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0
Rabies	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0
Severe acute respiratory syndrome (SARS)	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0
Smallpox	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0
Viral haemorrhagic fever (NEC)	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0

Disease	ACT	NSW	NT	QLD	SA	TAS	VIC	WA	Total current quarter	Total last quarter	Total same period previous year	Last 5 years mean current quarter	Ratio	Year to date	Last 5 years YTD mean
Yellow fever	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0
Sexually transmissible infections															
Chlamydial infection ‖,¶	390	6,910	645	5,941	1,527	414	6,428	2,800	25,055	27,493	24,904	22,037.4	1.1	52,486	44,781
Donovanosis	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0.2
Gonococcal infection ¶	52	2,153	429	1,294	340	24	1,839	833	6,964	8,089	5,961	4,379.6	1.6	15,023	8,780.6
Syphilis < 2 years ¶	4	265	84	294	42	2	321	60	1,072	1,093	875	565.6	1.9	2,163	1,105.4
Syphilis > 2 years or unspecified duration ‡,¶	4	133	46	73	13	4	186	36	495	537	504	450.8	1.1	1,032	890.8
Syphilis congenital	0	0	0	1	0	0	1	0	2	2	0	0.6	3.3	4	0.8
Vaccine preventable diseases															
Diphtheria	0	0	0	2	0	0	1	0	3	1	3	0.6	5	4	1.2
Haemophilus influenzae type b	0	2	0	0	0	0	1	0	3	3	5	4.8	0.6	6	7.8
Influenza (laboratory confirmed)	95	4,811	83	3,295	1,928	130	2,150	521	13,013	8,339	7,326	6,297.8	2.1	21,302	9,852.4
Measles	0	5	0	1	0	0	4	1	11	41	19	31.4	0.4	52	83
Mumps	0	33	38	90	19	0	8	3	191	179	194	92.2	2.1	368	206
Pertussis	61	1,334	24	262	411	7	415	299	2,813	3,455	3,869	3,622.2	0.8	6,240	8,255.2
Pneumococcal disease (invasive)	7	165	15	71	59	7	124	50	498	254	436	437.4	1.1	752	646.6
Poliovirus infection	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0
Rotavirus	0	197	260	581	177	10	0	237	1,462	682	525	540.6	2.7	2,136	1,210.8
Rubella	0	2	0	0	0	0	0	0	2	5	9	6.2	0.3	7	11.8
Rubella congenital	0	0	0	0	0	0	0	0	0	0	0	0.2	0	0	0.2
Tetanus	0	0	0	0	2	0	0	0	2	1	1	0.6	3.3	3	2.2
Varicella zoster (chickenpox)	19	NN	16	73	116	18	303	120	665	725	606	510.8	1.3	1,384	993.8
Varicella zoster (shingles)	64	NN	101	14	686	83	722	448	2,118	2,209	1,722	1,416	1.5	4,314	2,836.2
Varicella zoster (unspecified)	52	NN	0	1,877	25	39	1,534	389	3,916	4,124	3,838	2,948	1.3	8,016	5,918.6
Vectorborne diseases															
Barmah Forest virus infection	0	55	7	74	0	0	2	11	149	111	89	478.6	0.3	260	999
Chikungunya virus infection	0	10	0	0	0	2	6	3	21	21	15	23.4	0.9	42	49.6
Dengue virus infection	6	62	12	54	5	1	50	45	235	422	710	515.6	0.5	656	1,180.2
Flavivirus infection (unspecified)	0	2	0	4	0	0	0	0	6	8	19	7	0.9	14	21.6

Disease	ACT	NSW	NT	QLD	SA	TAS	VIC	WA	Total current quarter	Total last quarter	Total same period previous year	Last 5 years mean current quarter	Ratio	Year to date	Last 5 years YTD mean
Japanese encephalitis virus infection	0	0	0	0	0	0	1	0	1	0	0	0.4	2.5	1	1
Malaria	6	19	3	19	2	0	7	17	73	122	74	73.2	1	195	161.8
Murray Valley encephalitis virus infection	0	0	0	0	0	0	0	0	0	0	0	0.2	0	0	0.6
Ross River virus infection	2	404	35	861	65	18	134	332	1,851	3,941	1,038	1,371	1.4	5,773	3,806.4
West Nile/Kunjin virus infection	0	0	0	0	0	0	0	4	4	0	0	0.2	20	4	0.2
Zoonoses															
Anthrax	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0
Australian bat lyssavirus infection	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0.2
Brucellosis	0	3	0	3	0	0	0	0	6	1	4	4	1.5	7	8.8
Leptospirosis	0	5	2	23	1	0	0	1	32	60	36	30.8	1	92	62.6
Lyssavirus infection (NEC)	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0
Ornithosis	0	0	0	0	0	0	5	0	5	6	2	8.2	0.6	10	15
Q fever	0	53	0	53	5	1	6	2	120	135	124	121.8	1	254	252.6
Tularaemia	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0
Other bacterial infections															
Legionellosis	0	39	0	25	9	2	34	10	119	87	102	103.4	1.2	202	194.8
Leprosy	0	1	0	0	0	0	0	0	1	2	4	3.6	0.3	3	5.8
Meningococcal disease (invasive)**	0	16	0	13	9	3	18	6	65	74	46	45.6	1.4	138	79.4
Tuberculosis	7	127	3	47	15	4	102	19	324	326	296	292	1.1	649	604.4
	1,023	**19,620**	**2,347**	**18,945**	**6,780**	**1,033**	**18,037**	**8,150**	**75,935**	**82,871**	**70,129**			**158,446**	

* The date of diagnosis is the onset date or where the date of onset was not known, the earliest of the specimen collection date, the notification date, or the notification receive date. For hepatitis B (unspecified), hepatitis C (unspecified), leprosy, syphilis (> 2 years or unspecified duration) and tuberculosis, the public health unit notification receive date was used.
† Newly acquired hepatitis includes cases where the infection was determined to be acquired within 24 months prior to diagnosis. Queensland began reporting hepatitis C newly acquired from 1 September 2016. Previous notifications are reported under hepatitis unspecified.
‡ Unspecified hepatitis and syphilis includes cases where the duration of infection could not be determined or is greater than 24 months.
§ Infection with Shiga toxin/verotoxin-producing Escherichia coli.
|| Includes Chlamydia trachomatis identified from cervical, rectal, urine, urethral and throat samples, except for South Australia, which reports only cervical, urine and urethral specimens.
¶ The national case definitions for chlamydia, gonococcal and syphilis diagnoses include infections that may be acquired through a non-sexual mode (especially in children – e.g. perinatal infections, epidemic gonococcal conjunctivitis).
** Only invasive meningococcal disease is nationally notifiable. However, New South Wales and the Australian Capital Territory also report conjunctival cases.
NN Not notifiable
NEC Not elsewhere classified
Totals comprise data from all states and territories. Cumulative figures are subject to retrospective revision so there may be discrepancies between the number of new notifications and the increment in the cumulative figure from the previous period.

Table 3: Notification rates of diseases, 1 April to 30 June 2017, by state or territory. (Annualised rate per 100,000 population)[*, †]

Disease	ACT	NSW	NT	QLD	SA	TAS	VIC	WA	Australia
Bloodborne diseases									
Hepatitis B (newly acquired) [‡]	0.0	0.1	4.9	1.0	0.9	1.5	0.6	0.9	0.6
Hepatitis B (unspecified) [§]	28.8	31.7	37.4	16.7	14.7	8.5	29.1	19.9	25.1
Hepatitis C (newly acquired) [‡]	3.0	0.5	6.5	6.5	1.6	5.4	1.1	3.6	2.5
Hepatitis C (unspecified) [§]	36.7	51.5	63.5	40.6	21.2	37.1	32.0	43.0	40.8
Hepatitis D	0.0	0.2	0.0	0.2	0.0	0.0	0.2	0.0	0.1
Gastrointestinal diseases									
Botulism	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
Campylobacteriosis	89.2	NN	200.2	136.0	147.6	104.3	91.5	110.2	115.3
Cryptosporidiosis	25.8	13.7	55.3	27.1	29.9	8.5	25.7	13.9	21.1
Haemolytic uraemic syndrome (HUS)	0.0	0.1	0.0	0.1	0.0	0.0	0.0	0.2	0.0
Hepatitis A	0.0	0.3	0.0	0.2	0.9	0.0	0.5	0.3	0.3
Hepatitis E	0.0	0.3	0.0	0.2	0.0	0.0	0.1	0.3	0.2
Listeriosis	0.0	0.4	0.0	0.7	0.2	0.0	0.1	0.3	0.3
Paratyphoid	0.0	0.0	0.0	0.1	0.2	0.0	0.3	0.2	0.1
STEC [‖]	0.0	0.8	1.6	0.5	12.1	0.0	0.1	1.1	1.4
Salmonellosis	67.4	42.9	278.3	86.0	78.2	36.3	47.5	97.2	63.6
Shigellosis	1.0	2.4	237.6	5.8	1.2	2.3	6.2	5.6	6.7
Typhoid Fever	0.0	0.6	0.0	0.2	0.2	0.0	0.4	0.8	0.4
Quarantinable diseases									
Avian influenza in humans (AIH)	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
Cholera	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
Middle East respiratory syndrome coronavirus (MERS-CoV)	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
Plague	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
Rabies	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
Severe acute respiratory syndrome (SARS)	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
Smallpox	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
Viral haemorrhagic fever (NEC)	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
Yellow fever	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0

Disease	ACT	NSW	NT	QLD	SA	TAS	VIC	WA	Australia
Sexually transmissible infections									
Chlamydial infection [¶,**]	386.6	357.1	1,049.9	490.1	356.6	319.9	416.1	437.7	413.9
Donovanosis	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
Gonococcal infection[**]	51.6	111.3	698.3	106.7	79.4	18.5	119.0	130.2	115.1
Syphilis < 2 years [**]	4.0	13.7	136.7	24.3	9.8	1.5	20.8	9.4	17.7
Syphilis > 2 years or unspecified duration [§,**]	4.0	6.9	74.9	6.0	3.0	3.1	12.0	5.6	8.2
Syphilis congenital	0.0	0.0	0.0	0.1	0.0	0.0	0.1	0.0	0.0
Vaccine preventable diseases									
Diphtheria	0.0	0.0	0.0	0.2	0.0	0.0	0.1	0.0	0.0
Haemophilus influenzae type b	0.0	0.1	0.0	0.0	0.0	0.0	0.1	0.0	0.0
Influenza (laboratory confirmed)	94.2	248.7	135.1	271.8	450.2	100.5	139.2	81.4	215.0
Measles	0.0	0.3	0.0	0.1	0.0	0.0	0.3	0.2	0.2
Mumps	0.0	1.7	61.9	7.4	4.4	0.0	0.5	0.5	3.2
Pertussis	60.5	68.9	39.1	21.6	96.0	5.4	26.9	46.7	46.5
Pneumococcal disease (invasive)	6.9	8.5	24.4	5.9	13.8	5.4	8.0	7.8	8.2
Poliovirus infection	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
Rotavirus	0.0	10.2	423.2	47.9	41.3	7.7	0.0	37.0	24.2
Rubella	0.0	0.1	0.0	0.0	0.0	0.0	0.0	0.0	0.0
Rubella congenital	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
Tetanus	0.0	0.0	0.0	0.0	0.5	0.0	0.0	0.0	0.0
Varicella zoster (chickenpox)	18.8	NN	26.0	6.0	27.1	13.9	19.6	18.8	16.1
Varicella zoster (shingles)	63.4	NN	164.4	1.2	160.2	64.1	46.7	70.0	51.4
Varicella zoster (unspecified)	51.6	NN	0.0	154.8	5.8	30.1	99.3	60.8	95.1
Vectorborne diseases									
Barmah Forest virus infection	0.0	2.8	11.4	6.1	0.0	0.0	0.1	1.7	2.5
Chikungunya virus infection	0.0	0.5	0.0	0.0	0.0	1.5	0.4	0.5	0.3
Dengue virus infection	5.9	3.2	19.5	4.5	1.2	0.8	3.2	7.0	3.9
Flavivirus infection (unspecified)	0.0	0.1	0.0	0.3	0.0	0.0	0.0	0.0	0.1
Japanese encephalitis virus infection	0.0	0.0	0.0	0.0	0.0	0.0	0.1	0.0	0.0
Malaria	5.9	1.0	4.9	1.6	0.5	0.0	0.5	2.7	1.2
Murray Valley encephalitis virus infection	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
Ross River virus infection	2.0	20.9	57.0	71.0	15.2	13.9	8.7	51.9	30.6

Disease	ACT	NSW	NT	QLD	SA	TAS	VIC	WA	Australia
West Nile/Kunjin virus infection	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.6	0.1
Zoonoses									
Anthrax	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
Australian bat lyssavirus infection	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
Brucellosis	0.0	0.2	0.0	0.2	0.0	0.0	0.0	0.0	0.1
Leptospirosis	0.0	0.3	3.3	1.9	0.2	0.0	0.0	0.2	0.5
Lyssavirus infection (NEC)	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
Ornithosis	0.0	0.0	0.0	0.0	0.0	0.0	0.3	0.0	0.1
Q fever	0.0	2.7	0.0	4.4	1.2	0.8	0.4	0.3	2.0
Tularaemia	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
Other bacterial infections									
Legionellosis	0.0	2.0	0.0	2.1	2.1	1.5	2.2	1.6	2.0
Leprosy	0.0	0.1	0.0	0.0	0.0	0.0	0.0	0.0	0.0
Meningococcal disease (invasive) ††	0.0	0.8	0.0	1.1	2.1	2.3	1.2	0.9	1.1
Tuberculosis	6.9	6.6	4.9	3.9	3.5	3.1	6.6	3.0	5.4

* The date of diagnosis is the onset date or where the date of onset was not known, the earliest of the specimen collection date, the notification date, or the notification receive date. For hepatitis B (unspecified), hepatitis C (unspecified), leprosy, syphilis (> 2 years or unspecified duration) and tuberculosis, the public health unit notification receive date was used.
† Rate per 100,000 of population. Annualisation Factor was 4.0
‡ Newly acquired hepatitis includes cases where the infection was determined to be acquired within 24 months prior to diagnosis. Queensland began reporting hepatitis C newly acquired from 1 September 2016. Previous notifications are reported under hepatitis unspecified.
§ Unspecified hepatitis and syphilis includes cases where the duration of infection could not be determined or is greater than 24 months.
|| Infection with Shiga toxin/verotoxin-producing Escherichia coli.
¶ Includes Chlamydia trachomatis identified from cervical, rectal, urine, urethral and throat samples, except for South Australia, which reports only cervical, urine and urethral specimens.
** The national case definitions for chlamydia, gonococcal and syphilis diagnoses include infections that may be acquired through a non-sexual mode (especially in children – e.g. perinatal infections, epidemic gonococcal conjunctivitis).
†† Only invasive meningococcal disease is nationally notifiable. However, New South Wales and the Australian Capital Territory also report conjunctival cases.
NEC Not elsewhere classified.
NN Not notifiable.

Submit an Article

You are invited to submit your next communicable disease related article to the Communicable Diseases Intelligence (CDI) for consideration.

More information regarding CDI can be found at: http://health.gov.au/cdi.

Further enquiries should be direct to: cdi.editor@health.gov.au.

Quarterly report

National Notifiable Diseases Surveillance System, 1 July to 30 September 2017

Office of Health Protection, Department of Health

A summary of diseases currently being reported by each jurisdiction is provided in Table 1. There were 267,220 notifications to the National Notifiable Diseases Surveillance System (NNDSS) between 1 July to 30 September 2017 (Table 2). The notification rate of diseases per 100,000 population for each state or territory is presented in Table 3.

Table 1: Reporting of notifiable diseases by jurisdiction

Disease	Data received from:
Bloodborne diseases	
Hepatitis (NEC)	All jurisdictions
Hepatitis B (newly acquired)	All jurisdictions
Hepatitis B (unspecified)	All jurisdictions
Hepatitis C (newly acquired)	All jurisdictions
Hepatitis C (unspecified)	All jurisdictions
Hepatitis D	All jurisdictions
Gastrointestinal diseases	
Botulism	All jurisdictions
Campylobacteriosis	All jurisdictions except New South Wales
Cryptosporidiosis	All jurisdictions
Haemolytic uraemic syndrome	All jurisdictions
Hepatitis A	All jurisdictions
Hepatitis E	All jurisdictions
Listeriosis	All jurisdictions
Paratyphoid	All jurisdictions
Shiga toxin/verotoxin-producing *Escherichia coli*	All jurisdictions
Salmonellosis	All jurisdictions
Shigellosis	All jurisdictions
Typhoid fever	All jurisdictions
Quarantinable diseases	
Avian influenza in humans	All jurisdictions
Cholera	All jurisdictions
Middle East respiratory syndrome coronavirus	All jurisdictions
Plague	All jurisdictions
Rabies	All jurisdictions
Severe acute respiratory syndrome	All jurisdictions
Smallpox	All jurisdictions
Viral haemorrhagic fever	All jurisdictions
Yellow fever	All jurisdictions
Sexually transmissible infections	
Chlamydial infection	All jurisdictions
Donovanosis	All jurisdictions

Disease	Data received from:
Gonococcal infection	All jurisdictions
Syphilis < 2 years duration	All jurisdictions
Syphilis > 2 years or unspecified duration	All jurisdictions
Syphilis - congenital	All jurisdictions
Vaccine preventable diseases	
Diphtheria	All jurisdictions
Haemophilus influenzae type b	All jurisdictions
Influenza (laboratory confirmed)	All jurisdictions
Measles	All jurisdictions
Mumps	All jurisdictions
Pertussis	All jurisdictions
Pneumococcal disease – invasive	All jurisdictions
Poliovirus infection	All jurisdictions
Rubella	All jurisdictions
Rubella - congenital	All jurisdictions
Tetanus	All jurisdictions
Varicella zoster (chickenpox)	All jurisdictions except New South Wales
Varicella zoster (shingles)	All jurisdictions except New South Wales
Varicella zoster (unspecified)	All jurisdictions except New South Wales
Vectorborne diseases	
Barmah Forest virus infection	All jurisdictions
Chikungunya virus infection	All jurisdictions except Australian Capital Territory
Dengue virus infection	All jurisdictions
Flavivirus infection (unspecified)	All jurisdictions
Japanese encephalitis virus infection	All jurisdictions
Kunjin virus infection	All jurisdictions
Malaria	All jurisdictions
Murray Valley encephalitis virus infection	All jurisdictions
Ross River virus infection	All jurisdictions
Zoonoses	
Anthrax	All jurisdictions
Australian bat lyssavirus infection	All jurisdictions
Brucellosis	All jurisdictions
Leptospirosis	All jurisdictions
Lyssavirus infection (NEC)	All jurisdictions
Ornithosis	All jurisdictions
Q fever	All jurisdictions
Tularaemia	All jurisdictions
Other bacterial infections	
Legionellosis	All jurisdictions
Leprosy	All jurisdictions
Meningococcal infection – invasive	All jurisdictions
Tuberculosis	All jurisdictions

NEC Not elsewhere classified.

Table 2: Notifications of diseases received by state and territory health authorities, 1 July to 30 September 2017, by date of diagnosis*

Disease	ACT	NSW	NT	QLD	SA	TAS	VIC	WA	Total current quarter	Total last quarter	Total same period previous year	Last 5 years mean current quarter	Ratio	Year to date	Last 5 years YTD mean
Bloodborne diseases															
Hepatitis B (newly acquired) †	0	4	1	11	2	1	10	4	33	37	35	39.6	0.8	109	128.4
Hepatitis B (unspecified) ‡	16	562	19	217	74	12	437	145	1,482	1,541	1,569	1,695	0.9	4,542	4,902.2
Hepatitis C (newly acquired) †	3	13	2	73	3	8	9	36	147	151	164	151.6	1	454	467.8
Hepatitis C (unspecified) ‡	25	1,069	27	518	92	60	500	242	2,533	2,509	2,626	2,538	1	7,629	7,506.2
Hepatitis D	0	9	0	5	3	0	9	0	26	8	14	11.4	2.3	48	38.4
Gastrointestinal diseases															
Botulism	0	0	0	0	0	0	0	0	0	0	0	0.2	0	0	1.4
Campylobacteriosis	83	NN	101	1,541	751	158	1,514	851	4,999	4,790	6,021	4,687.2	1.1	16,282	13,865.2
Cryptosporidiosis	4	103	9	87	63	16	195	39	516	1,294	656	462.6	1.1	4,069	3,068.4
Haemolytic uraemic syndrome	0	0	0	1	0	0	0	0	1	4	5	4	0.3	11	13.2
Hepatitis A	1	36	0	6	2	0	12	3	60	21	23	37.2	1.6	131	140.4
Hepatitis E	0	2	0	5	0	0	2	1	10	12	8	6	1.7	40	28.4
Listeriosis	0	3	0	3	3	0	2	2	13	20	13	15.6	0.8	55	59.4
Paratyphoid	0	3	0	1	1	0	4	0	9	7	13	10	0.9	43	56.2
STEC §	0	8	1	5	79	1	9	13	116	86	94	46.8	2.5	349	120.6
Salmonellosis	43	548	84	553	199	54	604	484	2,569	3,917	3,155	2,557.8	1	12,714	11,414.8
Shigellosis	4	61	112	40	31	8	121	52	429	406	329	212.4	2	1,236	683.6
Typhoid Fever	1	10	0	2	0	0	6	4	23	27	21	18.4	1.3	108	92.2
Quarantinable diseases															
Avian Influenza in Humans	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0
Cholera	0	0	0	0	0	0	0	1	1	0	0	0.2	5	1	1.8
Middle East respiratory syndrome coronavirus	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0
Plague	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0
Rabies	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0
Severe Acute Respiratory Syndrome	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0
Smallpox	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0
Viral haemorrhagic fever	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0

Disease	ACT	NSW	NT	QLD	SA	TAS	VIC	WA	Total current quarter	Total last quarter	Total same period previous year	Last 5 years mean current quarter	Ratio	Year to date	Last 5 years YTD mean
Yellow fever	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0
Sexually transmissible infections															
Chlamydial infection ‖,¶	321	6,913	723	5,605	1,450	407	4,674	2,705	22,798	25,406	23,195	21,244.4	1.1	75,284	66,025.4
Donovanosis	0	0	0	0	0	0	0	0	0	0	0	0.2	0	0	0.4
Gonococcal infection ¶	54	2,193	474	1,158	326	28	1,673	807	6,713	7,046	6,105	4,235	1.6	21,736	1,3015.6
Syphilis < 2 years ¶	7	305	95	254	47	4	343	72	1,127	1,083	922	627	1.8	3,290	1,732.4
Syphilis > 2 years or unspecified duration ‡,¶	4	118	25	88	12	6	215	43	511	501	541	459.2	1.1	1,543	1,350
Syphilis congenital	0	0	0	1	0	0	1	0	2	2	2	2	1	6	2.8
Vaccine preventable diseases															
Diphtheria	0	0	0	0	0	0	1	0	1	3	1	0.8	1.3	5	2
Haemophilus influenzae type b	0	1	0	1	0	0	1	0	3	3	5	6.6	0.5	9	14.4
Influenza (laboratory confirmed)	2,663	91,800	678	44,697	20,526	2,957	40,330	3,612	207,263	13,107	62,686	49,048.6	4.2	228,565	58,901
Measles	0	3	0	0	1	0	11	5	20	12	14	52.6	0.4	72	135.6
Mumps	3	22	61	103	13	1	9	7	219	196	164	101	2.2	587	307
Pertussis	45	1,241	13	399	420	11	480	490	3,099	2,848	4,775	4,407.4	0.7	9,339	12,662.6
Pneumococcal disease (invasive)	5	296	32	139	82	21	209	86	870	502	648	622.2	1.4	1,622	1,268.8
Poliovirus infection	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0
Rotavirus	0	1,225	47	927	545	32	0	74	2,850	1,467	550	994.6	2.9	4,986	2,205.4
Rubella	0	3	0	0	0	0	0	0	3	2	2	5.6	0.5	10	17.4
Rubella congenital	0	0	0	0	0	0	0	0	0	0	0	0.4	0	0	0.6
Tetanus	0	0	0	0	0	0	0	0	0	2	2	0.8	0	3	3
Varicella zoster (chickenpox)	27	NN	22	168	113	12	282	230	854	672	823	653	1.3	2,238	1,646.8
Varicella zoster (shingles)	57	NN	116	18	712	85	639	515	2,142	2,139	1,849	1,377	1.6	6,456	4,213.2
Varicella zoster (unspecified)	64	NN	1	2,009	68	37	1,513	396	4,088	3,965	3,950	3,131.2	1.3	12,104	9,049.8
Vectorborne diseases															
Barmah Forest virus infection	0	24	2	46	0	0	3	6	81	150	45	259.2	0.3	341	1,258.2
Chikungunya virus infection	0	21	0	1	0	0	7	2	31	21	34	22.2	1.4	73	71.8
Dengue virus infection	7	60	2	21	9	1	57	33	190	240	439	339.8	0.6	846	1,520
Flavivirus infection (unspecified)	0	0	0	0	0	0	0	0	0	6	29	7.8	0	14	29.4

Disease	ACT	NSW	NT	QLD	SA	TAS	VIC	WA	Total current quarter	Total last quarter	Total same period previous year	Last 5 years mean current quarter	Ratio	Year to date	Last 5 years YTD mean
Japanese encephalitis virus infection	0	0	0	0	0	0	0	0	0	1	0	0.6	0	1	1.6
Malaria	3	16	4	25	1	0	33	13	95	75	84	87.4	1.1	290	249.2
Murray Valley encephalitis virus infection	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0.6
Ross River virus infection	0	118	25	275	12	2	25	121	578	1,888	281	625.2	0.9	6,351	4,431.6
West Nile/Kunjin virus infection	0	0	0	0	0	0	1	1	2	4	0	0	0	6	0.2
Zoonoses															
Anthrax	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0
Australian bat lyssavirus infection	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0.2
Brucellosis	0	0	0	1	1	0	1	0	3	6	7	6.4	0.5	10	15.2
Leptospirosis	0	3	0	18	0	0	10	1	32	32	19	16.8	1.9	124	79.4
Lyssavirus infection	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0
Ornithosis	0	3	0	1	0	0	0	1	5	5	6	9	0.6	15	24
Q fever	0	45	0	41	5	0	6	1	98	121	136	124.6	0.8	352	377.2
Tularaemia	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0
Other bacterial infections															
Legionellosis	0	34	0	15	6	2	8	6	71	119	86	110.8	0.6	273	305.6
Leprosy	0	0	0	1	0	0	0	1	2	1	7	3.8	0.5	5	9.6
Meningococcal disease (invasive) **	2	40	19	17	17	3	31	12	141	65	79	66.4	2.1	279	145.8
Tuberculosis	7	135	4	50	17	3	108	37	361	327	365	339.6	1.1	1,010	944
	3,449	107,050	2,699	59,147	25,686	3,930	54,105	11,154	267,220	76,847	122,597			409,275	

* The date of diagnosis is the onset date or where the date of onset was not known, the earliest of the specimen collection date, the notification date, or the notification receive date. For hepatitis B (unspecified), hepatitis C (unspecified), leprosy, syphilis (> 2 years or unspecified duration) and tuberculosis, the public health unit notification receive date was used.
† Newly acquired hepatitis includes cases where the infection was determined to be acquired within 24 months prior to diagnosis. Queensland began reporting hepatitis C newly acquired from 1 September 2016. Previous notifications are reported under hepatitis unspecified.
‡ Unspecified hepatitis and syphilis includes cases where the duration of infection could not be determined or is greater than 24 months.
§ Infection with Shiga toxin/verotoxin-producing Escherichia coli.
|| Includes Chlamydia trachomatis identified from cervical, rectal, urine, urethral and throat samples, except for South Australia, which reports only cervical, urine and urethral specimens.
¶ The national case definitions for chlamydia, gonococcal and syphilis diagnoses include infections that may be acquired through a non-sexual mode (especially in children – e.g. perinatal infections, epidemic gonococcal conjunctivitis).
** Only invasive meningococcal disease is nationally notifiable. However, New South Wales and the Australian Capital Territory also report conjunctival cases.
NN Not notifiable
NECNot elsewhere classified
Totals comprise data from all states and territories. Cumulative figures are subject to retrospective revision so there may be discrepancies between the number of new notifications and the increment in the cumulative figure from the previous period.

Table 3: Notification rates of diseases, 1 July to 30 September 2017, by state or territory. (Annualised rate per 100,000 population)[*,†]

Disease	ACT	NSW	NT	QLD	SA	TAS	VIC	WA	Australia
Bloodborne diseases									
Hepatitis B (newly acquired) [‡]	0.0	0.2	1.6	0.9	0.5	0.8	0.6	0.6	0.5
Hepatitis B (unspecified) [§]	15.9	29.0	30.9	17.9	17.3	9.3	28.3	22.7	24.5
Hepatitis C (newly acquired) [‡]	3.0	0.7	3.3	6.0	0.7	6.2	0.6	5.6	2.4
Hepatitis C (unspecified) [§]	24.8	55.3	43.9	42.7	21.5	46.4	32.4	37.8	41.8
Hepatitis D	0.0	0.5	0.0	0.4	0.7	0.0	0.6	0.0	0.4
Gastrointestinal diseases									
Botulism	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
Campylobacteriosis	82.3	NN	164.4	127.1	175.4	122.1	98.0	133.0	121.4
Cryptosporidiosis	4.0	5.3	14.6	7.2	14.7	12.4	12.6	6.1	8.5
Haemolytic uraemic syndrome (HUS)	0.0	0.0	0.0	0.1	0.0	0.0	0.0	0.0	0.0
Hepatitis A	1.0	1.9	0.0	0.5	0.5	0.0	0.8	0.5	1.0
Hepatitis E	0.0	0.1	0.0	0.4	0.0	0.0	0.1	0.2	0.2
Listeriosis	0.0	0.2	0.0	0.2	0.7	0.0	0.1	0.3	0.2
Paratyphoid	0.0	0.2	0.0	0.1	0.2	0.0	0.3	0.0	0.1
STEC [‖]	0.0	0.4	1.6	0.4	18.4	0.8	0.6	2.0	1.9
Salmonellosis	42.6	28.3	136.7	45.6	46.5	41.7	39.1	75.7	42.4
Shigellosis	4.0	3.2	182.3	3.3	7.2	6.2	7.8	8.1	7.1
Typhoid Fever	1.0	0.5	0.0	0.2	0.0	0.0	0.4	0.6	0.4
Quarantinable diseases									
Avian influenza in humans (AIH)	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
Cholera	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.2	0.0
Middle East respiratory syndrome coronavirus (MERS-CoV)	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
Plague	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
Rabies	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
Severe acute respiratory syndrome (SARS)	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
Smallpox	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
Viral haemorrhagic fever (NEC)	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
Yellow fever	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0

Disease	ACT	NSW	NT	QLD	SA	TAS	VIC	WA	Australia
Sexually transmissible infections									
Chlamydial infection [¶,**]	318.2	357.3	1,176.9	462.4	338.6	314.5	302.6	422.8	376.7
Donovanosis	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
Gonococcal infection[**]	53.5	113.3	771.5	95.5	76.1	21.6	108.3	126.1	110.9
Syphilis < 2 years [**]	6.9	15.8	154.6	21.0	11.0	3.1	22.2	11.3	18.6
Syphilis > 2 years or unspecified duration [§,**]	4.0	6.1	40.7	7.3	2.8	4.6	13.9	6.7	8.4
Syphilis congenital	0.0	0.0	0.0	0.1	0.0	0.0	0.1	0.0	0.0
Vaccine preventable diseases									
Diphtheria	0.0	0.0	0.0	0.0	0.0	0.0	0.1	0.0	0.0
Haemophilus influenzae type b	0.0	0.1	0.0	0.1	0.0	0.0	0.1	0.0	0.0
Influenza (laboratory confirmed)	2,640.1	4,744.6	1,103.6	3,687.2	4,792.8	2,285.2	2,610.7	564.6	3,424.3
Measles	0.0	0.2	0.0	0.0	0.2	0.0	0.7	0.8	0.3
Mumps	3.0	1.1	99.3	8.5	3.0	0.8	0.6	1.1	3.6
Pertussis	44.6	64.1	21.2	32.9	98.1	8.5	31.1	76.6	51.2
Pneumococcal disease (invasive)	5.0	15.3	52.1	11.5	19.1	16.2	13.5	13.4	14.4
Poliovirus infection	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
Rotavirus	0.0	63.3	76.5	76.5	127.3	24.7	0.0	11.6	47.1
Rubella	0.0	0.2	0.0	0.0	0.0	0.0	0.0	0.0	0.0
Rubella congenital	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
Tetanus	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
Varicella zoster (chickenpox)	26.8	NN	35.8	13.9	26.4	9.3	18.3	36.0	20.7
Varicella zoster (shingles)	56.5	NN	188.8	1.5	166.3	65.7	41.4	80.5	52.0
Varicella zoster (unspecified)	63.4	NN	1.6	165.7	15.9	28.6	97.9	61.9	99.3
Vectorborne diseases									
Barmah Forest virus infection	0.0	1.2	3.3	3.8	0.0	0.0	0.2	0.9	1.3
Chikungunya virus infection	0.0	1.1	0.0	0.1	0.0	0.0	0.5	0.3	0.5
Dengue virus infection	6.9	3.1	3.3	1.7	2.1	0.8	3.7	5.2	3.1
Flavivirus infection (unspecified)	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
Japanese encephalitis virus infection	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
Malaria	3.0	0.8	6.5	2.1	0.2	0.0	2.1	2.0	1.6
Murray Valley encephalitis virus infection	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
Ross River virus infection	0.0	6.1	40.7	22.7	2.8	1.5	1.6	18.9	9.5

Disease	ACT	NSW	NT	QLD	SA	TAS	VIC	WA	Australia
West Nile/Kunjin virus infection	0.0	0.0	0.0	0.0	0.0	0.0	0.1	0.2	0.0
Zoonoses									
Anthrax	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
Australian bat lyssavirus infection	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
Brucellosis	0.0	0.0	0.0	0.1	0.2	0.0	0.1	0.0	0.0
Leptospirosis	0.0	0.2	0.0	1.5	0.0	0.0	0.6	0.2	0.5
Lyssavirus infection (NEC)	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
Ornithosis	0.0	0.2	0.0	0.1	0.0	0.0	0.0	0.2	0.1
Q fever	0.0	2.3	0.0	3.4	1.2	0.0	0.4	0.2	1.6
Tularaemia	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
Other bacterial infections									
Legionellosis	0.0	1.8	0.0	1.2	1.4	1.5	0.5	0.9	1.2
Leprosy	0.0	0.0	0.0	0.1	0.0	0.0	0.0	0.2	0.0
Meningococcal disease (invasive)††	2.0	2.1	30.9	1.4	4.0	2.3	2.0	1.9	2.3
Tuberculosis	6.9	7.0	6.5	4.1	4.0	2.3	7.0	5.8	6.0

* The date of diagnosis is the onset date or where the date of onset was not known, the earliest of the specimen collection date, the notification date, or the notification receive date. For hepatitis B (unspecified), hepatitis C (unspecified), leprosy, syphilis (> 2 years or unspecified duration) and tuberculosis, the public health unit notification receive date was used.
† Rate per 100,000 of population. Annualisation Factor was 4.0
‡ Newly acquired hepatitis includes cases where the infection was determined to be acquired within 24 months prior to diagnosis. Queensland began reporting hepatitis C newly acquired from 1 September 2016. Previous notifications are reported under hepatitis unspecified.
§ Unspecified hepatitis and syphilis includes cases where the duration of infection could not be determined or is greater than 24 months.
|| Infection with Shiga toxin/verotoxin-producing Escherichia coli.
¶ Includes Chlamydia trachomatis identified from cervical, rectal, urine, urethral and throat samples, except for South Australia, which reports only cervical, urine and urethral specimens.
** The national case definitions for chlamydia, gonococcal and syphilis diagnoses include infections that may be acquired through a non-sexual mode (especially in children – e.g. perinatal infections, epidemic gonococcal conjunctivitis).
†† Only invasive meningococcal disease is nationally notifiable. However, New South Wales and the Australian Capital Territory also report conjunctival cases.
NEC Not elsewhere classified.
NN Not notifiable.

Submit an Article

You are invited to submit your next communicable disease related article to the Communicable Diseases Intelligence (CDI) for consideration.

More information regarding CDI can be found at: http://health.gov.au/cdi.

Further enquiries should be direct to: cdi.editor@health.gov.au.

Quarterly report

National Notifiable Diseases Surveillance System, 1 October to 31 December 2017

Office of Health Protection, Department of Health

A summary of diseases currently being reported by each jurisdiction is provided in Table 1. There were 80,388 notifications to the National Notifiable Diseases Surveillance System (NNDSS) between 1 October to 31 December 2017 (Table 2). The notification rate of diseases per 100,000 population for each state or territory is presented in Table 3.

Table 1: Reporting of notifiable diseases by jurisdiction

Disease	Data received from:
Bloodborne diseases	
Hepatitis (NEC)	All jurisdictions
Hepatitis B (newly acquired)	All jurisdictions
Hepatitis B (unspecified)	All jurisdictions
Hepatitis C (newly acquired)	All jurisdictions
Hepatitis C (unspecified)	All jurisdictions
Hepatitis D	All jurisdictions
Gastrointestinal diseases	
Botulism	All jurisdictions
Campylobacteriosis	All jurisdictions except New South Wales
Cryptosporidiosis	All jurisdictions
Haemolytic uraemic syndrome	All jurisdictions
Hepatitis A	All jurisdictions
Hepatitis E	All jurisdictions
Listeriosis	All jurisdictions
Paratyphoid	All jurisdictions
Shiga toxin/verotoxin-producing *Escherichia coli*	All jurisdictions
Salmonellosis	All jurisdictions
Shigellosis	All jurisdictions
Typhoid fever	All jurisdictions
Quarantinable diseases	
Avian influenza in humans	All jurisdictions
Cholera	All jurisdictions
Middle East respiratory syndrome coronavirus	All jurisdictions
Plague	All jurisdictions
Rabies	All jurisdictions
Severe acute respiratory syndrome	All jurisdictions
Smallpox	All jurisdictions
Viral haemorrhagic fever	All jurisdictions
Yellow fever	All jurisdictions
Sexually transmissible infections	
Chlamydial infection	All jurisdictions
Donovanosis	All jurisdictions

Disease	Data received from:
Gonococcal infection	All jurisdictions
Syphilis < 2 years duration	All jurisdictions
Syphilis > 2 years or unspecified duration	All jurisdictions
Syphilis - congenital	All jurisdictions
Vaccine preventable diseases	
Diphtheria	All jurisdictions
Haemophilus influenzae type b	All jurisdictions
Influenza (laboratory confirmed)	All jurisdictions
Measles	All jurisdictions
Mumps	All jurisdictions
Pertussis	All jurisdictions
Pneumococcal disease – invasive	All jurisdictions
Poliovirus infection	All jurisdictions
Rubella	All jurisdictions
Rubella - congenital	All jurisdictions
Tetanus	All jurisdictions
Varicella zoster (chickenpox)	All jurisdictions except New South Wales
Varicella zoster (shingles)	All jurisdictions except New South Wales
Varicella zoster (unspecified)	All jurisdictions except New South Wales
Vectorborne diseases	
Barmah Forest virus infection	All jurisdictions
Chikungunya virus infection	All jurisdictions except Australian Capital Territory
Dengue virus infection	All jurisdictions
Flavivirus infection (unspecified)	All jurisdictions
Japanese encephalitis virus infection	All jurisdictions
Kunjin virus infection	All jurisdictions
Malaria	All jurisdictions
Murray Valley encephalitis virus infection	All jurisdictions
Ross River virus infection	All jurisdictions
Zoonoses	
Anthrax	All jurisdictions
Australian bat lyssavirus infection	All jurisdictions
Brucellosis	All jurisdictions
Leptospirosis	All jurisdictions
Lyssavirus infection (NEC)	All jurisdictions
Ornithosis	All jurisdictions
Q fever	All jurisdictions
Tularaemia	All jurisdictions
Other bacterial infections	
Legionellosis	All jurisdictions
Leprosy	All jurisdictions
Meningococcal infection – invasive	All jurisdictions
Tuberculosis	All jurisdictions

NEC Not elsewhere classified.

Table 2: Notifications of diseases received by state and territory health authorities, 1 October to 31 December 2017, by date of diagnosis*

Disease	ACT	NSW	NT	QLD	SA	TAS	VIC	WA	Total current quarter	Total last quarter	Total same period previous year	Last 5 years mean current quarter	Ratio	Year to date	Last 5 years YTD mean
Bloodborne diseases															
Hepatitis B (newly acquired) †	0	5	0	8	2	1	8	8	32	33	37	39	0.8	141	167.4
Hepatitis B (unspecified) ‡	17	553	29	201	72	11	416	126	1,425	1,482	1,411	1,544	0.9	5,967	6,446.2
Hepatitis C (newly acquired) †	2	13	0	85	6	5	11	34	156	147	186	157.8	1	610	625.6
Hepatitis C (unspecified) ‡	31	1,041	37	517	82	61	425	266	2,460	2,533	2,796	2,532	1	10,089	10,038.2
Hepatitis D	0	6	0	1	5	0	4	0	16	26	15	12.8	1.3	64	51.2
Gastrointestinal diseases															
Botulism	0	1	0	0	1	0	0	0	2	0	0	0.2	10	2	1.6
Campylobacteriosis	142	NN	73	2,139	789	221	1,807	932	6,103	4,999	6,993	5,545.8	1.1	22,385	19,411
Cryptosporidiosis	2	136	17	157	28	12	234	28	614	516	952	708.4	0.9	4,683	3,776.8
Haemolytic uraemic syndrome (HUS)	0	0	0	0	2	0	1	0	3	1	6	4.8	0.6	14	18
Hepatitis A	0	22	0	7	15	0	35	1	80	60	41	41.8	1.9	211	182.2
Hepatitis E	0	4	0	1	0	0	2	0	7	10	15	13.2	0.5	47	41.6
Listeriosis	1	6	0	2	1	0	5	0	15	13	21	21.2	0.7	70	80.6
Paratyphoid	2	10	0	1	2	0	8	0	23	9	17	19.2	1.2	66	75.4
STEC §	0	14	0	7	85	1	12	22	141	116	146	56.2	2.5	490	176.8
Salmonellosis	33	834	137	883	407	96	776	558	3,724	2,569	3,872	3,623.4	1	16,438	15,038.2
Shigellosis	2	65	137	58	20	8	162	69	521	429	362	230	2.3	1,757	913.6
Typhoid Fever	0	10	0	2	1	0	10	10	33	23	25	29.2	1.1	141	121.4
Quarantinable diseases															
Avian influenza in humans (AIH)	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0
Cholera	0	1	0	0	0	0	0	0	1	1	1	0.8	1.3	2	2.6
Middle East respiratory syndrome coronavirus (MERS-CoV)	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0
Plague	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0
Rabies	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0
Severe acute respiratory syndrome (SARS)	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0
Smallpox	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0
Viral haemorrhagic fever (NEC)	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0

Disease	ACT	NSW	NT	QLD	SA	TAS	VIC	WA	Total current quarter	Total last quarter	Total same period previous year	Last 5 years mean current quarter	Ratio	Year to date	Last 5 years YTD mean
Yellow fever	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0
Sexually transmissible infections															
Chlamydial infection ‖, ¶	369	7,171	678	5,497	1,368	339	2,944	2,702	21,068	22,798	22,586	20,921.8	1	96,352	86,947.2
Donovanosis	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0.4
Gonococcal infection ¶	73	2,266	457	1,139	312	24	1,758	646	6,675	6,713	6,216	4,361.8	1.5	28,411	17,377.4
Syphilis < 2 years ¶	8	265	81	250	40	2	324	87	1,057	1,127	833	587	1.8	4,347	2,319.4
Syphilis > 2 years or unspecified duration ‡, ¶	2	137	22	68	21	0	186	48	484	511	491	429	1.1	2,027	1,779
Syphilis congenital	0	1	0	1	0	0	0	0	2	2	0	0.4	5	8	3.2
Vaccine preventable diseases															
Diphtheria	0	0	0	1	0	0	1	1	3	1	3	1	3	8	3
Haemophilus influenzae type b	0	4	1	1	0	0	0	0	6	3	3	3.6	1.7	15	18
Influenza (laboratory confirmed)	272	4,927	223	5,195	4,248	311	4,582	1,464	21,222	207,263	15,034	7,505.8	2.8	249,787	66,406.8
Measles	2	4	0	0	0	0	3	0	9	20	29	38.2	0.2	81	173.8
Mumps	0	37	27	138	3	2	10	5	222	219	110	103.6	2.1	809	410.6
Pertussis	23	1,083	32	337	360	15	517	412	2,779	3,099	5,571	5,532.6	0.5	12,118	18,195.2
Pneumococcal disease (invasive)	9	143	14	59	43	12	106	32	418	870	396	351.4	1.2	2,040	1,620.2
Poliovirus infection	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0
Rotavirus	0	739	7	527	558	87	0	97	2,015	2,850	855	1,110.6	1.8	7,001	3,316
Rubella	0	1	0	0	0	0	0	0	1	3	1	4.4	0.2	11	21.8
Rubella congenital	0	0	0	0	0	0	0	0	0	0	0	0.2	0	0	0.8
Tetanus	0	0	0	1	0	0	0	0	1	0	1	1.6	0.6	4	4.6
Varicella zoster (chickenpox)	42	NN	17	182	134	13	349	178	915	854	1,001	702.6	1.3	3,153	2,349.4
Varicella zoster (shingles)	82	NN	95	554	662	104	667	508	2,672	2,142	2,106	1,558	1.7	9,128	5,771.2
Varicella zoster (unspecified)	49	NN	1	1,364	93	43	1,784	401	3,735	4,088	3,983	3,303.8	1.1	15,839	12,353.6
Vectorborne diseases															
Barmah Forest virus infection	0	22	2	64	1	0	2	16	107	81	68	275	0.4	448	1,533.2
Chikungunya virus infection	0	10	0	3	0	0	10	2	25	31	47	25.8	1	98	97.6
Dengue virus infection	16	87	3	36	6	4	39	33	224	190	339	290.8	0.8	1,070	1,810.8
Flavivirus infection (unspecified)	0	0	0	1	0	0	0	0	1	0	14	4.2	0.2	15	33.6

Disease	ACT	NSW	NT	QLD	SA	TAS	VIC	WA	Total current quarter	Total last quarter	Total same period previous year	Last 5 years mean current quarter	Ratio	Year to date	Last 5 years YTD mean
Japanese encephalitis virus infection	0	0	0	0	0	0	0	0	0	0	0	0.2	0	1	1.8
Malaria	2	11	0	28	1	2	18	7	69	95	76	76.6	0.9	359	325.8
Murray Valley encephalitis virus infection	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0.6
Ross River virus infection	0	121	27	202	13	0	34	165	562	578	1,183	1,085.2	0.5	6,913	5,516.8
West Nile/Kunjin virus infection	0	0	0	0	0	0	0	0	0	2	0	0.6	0	6	0.8
Zoonoses															
Anthrax	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0
Australian bat lyssavirus infection	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0.2
Brucellosis	0	4	0	4	0	0	0	0	8	3	5	4.6	1.7	18	19.8
Leptospirosis	0	2	2	12	0	0	1	1	18	32	21	15.6	1.2	142	95
Lyssavirus infection (NEC)	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0
Ornithosis	0	0	0	0	0	0	6	0	6	5	13	16.4	0.4	21	40.4
Q fever	0	41	1	43	7	2	6	2	102	98	149	121.6	0.8	454	498.8
Tularaemia	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0
Other bacterial infections															
Legionellosis	0	33	0	21	9	1	36	12	112	71	97	104.2	1.1	385	409.8
Leprosy	0	1	0	2	1	0	0	1	5	2	6	3.6	1.4	10	13.2
Meningococcal disease (invasive)**	0	20	13	16	5	6	24	19	103	141	82	48.6	2.1	382	194.4
Tuberculosis	3	144	7	51	12	3	136	50	406	361	389	363.2	1.1	1,416	1,307.2
	1,184	19,995	2,140	19,866	9,415	1,386	17,459	8,943	80,388	267,220	78,604			506,054	

* The date of diagnosis is the onset date or where the date of onset was not known, the earliest of the specimen collection date, the notification date, or the notification receive date. For hepatitis B (unspecified), hepatitis C (unspecified), leprosy, syphilis (> 2 years or unspecified duration) and tuberculosis, the public health unit notification receive date was used.
† Newly acquired hepatitis includes cases where the infection was determined to be acquired within 24 months prior to diagnosis. Queensland began reporting hepatitis C newly acquired from 1 September 2016. Previous notifications are reported under hepatitis unspecified.
‡ Unspecified hepatitis and syphilis includes cases where the duration of infection could not be determined or is greater than 24 months.
§ Infection with Shiga toxin/verotoxin-producing Escherichia coli.
|| Includes Chlamydia trachomatis identified from cervical, rectal, urine, urethral and throat samples, except for South Australia, which reports only cervical, urine and urethral specimens.

¶ The national case definitions for chlamydia, gonococcal and syphilis diagnoses include infections that may be acquired through a non-sexual mode (especially in children – e.g. perinatal infections, epidemic gonococcal conjunctivitis).
** Only invasive meningococcal disease is nationally notifiable. However, New South Wales and the Australian Capital Territory also report conjunctival cases.
NN Not notifiable
NEC Not elsewhere classified
Totals comprise data from all states and territories. Cumulative figures are subject to retrospective revision so there may be discrepancies between the number of new notifications and the increment in the cumulative figure from the previous period.

Table 3: Notification rates of diseases, 1 October to 31 December 2017, by state or territory. (Annualised rate per 100,000 population)*, †

Disease	ACT	NSW	NT	QLD	SA	TAS	VIC	WA	Australia
Bloodborne diseases									
Hepatitis B (newly acquired) ‡	0.0	0.3	0.0	0.7	0.5	0.8	0.5	1.3	0.5
Hepatitis B (unspecified) §	16.9	28.6	47.2	16.6	16.8	8.5	26.9	19.7	23.5
Hepatitis C (newly acquired) ‡	2.0	0.7	0.0	7.0	1.4	3.9	0.7	5.3	2.6
Hepatitis C (unspecified) §	30.7	53.8	60.2	42.6	19.1	47.1	27.5	41.6	40.6
Hepatitis D	0.0	0.3	0.0	0.1	1.2	0.0	0.3	0.0	0.3
Gastrointestinal diseases									
Botulism	0.0	0.1	0.0	0.0	0.2	0.0	0.0	0.0	0.0
Campylobacteriosis	140.8	NN	118.8	176.5	184.2	170.8	117.0	145.7	148.2
Cryptosporidiosis	2.0	7.0	27.7	13.0	6.5	9.3	15.1	4.4	10.1
Haemolytic uraemic syndrome (HUS)	0.0	0.0	0.0	0.0	0.5	0.0	0.1	0.0	0.0
Hepatitis A	0.0	1.1	0.0	0.6	3.5	0.0	2.3	0.2	1.3
Hepatitis E	0.0	0.2	0.0	0.1	0.0	0.0	0.1	0.0	0.1
Listeriosis	1.0	0.3	0.0	0.2	0.2	0.0	0.3	0.0	0.2
Paratyphoid	2.0	0.5	0.0	0.1	0.5	0.0	0.5	0.0	0.4
STEC ‖	0.0	0.7	0.0	0.6	19.8	0.8	0.8	3.4	2.3
Salmonellosis	32.7	43.1	223.0	72.8	95.0	74.2	50.2	87.2	61.5
Shigellosis	2.0	3.4	223.0	4.8	4.7	6.2	10.5	10.8	8.6
Typhoid Fever	0.0	0.5	0.0	0.2	0.2	0.0	0.6	1.6	0.5
Quarantinable diseases									
Avian influenza in humans (AIH)	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
Cholera	0.0	0.1	0.0	0.0	0.0	0.0	0.0	0.0	0.0
Middle East respiratory syndrome coronavirus (MERS-CoV)	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
Plague	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
Rabies	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
Severe acute respiratory syndrome (SARS)	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
Smallpox	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
Viral haemorrhagic fever (NEC)	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
Yellow fever	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0

Disease	ACT	NSW	NT	QLD	SA	TAS	VIC	WA	Australia
Sexually transmissible infections									
Chlamydial infection [¶,**]	365.8	370.6	1,103.6	453.5	319.4	262.0	190.6	422.4	348.1
Donovanosis	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
Gonococcal infection[**]	72.4	117.1	743.9	94.0	72.9	18.5	113.8	101.0	110.3
Syphilis < 2 years [**]	7.9	13.7	131.8	20.6	9.3	1.5	21.0	13.6	17.5
Syphilis > 2 years or unspecified duration [§,**]	2.0	7.1	35.8	5.6	4.9	0.0	12.0	7.5	8.0
Syphilis congenital	0.0	0.1	0.0	0.1	0.0	0.0	0.0	0.0	0.0
Vaccine preventable diseases									
Diphtheria	0.0	0.0	0.0	0.1	0.0	0.0	0.1	0.2	0.0
Haemophilus influenzae type b	0.0	0.2	1.6	0.1	0.0	0.0	0.0	0.0	0.1
Influenza (laboratory confirmed)	269.7	254.6	363.0	428.6	991.9	240.3	296.6	228.8	350.6
Measles	2.0	0.2	0.0	0.0	0.0	0.0	0.2	0.0	0.1
Mumps	0.0	1.9	43.9	11.4	0.7	1.5	0.6	0.8	3.7
Pertussis	22.8	56.0	52.1	27.8	84.1	11.6	33.5	64.4	45.9
Pneumococcal disease (invasive)	8.9	7.4	22.8	4.9	10.0	9.3	6.9	5.0	6.9
Poliovirus infection	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
Rotavirus	0.0	38.2	11.4	43.5	130.3	67.2	0.0	15.2	33.3
Rubella	0.0	0.1	0.0	0.0	0.0	0.0	0.0	0.0	0.0
Rubella congenital	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
Tetanus	0.0	0.0	0.0	0.1	0.0	0.0	0.0	0.0	0.0
Varicella zoster (chickenpox)	41.6	NN	27.7	15.0	31.3	10.0	22.6	27.8	22.2
Varicella zoster (shingles)	81.3	NN	154.6	45.7	154.6	80.4	43.2	79.4	64.9
Varicella zoster (unspecified)	48.6	NN	1.6	112.5	21.7	33.2	115.5	62.7	90.7
Vectorborne diseases									
Barmah Forest virus infection	0.0	1.1	3.3	5.3	0.2	0.0	0.1	2.5	1.8
Chikungunya virus infection	0.0	0.5	0.0	0.2	0.0	0.0	0.6	0.3	0.4
Dengue virus infection	15.9	4.5	4.9	3.0	1.4	3.1	2.5	5.2	3.7
Flavivirus infection (unspecified)	0.0	0.0	0.0	0.1	0.0	0.0	0.0	0.0	0.0
Japanese encephalitis virus infection	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
Malaria	2.0	0.6	0.0	2.3	0.2	1.5	1.2	1.1	1.1
Murray Valley encephalitis virus infection	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
Ross River virus infection	0.0	6.3	43.9	16.7	3.0	0.0	2.2	25.8	9.3

West Nile/Kunjin virus infection	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
Anthrax	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
Brucellosis	0.0	0.2	0.0	0.3	0.0	0.0	0.0	0.0	0.1
Lyssavirus infection (NEC)	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
Q fever	0.0	2.1	1.6	3.5	1.6	1.5	0.4	0.3	1.7
Leprosy	0.0	0.1	0.0	0.2	0.2	0.0	0.0	0.2	0.1
Tuberculosis	3.0	7.4	11.4	4.2	2.8	2.3	8.8	7.8	6.7

* The date of diagnosis is the onset date or where the date of onset was not known, the earliest of the specimen collection date, the notification date, or the notification receive date. For hepatitis B (unspecified), hepatitis C (unspecified), leprosy, syphilis (> 2 years or unspecified duration) and tuberculosis, the public health unit notification receive date was used.
† Rate per 100,000 of population. Annualisation Factor was 4.0
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NEC Not elsewhere classified.
NN Not notifiable.

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