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Original article

Medically-attended respiratory illnesses amongst pregnant women in Brisbane, Australia

Precious Rufus Ashiedu, Ross M Andrews, Stephen B Lambert, Lisa McHugh, Sallyanne LeGros-Wilson, Judith Zenchyson, Daniel Arnold, Clementine Shevell, Kerry-Ann F O'Grady

Abstract

There are limited community-based data on the burden of influenza and influenza-like illnesses during pregnancy to inform disease surveillance and control. We aimed to determine the incidence of medically-attended respiratory illnesses (MARI) in pregnant women and the proportion of women who are tested for respiratory pathogens at these visits. We conducted a nested retrospective cohort study of a non-random sample of women aged 18 years or over who had a live birth in maternity units in Brisbane, Queensland, from March 2012 to October 2014. The primary outcomes were self-reported doctor visits for MARI and laboratory investigations for respiratory pathogens. Descriptive analyses were performed. Among 1,202 participants, 222 (18.5%, 95%CI 16.3%–20.7%) self-reported MARI during their pregnancy. Of those with an MARI, 20.3% (45/222) self-reported a laboratory test was performed. We were able to confirm with health service providers that 46.7% (21/45) of tests were undertaken, responses from providers were not received for the remainder. Whilst one in 5 women in this population reported a MARI in pregnancy, only 3.7% (45/1,202) reported a clinical specimen had been arranged at the consultation and the ability to validate that self-report was problematic. As the focus on maternal immunisation increases, ascertainment of the aetiological agent causing MARI in this population will be required and efficient and reliable methods for obtaining these data at the community level need to be established. *Commun Dis Intell* 2015;39(3):E319–E322.

Keywords: influenza, incidence, pregnancy, laboratory confirmation

Introduction

Influenza is a global public health issue affecting both human life and economies in our ever-increasingly interconnected world.[1] Some groups in society are at higher risk of getting influenza; infants, the elderly, and people with certain chronic medical conditions.[2] Pregnant women are particularly susceptible to serious consequences from influenza infection,[3] particularly during pandemics.[4] Reasons for this are linked to the number of changes that occur to a woman's body during pregnancy, which may put pregnant women at higher risk of complications from influenza (e.g. changes to lung function, increased cardiac output, increased oxygen consumption, and impairments to the innate and adaptive immune response).[5–7] Consequently, the World Health Organization recommends that pregnant women be given the highest priority for influenza vaccine in countries that are initiating or expanding a seasonal influenza program.[8]

While data on hospitalisations for influenza during pregnancy are important, few studies have identified the incidence of medically-attended respiratory illnesses (MARI) during pregnancy at the primary health care level. Fewer still have reported the proportion of these women who have laboratory investigations performed to identify the aetiological agent. This lack of data limits the ability to accurately assess the burden of influenza during pregnancy and the likely effectiveness of interventions, such as influenza vaccination, aimed at preventing disease.

The objective of this study was to investigate the incidence of MARI during pregnancy in women in Brisbane, Queensland. A secondary objective was to identify the proportion of these women who had a laboratory test performed to identify an aetiological agent.

Methods

This study was part of an ongoing broader prospective cohort study (the FluMum Study)[9] investigating the effectiveness of influenza vaccine during pregnancy in preventing laboratory confirmed influenza in infants across 6 Australian capital cities. Women in Brisbane maternity units within 6 participating hospitals (public and/or private) were approached for recruitment by trained research staff, prior to hospital discharge. The selected hospitals included the 2 large tertiary public maternity units in inner Brisbane and 4 units (3 private, 1 public) in suburbs more than 10 km from the city centre.

Women were eligible for inclusion if they were: aged 18 years or over at the time of written informed consent, willing and able to adhere to all protocol requirements, had sufficient verbal English to permit questionnaire completion, and had given birth to a live infant. Women were excluded if they planned to move overseas before the infant reached 6 months of age.

At enrolment, a detailed questionnaire was completed that collected data on self-reported influenza and pertussis vaccination, self-reported maternal medical and obstetric history and socio-demographic indicators. MARIs were determined by asking the participant whether, during her pregnancy, did she ever have a respiratory illness with symptoms like fever, chills, cough, aches and pains, that caused her to see a doctor. If yes, participants were asked whether a test was performed at the visit (nose, throat or blood specimen) but they were not specifically asked if the test was for influenza or other respiratory pathogens. If a test was reported, this was validated by contacting the relevant healthcare provider. Three to five attempts were made by telephone, email and/or facsimile to confirm the test and obtain a diagnosis. Similar attempts were made to confirm self-report of influenza vaccination during and in the 12 months prior to the pregnancy.

We analysed data collected on enrolment from the 1,202 women recruited at the Brisbane site for the years 2012–2014. The primary endpoint was participant-reported attendance at a medical practitioner for a respiratory illness during pregnancy. The secondary endpoints were a) the participant reported clinical specimens collected for laboratory investigations at these visits, and b) the healthcare provider confirmation of those laboratory investigations.

The study was approved by the Royal Brisbane and Women's Hospital Human Research Ethics Committee (HREC/12/QRBW/85), the Mater Mothers Human Research Ethics Committee (2012–16), and The University of Queensland Medical Research Ethics Committee (2012000180).

Data analysis

The primary analysis was the proportion of women who reported an MARI during the pregnancy and presented with its 95% confidence interval (CI). Secondary analyses were the proportion of women who reported a test done and of those, the proportion that were confirmed by the health service provider. Descriptive analyses were performed using Stata SE V12 (StataCorp, Texas, USA), including producing proportions and means with 95% CIs, and medians with interquartile ranges.

Results

Participant characteristics

Between March 2012 and October 2014, 1,713 women were screened and 1,202 (70.2%) were enrolled into the FluMum study in Brisbane. The mean age was 31.5 years (95% CI 31.3–31.8), 2% (23/1,196) identified as Aboriginal and/or Torres Strait Islander and 698 (58.1%) were recruited through public hospitals. More than half of the women for whom data were available (947/1,194, 79%) were in paid employment during their pregnancy; the majority of these being in full-time employment (568, 60%). Approximately half (515/1,035, 49%) had completed a university degree or higher and approximately 8% of women (93/1,195) had smoked during their pregnancy. Of the 1,196 women for whom data were available, 340 (28.4%) self-reported a pre-existing health condition such as heart disease, respiratory conditions, immunosuppressive conditions, cancer or diabetes, or a history of pneumonia requiring hospitalisation in the past 12 months.

Medically attended respiratory illnesses during pregnancy

Overall, 222 of 1,202 women (18.5%, 95% CI, 16.3%–20.7%) reported a respiratory illness that caused them to visit a health practitioner during their pregnancy. Of these, 39 women reported 2 episodes, eight reported 3 episodes, and two reported 4 episodes. Forty-five (20.3%, 95% CI 15.0%–25.6%) of the 222 women with a MARI reported that a clinical specimen was collected at the time (nose swab n=15, and/or throat swab n=10, and/or a blood test n=35). No tests were reported in episodes subsequent to the initial presentation. Seventy-two (21.2%) of the 340 women with a self-reported pre-existing health condition reported a MARI during their pregnancy and 22 (30.6%) of these women reported they had a test done.

Despite multiple attempts to secure information from providers, confirmation of the test request was obtained in 21 (46.7%) episodes. Of the confirmed episodes in which a blood test was taken (n=13), 7 providers reported the bloods were not tested for respiratory viruses. This information was not provided for the remaining 6 episodes.

Discussion

Influenza is an important cause of morbidity during pregnancy[5] but the lack of systematic surveillance for disease during pregnancy at the community level limits the ability to reliably estimate the burden of disease and the effectiveness

of interventions, particularly vaccination. This study had identified that almost one in 5 women will present to a health care provider during their pregnancy for a respiratory illness. Collection of clinical specimens during the visit is reported but is difficult to confirm. This leads to doubts about the reliability of those reports with respect to testing for respiratory pathogens.

There are limited comparable data with which to compare the MARI incidence in our study given differences in study designs, study populations, and the case definitions used for respiratory illness and/or influenza-like illness. In a randomised controlled trial of inactivated influenza vaccine in HIV-negative pregnant women in South Africa that employed active surveillance for respiratory illnesses,[10] 17.2% (95% CI 14.9–19.6) of women in the control group reported an influenza-like illness in the period from the time of vaccination to up to 24 weeks post-birth of the infant; 65.2% (95% CI 62.2–68.1) reported having any respiratory illness.[10] The incidence of illness during pregnancy only was not reported. In a cohort study that used administrative datasets, Lindsay et al[11] reported 8% of 8,323 healthy (no underlying chronic conditions) pregnant and post-partum women in Washington, United States of America, experienced an influenza-like episode that resulted in health-care use (total person weeks of observation = 301,778).[11]

Blood was the predominant specimen women reported as being collected. This is unusual given there are few clinical indications for serology in acute, uncomplicated respiratory illness at the community level.[12] For those who reported an MARI episode, we asked "thinking now about the 1st episode of respiratory illness during your pregnancy that caused you to see a doctor, can you tell me the tests done?" We then sought further information on who had done the test, the diagnosis, gestational age at the time of the test and the treating doctors contact details. We also sought similar information for each subsequent MARI. Whilst we made reference to episodes of respiratory illness, it is possible that there may have been some misunderstanding or uncertainty for participants such that the information provided may not have directly related to specimens collected at a MARI presentation that were specifically for a respiratory diagnosis. This is partially supported by the number of reports from providers stating bloods were not tested for respiratory viruses.

The difficulties encountered in confirming the test with the health care provider is problematic for influenza surveillance and control in this population and for estimating the effectiveness of maternal influenza and/or pertussis vaccination during pregnancy at the population level, particularly in non-pandemic periods. With the exception of hospitalised cases, there are limited population-based data on both the burden of influenza and the effectiveness of influenza vaccination during pregnancy. This lack of data is recognised as a contributing factor towards determining the real risk of influenza associated with pregnancy.[13, 14] Such data would enhance public health policy recommendations and facilitate discussions between health care providers and pregnant women on the risks of influenza and why the vaccine is recommended in pregnancy.

This study has some limitations that necessitate caution in interpreting the findings. The FluMum study[9] population is derived from English speaking women giving birth to a live infant in metropolitan maternity units and may not be representative of non-English speaking women and those with high risk pregnancies and adverse pregnancy outcomes, nor of women in rural and remote areas where access to health care and the viability of specimen collection, transport, and processing for respiratory illnesses may differ. While the proportion of Aboriginal and Torres Strait Islander women enrolled in the study was low (1.7%), it does approximate the 2011 estimated resident Indigenous population of the greater Brisbane region (2.0%).[15] Further possible selection and measurement biases that may be affecting our findings are potential differences between vaccinated and unvaccinated women that would influence their decision to participate in the study and their recall of MARI. Finally, as data were collected retrospectively, misclassification due to poor recall may have occurred resulting in an over– or under-estimation of MARI frequency and of testing for MARI.

MARI during pregnancy is not uncommon yet the investigation of these illnesses to determine an aetiological agent is infrequent. While laboratory investigation of all community-based MARI during pregnancy may be unwarranted clinically, sentinel surveillance of these events in sites representative of Australian pregnant women would be a useful contribution to further understanding the risk and outcomes of influenza during pregnancy. Such surveillance would provide more comprehensive estimates of influenza vaccine effectiveness to inform public health policy.

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Author contributions

PFA analysed the data and wrote the first draft of the manuscript. RMA, SBL and KFO are chief investigators on the overall FluMum study. RMA and KFO devised this current paper & KFO prepared the final draft. LMcH, SLW, JZ, DA & CS all contributed substantially to study implementation and the preparation of data for analysis. All authors had full access to the study data and contributed to and approved the final manuscript.

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Notification and management of congenital syphilis in the Northern Territory 2009 to 2014

Charlie McLeod, Jiunn-Yih Su, Joshua R Francis, Alice Ishwar, Nathan Ryder

Abstract

Objective: To determine whether cases of congenital syphilis in the Northern Territory between 2009 and 2014 were correctly notified based on probable or confirmed case criteria stipulated by the Communicable Diseases Network Australia (CDNA).

Methods: Pregnant women with positive syphilis serology defined as reactive treponemal test and rapid plasma reagin titre ≥1:8 were identified from the Northern Territory Syphilis Register Information System. Risk classification was performed based on local guidelines, and CDNA criteria for probable/confirmed cases of congenital syphilis were applied to determine whether cases were appropriately notified.

Results: Thirty-four cases of positive maternal syphilis serology in pregnancy were identified from 31 women; all were Indigenous. Twenty-one cases fulfilled criteria for probable congenital syphilis; 1 case was formally notified to the Centre for Disease Control. Twenty cases (95%) fulfilling CDNA criteria for probable congenital syphilis were not notified over the study period.

Conclusions: Application of standard case definitions significantly increases the rate of congenital syphilis cases in the Northern Territory. Improved education regarding CDNA criteria for notification of congenital syphilis is necessary for clinicians and public health staff. Emerging evidence has supported the recent simplification of CDNA criteria for notification of congenital syphilis, effective 1 July 2015. *Commun Dis Intell* 2015;39(3):E323–E328.

Keywords: syphilis, congenital syphilis, notification(s), paediatric, mother-to-child transmission, pregnancy.

Introduction

Mother-to-child transmission (MTCT) of *Treponema pallidum* during pregnancy results in congenital syphilis.[1] Globally, 1.36 million pregnant women are estimated to have active syphilis per annum.[2] The World Health Organization (WHO) regards congenital syphilis as a public health priority, and in 2007 a campaign for its global elimination was launched.[3]

The Syphilis Register Information System (SRIS) is a centralised patient database in the Northern Territory containing all positive syphilis test results and treatment histories. This resource is managed by nursing staff and overseen by a sexual health physician at the Centre for Disease Control (CDC), who are available to provide information to clinicians to help inform treatment decisions.

In the Northern Territory, risk classification and management of syphilis in pregnancy and the neonatal period is directed by the *Guidelines for the Investigation and Treatment of Infants at Risk of Congenital Syphilis in the Northern Territory*.[4] This guideline recommends clinicians notify all low and high risk cases to the CDC. This includes all mothers with rapid plasma reagin (RPR) titres ≥1:8 during pregnancy for whom re-treatment is recommended even if previous adequate syphilotherapy has occurred. Under the *Notifiable Diseases Act* the CDC Northern Territory maintains a database of probable or confirmed congenital syphilis cases as defined by the Communicable Diseases Network Australia (CDNA).[5] These case definitions are also used by clinicians for the purpose of making a formal diagnosis of congenital syphilis. Table 1 shows the correlation of the Northern Territory risk category with CDNA case definitions.

The Northern Territory is a jurisdiction that spans 1.4 million km,[2] with a population of 240,000 people, 30% of whom are Indigenous.[6] Rates of syphilis seropositivity in the Northern Territory are estimated in the order of 1 in 3 Indigenous persons by the age of 40 years based on data derived from a comparable population in the Kimberley region of Western Australia.[7] Until recently, the notification rate of infectious syphilis in the Northern Territory was by far the highest among Australian states and territories;[4] however this has declined substantially from 35.1 per 100,000 in 2008 to 9.1 per 100,000 in 2013.[8] A comparably large reduction in congenital syphilis notifications has also occurred, with 4 cases notified between 2009 and 2013 (3 in 2009 and 1 in 2013).[9]

Table 1: Correlation of the Northern Territory risk category with the Communicable Diseases Network Australia case definitions

NT risk category	Risk category inclusion criteria	CDNA classification
No risk	Mothers who have never had syphilis OR Mothers with adequately treated* syphilis prior to pregnancy AND All rapid plasma reagins in pregnancy <1:8 AND No suspicion of late infection	N/A
Low risk (notify to Communicable Disease Centre)	Mother adequately treated* for syphilis in pregnancy	May meet criteria for probable/confirmed congenital syphilis
High risk (notify to Communicable Disease Centre)	Mother seropositive during pregnancy AND ≥1 of (i) Clinical signs (ii) Inadequate maternal treatment (iii) Child's RPR ≥4 times maternal titre or (iv) Maternal re-infection likely	Probable or confirmed congenital syphilis

* Adequate treatment requires adequate syphilotherapy (appropriate penicillin regime) and adequate serological response (Early disease: 2-titre or 4-fold decline in rapid plasma reagin; Late disease: If no rapid plasma reagin decline then maintenance of a low stable titre <1:8 and not increasing more than 1 titre).

Low numbers of notifications of congenital syphilis in the Northern Territory in recent years may be the result of low incidence, or cases not being appropriately recognised and/or notified. We conducted an audit of pregnant women with RPR titres ≥1:8 during pregnancy in the Northern Territory between 1 January 2009 and 20 May 2014 in order to capture women posing the highest risk for mother-to-child transmission. The primary objective was to determine whether congenital syphilis cases in the Northern Territory between 2009 and 2014 were correctly notified based on probable or confirmed case criteria stipulated by the CDNA. Additionally, we aimed to characterise the reasons for missed notifications and to determine whether at-risk infants received treatment.

Methods

Selection of participants and extraction of data

Pregnant women with RPR titres ≥1:8 were identified from the Northern Territory SRIS over the study period.[8] Attempts were made to link maternal and newborn patient files in the SRIS and hospital patient databases. Data were obtained from the SRIS and electronically available hospital patient records (including discharge and clinic letters, radiology and pathology results).

Criteria for risk classification, disease notification, and adequate treatment

Risk classification for MTCT of syphilis in pregnancy into no risk, low risk or high risk groups was based on criteria outlined in the *Guidelines for the Investigation and Treatment of Infants at Risk of Congenital Syphilis in the Northern Territory*.[10] Classification was performed by the chief investigator and counter-checked by co-investigators JYS, AI and NR.

Adequate treatment for maternal syphilis was based on local guidelines.[4] For mothers with late syphilis, 3 benzathine penicillin G injections are recommended at 7-day intervals, although up to 14 days between injections was considered acceptable in our cohort based on local and international expert opinion.[11] Serofast status was assigned when an individual's serum showed little or no change in antibody titres (<2-titres or 4-fold drop) despite adequate treatment.

Postnatal treatment for low risk infants comprised 1 dose of intramuscular benzathine penicillin G (37.5 mg/kg). Adequate treatment for high risk infants was defined as benzyl penicillin 50 mg/kg per dose delivered twice daily intravenously for 10 days, which is considered a neuro-penetrating regime.[10] Appropriate follow-up for all low and high risk infants required clinical review and syphilis serology at birth, and 3 and 6 months of age.

Criteria for notification of probable or confirmed congenital syphilis cases were based on CDNA criteria (2010).[5]

Statistical analysis and ethics approval

Descriptive statistical analyses were performed using Microsoft Excel version 14.1.0 (2010). Approval for the study was obtained from the Human Research

Ethics Committee of the Northern Territory Department of Health and Menzies School of Heath Research (HREC-2014-2263).

Results

There were 34 instances of positive maternal syphilis serology (RPR titre ≥1:8) from 31 women over the study period; all were Indigenous. The median maternal age at delivery was 27 years (range 16 to 43 years). The women resided in a wide range of locations; 8 from Katherine, 13 from Central Australia, 2 from East Arnhem, 6 from urban Darwin and 2 from interstate. It was possible to link maternal and neonatal files in the register in 21 instances (62%). For the remaining 13 neonates whose identities were unknown, a date of birth was recorded for seven. Despite an inability to link maternal and neonatal files in a significant proportion of cases, risk classification was possible in all instances using either maternal or neonatal data.

Maternal treatment and risk classification

Risk classification is depicted in the Figure. Overall, 1 woman met criteria for no risk of MTCT, 12 were low risk (35%) and 21 (62%) were high risk. The maternal risk classification recorded in the SRIS was incorrect in 15 (44%) of the 34 pregnancies. Twenty-one of the 34 pregnancies were not adequately treated or did not demonstrate adequate treatment response during pregnancy. Of these, 8 women were not treated (38%), 5 women were not treated in time (24%; 1 case was notified), and 8 women did not meet serological criteria for adequate response (38%).

Congenital syphilis notifications

Of the audited population, there were 21 neonates who met CDNA criteria for probable congenital syphilis over the study period, and none for the confirmed category (Table 2). All 21 cases also met criteria for high-risk classification based on local Northern Territory guidelines, yet only 1 case was formally notified (5%).

Of the 20 cases that were not notified but met CDNA criteria for probable congenital syphilis, 9 cases were incorrectly classified on the SRIS as no risk (n=5) or low risk (n=4), and in 2 cases, risk classification was not recorded. Nine neonates were accurately classified on the SRIS as high risk, yet not notified. One of the 20 cases not notified was delivered interstate.

Neonatal management

Overall, 14 (42%) of the 26 low and high-risk neonates for whom information was available were adequately treated at birth (information was not available in 7 instances).

Only 17 of the 33 low and high-risk neonates underwent the required serological testing for

Figure: Audited risk classification for mother-to-child-transmission of syphilis

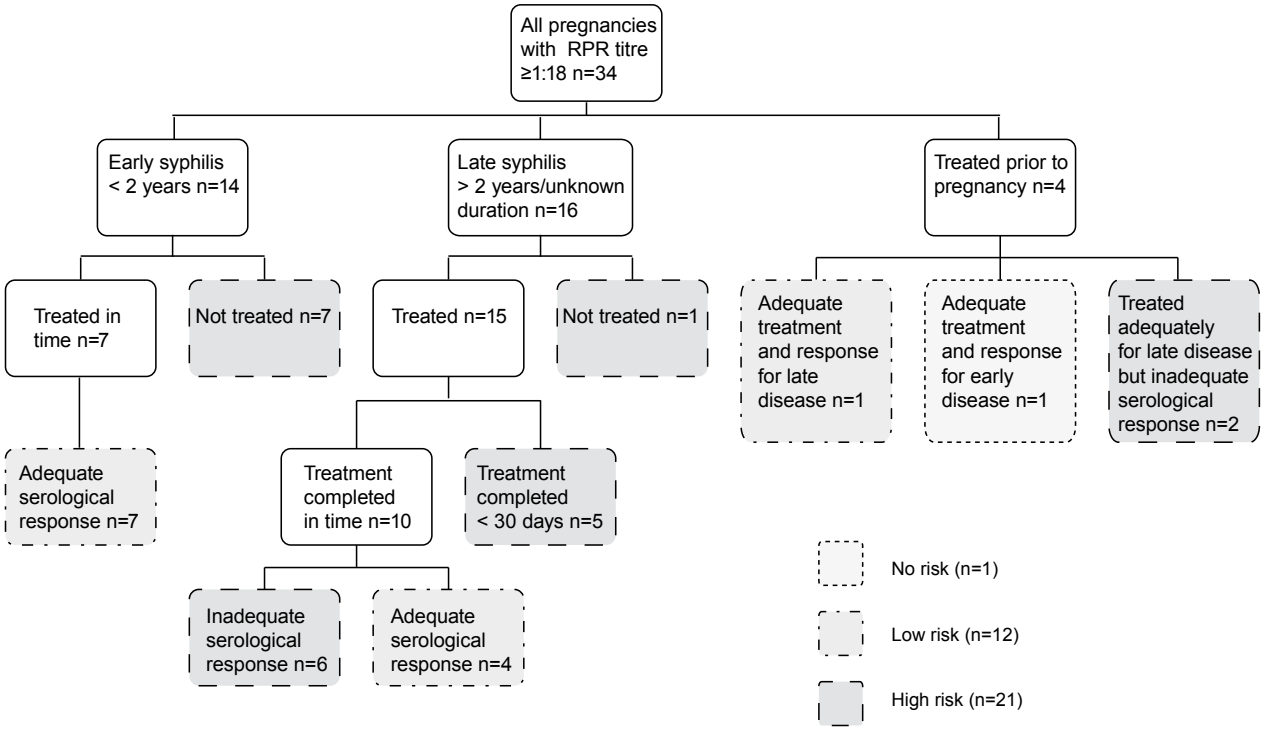

Table 2: Reasons probable congenital syphilis cases were not notified based on Communicable Diseases Network Australia case definitions

Cases at risk of mother-to-child-transmission of syphilis (n=34)	Total	Notifications
Cases not requiring notification	13	–
Cases requiring notification	21	1
Indications for notification		
Maternal factors– early disease		
Not treated	7	–
Maternal factors– late disease		
Not treated	1	–
Treatment completed <30 days	5	1
Inadequate serological response (no 2-titre/4-fold drop)	8	–

syphilis at birth (52%). Eight of the 33 infants (24%) had follow-up serology performed (ranging from 11 days post-delivery to 11 months of age). In 7 instances, follow-up serology was negative, while in 1 case serology was positive, but less than 4-fold higher than the maternal results.

There were no neonates who were notified based on signs detected on physical examination or radiography alone. One pregnant woman with early syphilis who was adequately treated, suffered a foetal death in utero at 19 weeks. Ureaplasma was detected on placental culture. This foetus did not meet CDNA case criteria for probable congenital syphilis.

Discussion

Our study found that the overwhelming majority of audited cases (20/21, 95%) that met the criteria for probable congenital syphilis according to CDNA criteria, were not notified during the study period. As there were only 4 cases of congenital syphilis notified in the Northern Territory during this period overall, notifying the additional 20 cases identified during this audit would produce a 6-fold increase in the congenital syphilis rate. However, this may still be an underestimate, as only women with titres ≥1:8 were included in this audit. The other 3 notified cases during the study period, who were not included in our cohort, occurred in women who had RPR titres < 1:8 in pregnancy, who were classified as high risk due to inadequate previous treatment for late syphilis.

Treatment and follow-up did not comply with the Northern Territory and national guidelines in the majority of cases. Firstly, nearly two-thirds of women were not adequately treated for syphilis in pregnancy. Secondly, only half of the neonates received the recommended prophylactic treatment at birth. Lastly, few neonates received appropriate follow-up.

Late and/or inconsistent attendance at antenatal care may have contributed to the failure to adequately treat prior to delivery. Indigenous women, particularly those living in remote locations, are less likely to present for antenatal care in the first trimester.[12] However, while this may account for some missed (n=8) and late maternal treatment (n=5) it cannot account for either the failure to correctly treat neonates at birth or the failure to notify the cases. Furthermore, as most untreated cases were also not classified correctly on the SRIS, it seems unlikely that failure to present for antenatal care accounts for a large proportion of inadequately managed cases.

The diagnosis of syphilis reinfection in a previously treated person is open to a level of subjectivity. The intrinsic variability of RPR results between batches, operators and laboratories is an issue that merits attention. It is not uncommon to see different serological values for the same woman recorded on the same day at different laboratories. These 'outlier' results create a problem for clinicians and syphilis register staff responsible for interpreting results, especially if they support CDNA case criteria warranting notification. Six of the 8 untreated cases described in this paper were deemed by treating clinicians not to require treatment on the basis of 'outlier' laboratory results. However, as the CDNA criteria for adequate previous treatment are stringent we applied the same level of rigidity to the criteria for the diagnosis of new syphilis cases, and all women with a 2-titre increase in RPR were defined as cases.

Eight cases in our study that were not notified met CDNA criteria for notification due to inadequate maternal serological response to treatment. Interestingly, a recent retrospective review[13] of 166 pregnant women with syphilis (>18 weeks) who received adequate syphiliotherapy, found failure to achieve a 4-titre serological drop was more

a reflection of treatment timing than treatment failure ($P<0.001$). Inadequate serological response was significantly correlated with late syphilis or syphilis of unknown duration and older maternal age. The findings of this landmark study and recommendations of the early draft of this paper provided to the working group responsible for the revision of national case definitions for congenital syphilis, have contributed to the release of the new national case definitions on 1 July 2015. In this new case definition, inadequate maternal serological response has been removed as constituting laboratory evidence for probable congenital syphilis. This not only simplifies the Australian national case definitions, but also brings them more in line with those currently used in the United States of America.[14] The 8 cases described above would no longer meet criteria for probable congenital syphilis based on the new CDNA case definitions.

Clinical practice did appear to diverge from local management guidelines in a significant proportion of our cases, and CDNA criteria for notification are not currently being stringently applied. This is alarming, given congenital syphilis represents a significant public health threat in the Northern Territory, and notification of cases is vital in order to ensure an appropriate public health response. Transmission is easily prevented in a cost-effective manner when appropriately identified and treated.[15] Improved education about management of congenital syphilis and CDNA criteria for notification of probable or confirmed cases is needed, both for clinicians involved in patient care and relevant public health and CDC staff involved in local Northern Territory and national surveillance. This is likely to be true for other Australian states with similar epidemiology of infectious syphilis.

A further issue that merits review is the utility of re-treating all women with RPR titres ≥1:8 in pregnancy including those who have received adequate syphilotherapy prior to pregnancy. Whilst this approach helps ensure that all neonates with potential untreated congenital syphilis receive prophylactic treatment, the incremental benefit of re-treating serofast patients is likely to be minimal, with one study suggesting only 25% demonstrate serological response with re-treatment.[7] This calls into question the cost-effectiveness of this strategy.

This study has a number of limitations, including the fact that information was obtained from electronic records only, and a more extensive chart review was not performed owing to difficulties in accessing records from remote centres. Outcomes of post-natal follow-up were also not assessed in this audit, and so it is unknown whether adverse outcomes occurred for cases that met criteria for probable congenital syphilis but were not notified. However, the Northern Territory CDC is currently conducting another audit on the outcomes of cases of congenital syphilis, which will shed some light on this aspect. The likely cause of foetal death in utero at 19 weeks in the mother with early stage, adequately treated syphilis was ureaplasma chorioamnionits, and not syphilis, although an autopsy was not performed. In light of the findings of this audit, we have liaised with the Royal Darwin Hospital Paediatric unit to ensure that clinical follow-up for missed cases occurs, where possible.

There is a need for prospective longitudinal studies examining long-term outcomes for neonates at risk of MTCT of syphilis, detailing their initial management, follow-up and clinical course. This would allow an improved understanding of clearly at risk neonates, and facilitate improved risk categorisation and management.

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Short report

Pertussis immunisation in pregnancy: a summary of funded Australian state and territory programs

Frank H Beard

Abstract

The Australian Immunisation Handbook, 10th edition now recommends pertussis vaccination during pregnancy as the preferred option for protecting vulnerable young infants. Jurisdictionally funded pertussis immunisation programs for pregnant women were progressively introduced in all Australian states and territories between August 2014 and June 2015. A meeting convened by the National Centre for Immunisation Research and Surveillance of Vaccine Preventable Diseases was held on 31 May 2015 to share information regarding jurisdictional policies and program implementation. This report of that meeting provides the first published comparison of these jurisdictional programs, which are of a broadly similar nature but with important differences. Monitoring and evaluation of the uptake, safety and impact of the current programs in Australia will be important to inform future policy decisions. *Commun Dis Intell* 2015;39(3):E329–E336.

Keywords; immunisation programs; pertussis

Introduction

In 2013, the 10th edition of *The Australian Immunisation Handbook* included for the first time, the option of vaccinating pregnant women with pertussis vaccine in the 3rd trimester of pregnancy, rather than pre– or post-partum.[1] These 3 options were presented as equivalent in terms of protecting infants, due to the absence of sufficient evidence to support any clear preference. Cocooning (vaccinating close contacts of infants, including parents, to reduce the likelihood of exposure) has been recommended in Australia since 2003,[2] and some states and territories introduced funded cocoon programs in response to the recent pertussis epidemic. However, cocooning provides indirect protection and is only moderately effective.[3] Following the publication of evidence showing that pertussis vaccination during pregnancy is both highly effective in preventing infant disease[4,5] and safe,[6–8] *The Australian Immunisation Handbook* was updated in March 2015 to clearly recommend pertussis vaccination during pregnancy as the preferred option, recommending optimal timing between 28 and 32 weeks gestation but that the vaccine can be given at any time during the 3rd trimester up to delivery.[9]

Jurisdictionally funded pertussis immunisation programs for pregnant women have been progressively introduced in all Australian states and territories between August 2014 and June 2015. These programs are broadly similar in nature but with some differences in terms of policy and implementation.

A meeting, convened by the National Centre for Immunisation Research and Surveillance of Vaccine Preventable Diseases (NCIRS), was held on 31 May 2015 immediately prior to the Communicable Disease Control Conference in Brisbane, aiming to share information regarding jurisdictional policies and program implementation, along with plans for evaluation of uptake, adverse events following immunisation, and disease impact. This meeting was attended by representatives from the Australian Government Department of Health and all 8 Australian states and territories except the Australian Capital Territory. This report summarises the key outcomes of this meeting and provides the first published summary of the commonalities and differences across jurisdictional programs and plans for evaluation.

Meeting outcomes

Program details

Commencement dates and implementation details for funded jurisdictional pertussis immunisation programs for pregnant women, along with whether any cocoon program is to be run simultaneously, are presented in the Table. Queensland was the first jurisdiction to introduce a program, commencing in August 2014, with all other jurisdictional programs introduced between March and June 2015.

All jurisdictions recommend vaccination from 28 weeks gestation and most note that 28–32 weeks is ideal. At the time of the meeting, Queensland

recommended vaccination only where no pertussis vaccine dose had been received in the last 5 years. However this was subsequently amended to accord with the updated *Australian Immunisation Handbook* recommendations (personal communication Scott Brown, Acting Manager, Immunisation Program, Department of Health, Queensland, 29 July 2015). Only 2 jurisdictions (Victoria and the Northern Territory) are funding a cocoon program in addition to their program for pregnant women.

Implementation

All jurisdictions provide vaccine via general practitioners and hospital antenatal clinics, with some also utilising Aboriginal medical services, local councils, community health centres, and obstetricians.

Evaluation

Coverage assessment

The most common plan for assessing vaccination coverage was through data from the relevant jurisdictional perinatal data collection (PDC). Vaccination during pregnancy will be captured on the Victorian and Queensland PDCs from July 2015 and on the New South Wales PDC at a state-wide level from January 2016. Some other jurisdictions reported attempts to organise inclusion on their PDC but a number of challenges were identified in achieving this. A range of alternative methods of coverage assessment were also planned, with the Northern Territory to use its own whole-of-life immunisation register and Western Australia an annual survey of a random sample of around 400 recently-delivered mothers.

Queensland advised an interim estimate of 40%–50% coverage as of May 2015, based on the number of births and consent forms returned centrally, and the use of a different brand of vaccine to that used in the adolescent school-based program allowing differentiation. Western Australia advised an interim estimate of around 55% coverage, based on the number of births in May 2015 and the number of forms returned by immunisation providers documenting administration of vaccine to pregnant women. Western Australia also reported that influenza vaccine coverage during pregnancy appeared to have improved as a result of the pertussis program, with the vaccines often co-administered.

Vaccine safety

Most jurisdictions reported that they will rely on existing (passive) surveillance systems for the reporting of adverse events following immunisation (AEFI). These involve reporting of AEFI by immunisation providers, and sometimes patients, with subsequent follow-up by public health agencies.[10] In New South Wales this will be supplemented by emergency department syndromic surveillance while Western Australia will use active surveillance for AEFI with expansion of an SMS system used for AEFI monitoring for influenza vaccination during pregnancy.

Disease impact/vaccine effectiveness

Queensland, New South Wales and Western Australia reported the most advanced plans for evaluation of their respective programs. Queensland will evaluate vaccine effectiveness via a cohort study, linking data from their state-based immunisation register (Vaccination Information and Vaccination Administration System) with notifications database, perinatal data collection data, and birth registry data, while New South Wales will evaluate via a case-control study based on notified infant cases. Western Australia plans to expand its existing cohort study of influenza vaccine effectiveness during pregnancy to assess pertussis vaccine effectiveness, via linked midwives data collection, hospitalisation, notification and emergency department data, and data on vaccination in pregnancy reported by immunisation providers.

Conclusions

Currently, all available evidence supports vaccination during pregnancy as the best option for protecting vulnerable young infants from pertussis. Australia has high rates of pertussis, with high levels of hospitalisation (and occasional deaths) in young infants.[11] Australian states and territories have taken the lead in implementing pertussis immunisation programs for pregnant women on the basis of this evidence and updated *Australian Immunisation Handbook* recommendations.

With broadly similar pertussis immunisation programs for pregnant women now in place and funded by all jurisdictions, for the first time in recent history Australia has immunisation programs that are implemented across the entire country that universally target a particular population but are outside the National Immunisation Program (NIP). This situation is likely to be of questionable sustainability. Since 2005 vaccines have been required to go through a standardised process of application to and assessment by the Pharmaceutical Benefits Advisory Committee (PBAC) for consideration of suitability and cost-effectiveness for funding under the NIP.[12,13] It is currently unclear whether any vaccine manufacturer intends to submit an application

to the PBAC in regard to pertussis immunisation during pregnancy, and unclear what the outcome of such an application, if it eventuates, would be. Pertussis immunisation for pregnant women has been funded in national immunisation programs in the United Kingdom (though in the context of a temporary program with review after 5 years)[14] and in New Zealand,[15] and is recommended nationally by the Advisory Committee for Immunization Practices in the United States[16] and 'encouraged' by the Public Health Agency of Canada.[17] Monitoring and evaluation of the uptake, safety and impact of the current program arrangements in Australia will be important to inform future policy decisions.

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Table: Funded pregnancy/cocoon pertussis immunisation program implementation, by Australian states and territories (information as of 31 May 2015)

State or territory	Australian Capital Territory*	New South Wales	Northern Territory	Queensland	South Australia	Tasmania	Victoria	Western Australia
Program dates and details								
Start date	April 2015	April 2015	April 2015†	August 2014	March 2015	June 2015	June 2015	April 2015
Vaccine	Boostrix® and Adacel®	Boostrix®	Boostrix®	Adacel® (cf adolescent program using Boostrix®)	Adacel® (cf adolescent program using Boostrix®)	Adacel® (cf adolescent program using Boostrix®)	Boostrix®	Boostrix® (cf adolescent program using Adacel®)
Target group in terms of gestational age	From 28 weeks gestation in each pregnancy (recommended at 28 weeks gestation or as soon as possible after that)	From 28 weeks gestation in each pregnancy (ideally 28–32 weeks)	From 28 weeks gestation in each pregnancy, or as soon as possible after delivery	From 28 weeks gestation, if have not had a pertussis containing vaccine in the last 5 years‡	From 28 weeks gestation in each pregnancy (ideally 28–32 weeks)	From 28 weeks gestation in each pregnancy (ideally 28–32 weeks)	From 28 weeks gestation in each pregnancy (ideally 28–32 weeks) or as soon as possible after delivery	From 28 weeks gestation in each pregnancy (ideally 28–32 weeks)
Funded cocoon program?	No	No	Yes (since 2008 – currently targets fathers/carers in household of an infant under the age of 7 months – can be given from time expectant mother reaches 28 weeks gestation)	No	No	No	Yes (parents and guardians of infants up to 6 months of age and born on or after 1 June 2015, and partners of women who are at least 28 weeks pregnant, if they have not received a pertussis booster in the last 10 years)	No
Implementation								
Providers	GPs, antenatal clinics.	GPs, AMSs, antenatal clinics	All providers, majority in community health centres. Also GPs and antenatal clinics	Mainly GPs, also antenatal clinics	Mainly GPs, also antenatal clinics and councils	GPs mainly, some in antenatal clinics	All providers – GPs, antenatal clinics and possibly councils	GPs, antenatal clinics and obstetricians

Table (cont'd): Funded pregnancy/cocoon pertussis immunisation program implementation, by Australian states and territories (information as of 31 May 2015)

State or territory	Australian Capital Territory*	New South Wales	Northern Territory	Queensland	South Australia	Tasmania	Victoria	Western Australia
Evaluation								
Coverage assessment		Via perinatal data collection (will be collected state-wide by 1 January 2016)	Northern Territory immunisation register	Use of Adacel® allows differentiation from adolescent program, also consent forms returned centrally Coverage estimated at 40%–50% Vaccination status will be in perinatal data collection from 1 July 2015	Not currently in perinatal data collection		Via perinatal data collection	Annual survey of a random sample of ~400 recently-delivered mothers (baseline coverage 5%) Working to get pertussis vaccination into perinatal data collection Coverage estimated at around 55%
Vaccine safety		Emergency department syndromic surveillance	Usual way	Follow-up of reported adverse events following pertussis-containing vaccine in women of child-bearing age; reviewed by expert advisory group	Usual way	Usual way	Usual way	Via SMS back system – expansion of existing system used for monitoring adverse events following influenza vaccination during pregnancy
Formal evaluation of impact/ vaccine effectiveness		Case-control study of vaccine effectiveness, based on notified infant cases	Planning evaluation	Cohort study of vaccine effectiveness – linkage of data from state-based immunisation register with notifications database, perinatal data collection, and birth registry data	None planned, but will capture maternal vaccination status in all infant pertussis cases	None planned, but will capture maternal vaccination status in all infant pertussis cases	Scoping options	Aim to expand cohort study of influenza vaccine effectiveness during pregnancy to assess pertussis vaccine effectiveness – linkage of midwives data collection, hospitalisation, notification and emergency department data

Table (cont'd): Funded pregnancy/cocoon pertussis immunisation program implementation, by Australian states and territories (information as of 31 May 2015)

State or territory	Australian Capital Territory*	New South Wales	Northern Territory	Queensland	South Australia	Tasmania	Victoria	Western Australia
Relevant website links for jurisdictional programs								
	Antenatal Pertussis Vaccination Program General Practitioners & Immunisation Providers Q&A[18] Protecting your newborn from whooping cough Q&A[19]	NSW Health news[20] NSW Health Immunisation programs[21] NSW Pertussis Control Program 2015[22]	Immunisation[23] Pertussis (Whooping cough)[24] Adult and Special Groups Vaccination Schedule[25]	Pregnant and breastfeeding women[26] Whooping cough vaccine program for pregnant women[27] Whooping cough vaccine program for pregnant women information sheet[28]	Whooping cough vaccine in pregnancy program29 Diphtheria, tetanus and whooping cough combination vaccines[30]	Free whooping cough vaccine for pregnant women[31] Department of Health and Human Services Bulleting board[32]	Q&A for health professionals - Parent's whooping cough vaccine program[33] Better Health Channel Whooping Cough[34]	Healthy WA; Adult immunisation schedule[35] Healthy WA; Pertussis vaccine in pregnancy – what expectant mothers need to know[36] Operational Directives and Information Circulars[37]

GPs general practitioners

AMSs Aboriginal medical services

* Information sourced from ACT Health website

† Funded for use in third trimester of pregnancy from September 2013 but not the preferred option until April 2015

‡ Subsequently amended to accord with updated *Australian Immunisation Handbook* recommendations

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Annual report

Australian Rotavirus Surveillance Program annual report, 2014

Carl D Kirkwood, Susie Roczo-Farkas, and the Australian Rotavirus Surveillance Group

Abstract

The Australian Rotavirus Surveillance Program, together with collaborating laboratories Australia-wide, reports the rotavirus genotypes responsible for the hospitalisation of children with acute gastroenteritis. During the survey period of 1 January to 31 December 2014, 1,022 faecal samples were referred for rotavirus G and P genotype analysis, and of these 733 were confirmed as rotavirus positive. A total of 480 specimens were collected from children under 5 years of age, while 253 were from older children and adults. Genotype analysis of the 733 rotavirus samples collected from both children and adults revealed that G12P[8] was the dominant genotype in this reporting period, identified in 29.6% of strains nationally. Genotype G1P[8] was the 2nd most common strain nationally, representing 22.9% of samples, followed by genotype G3P[8] (14.9%). This report highlights the continued significance of G12P[8] strains as the major cause of disease in this population. The genotype distribution was slightly altered when the analysis was restricted to samples collected from children under 5 years of age, with G1P[8] being the dominant genotype (29%) followed by G12P[8] as the 2nd most common genotype (26%). Fluctuations in genotype distribution were also observed based on the vaccine type in use. Genotype G12P[8] was more common in states and territories using RotaTeq, while G1P[8] was more common in the locations using Rotarix. This survey highlights the yearly fluctuations in rotavirus genotypes observed since vaccine introduction. The continuation of G12P[8] as the dominant genotype further illustrates the dynamic and diversity present in the wild-type rotavirus population evident in the Australian population since vaccine introduction. *Commun Dis Intell* 2015;39(3):E337–E346.

Keywords: rotavirus, gastroenteritis, genotypes, disease surveillance

Introduction

Rotaviruses belong to the *Reoviridae* family and are triple layered dsRNA viruses that contain a segmented genome. The 11 gene segments encode 6 structural proteins and 6 non-structural proteins.[1]

Rotaviruses are the most common cause of severe diarrhoea in young children worldwide and is estimated to cause up to 453,000 deaths annually.[2] The significant morbidity and mortality associated with rotavirus infection has led to the development of vaccines. Two oral live attenuated rotavirus vaccines; Rotarix® (GlaxoSmithKline) and RotaTeq® (Merck), have been shown to be safe and highly effective in the prevention of severe diarrhoea due to rotavirus infection.[3,4] The successful development has seen both rotavirus vaccines licensed in over 125 countries and included in the national vaccination schedules of 63 predominantly high– and middle-income countries worldwide.[5] In Australia, rotavirus vaccines have been included into the National Immunisation Program from 1 July 2007, with excellent uptake in subsequent years. RotaTeq is administered in Victoria, South Australia, Western Australia and Queensland, while Rotarix is administered in New South Wales, the Northern Territory, Tasmania and Australian Capital Territory.[6]

In Australia, rotavirus infection accounted for up to 10,000 childhood hospitalisations for diarrhoea each year in the pre-vaccine era.[7] A significant impact on the disease burden has been observed since vaccine introduction, with state-based studies in New South Wales, Queensland, South Australia and Victoria showing a substantial decline in both rotavirus coded and non-rotavirus coded hospitalisations and emergency room visits since vaccine introduction.[6,8–10]

The Australian Rotavirus Surveillance Program has studied the annual circulation patterns of rotavirus genotypes causing disease in Australian children since 1997. Genotyping studies have shown that the strain diversity and temporal and geographic changes occur each year, and provide the baseline information vital to assist vaccine introduction and ongoing evaluation.[11] Therefore, characterisation of circulating rotavirus genotypes in the vaccine era will provide insight into whether vaccine introduction has impacted on virus epidemiology and altered circulating strains, which could have ongoing consequences for the success of the vaccination programs.

In this report, we describe the genotype of rotavirus strains causing severe gastroenteritis in Australia for the period 1 January to 31 December, 2014.

Methods

Rotavirus positive specimens detected by enzyme immunoassay (EIA) or latex agglutination in collaborating laboratories across Australia were collected, stored frozen and forwarded to Melbourne, together with relevant age and sex details. The laboratories contributing samples were:

- ACT Pathology, Canberra, 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
- Microbiology Department, John Hunter Hospital, Newcastle, New South Wales
- Microbiology Department, Royal Darwin Hospital, Casaurina, Northern Territory
- Microbiology Department, Alice Springs Hospital, Alice Springs, Northern Territory
- Forensic and Scientific Services, Queensland Health, Herston, Queensland
- Microbiology division, Pathology Queensland, Herston, Queensland
- Queensland Paediatric Infectious Diseases Laboratory, Royal Children's Hospital, Brisbane, Queensland
- Queensland Health laboratories in Townsville, Cairns and Gold Coast, Queensland
- Microbiology and Infectious Diseases Laboratory, SA Pathology, Adelaide, South Australia
- The Serology Department, Royal Children's Hospital, Parkville, Victoria
- Princess Margaret Hospital for Children, Subiaco, Western Australia
- Division of Microbiology, PathWest LM, The Queen Elizabeth Medical Centre, Nedlands, Western Australia.

Viral RNA was extracted from 10%–20% of faecal extracts of each specimen using the QIAamp Viral RNA mini extraction kit (Qiagen) according to the manufacturer's instructions. The rotavirus G and P genotype were determined for each sample by the application of independent hemi-nested multiplex reverse transcription polymerase chain reaction (RT-PCR) assays. The first round RT-PCR assays 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].[12–17] The G and P genotype of each sample was assigned using agarose gel analysis of second round PCR products.

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

Results

Number of isolates

A total of 1,022 faecal specimens were collected during the period 1 January to 31 December 2014 for analysis from 15 collaborating centres across Australia.

A total of 733 samples were confirmed as rotavirus positive by EIA (ProSpecT, OXOID) or RT-PCR analysis. Of these, 480 were collected from children under 5 years of age, and 253 were from older children and adults. An additional 289 specimens contained either insufficient specimen for genotyping (n= 30), or the specimen was not confirmed to be positive for rotavirus (n= 259) and these were not analysed further.

Age distribution

During the reporting period, 65.5% of samples were obtained from children under 5 years of age (Table 1). Overall, 21.8% of samples were from infants 0–6 months of age, 9.5% were from infants 7–12 months of age, 15.4% were from children 13–24 months of age, and 10.6% were from children 25–36 months of age. A total of 11.5% of samples were from children 5–10 years of age, and 20.2% of samples were from individuals older than 21 years of age.

In the samples from children under 5 years of age, a 3rd of all cases (33.3%) were identified in infants 0–6 months of age, while the next most common age group was 13–24 months where 23.5% of cases were found.

Genotype distribution

All of the 733 confirmed rotavirus samples collected from children and adults underwent geno-

Table 1: Age distribution of gastroenteritis cases

Age range (months)	Number	% of total	% under 5 years
0–6	160	21.8	33.3
7–12	70	9.5	14.6
13–24	113	15.4	23.5
25–36	78	10.6	16.3
37–48	33	4.5	6.9
49–60	26	3.5	5.4
Subtotal	480	65.5	100.0
61–120	84	11.5	
121–240	19	2.6	
241–960	103	14.1	
961+	45	6.1	
Unknown	2	0.3	
Total	733	–	

type analysis (Table 2). G12P[8] strains were the most common genotype identified nationally, representing 29.6% of all specimens analysed. This genotype was identified as the dominant type in 3 states: Queensland, Victoria and South Australia, representing 67%, 66% and 44% of strains respectively. Genotype G12P[8] strains were the 2nd most common type identified in Western Australia, representing 19.4% of strains, and was a minor type in New South Wales a representing 5.8% of samples.

G1P[8] strains were the 2nd most common genotype identified nationally, representing 22.9% of all specimens. This genotype was identified in 6 states, and was the dominant type in the Northern Territory and Western Australia, where it represented 71.2% and 23.6% respectively. In Victoria and South Australia, G1P[8] was the 2nd most common type, representing 15% and 20.8% respectively.

G3P[8] strains were the 3rd most common genotype nationally, representing 14.9% of all specimens. It was identified in all locations, and was the 2nd most common in New South Wales, representing 31.9% of strains.

New South Wales was the only state where neither G12P[8], G1P[8] nor G3P[8] were the dominant genotype. In this state, G9P[8] strains were the dominant type, identified in 31.9% of strains. Nationally, G9P[8] strains represented the 4th most common genotype, identified in 7.5% of strains overall.

Nine rare or uncommon genotype combinations were identified in 2014. Three G3P[9] strains were identified in South Australia, a single G9P[4] strain was identified in both Western Australia and the Australian Capital Territory, while a G9P[6] strain was identified in both Western Australia and New South Wales. A G8P[14] was identified in Victoria, and a GntP[14] was identified in Western Australia.

Seventy-one samples contained a non-typeable G or P genotype, with 50% observed in Western Australia. The majority of these were GntP[8]. These non-typeable samples are likely to be samples that contain inhibitors in extracted RNA that prevent the function of the enzymes used in RT and/or PCR steps.

Fifty-five faecal samples were identified that contained a component of the RotaTeq vaccine; 20 were from Western Australia, 34 were from South Australia and one from Victoria. In each instance a RotaTeq vaccine component was identified by RT-PCR and confirmed by sequence analysis.

Analysis of genotypes identified in samples from children under 5 years of age

A total of 480 rotavirus samples were collected from children under 5 years of age. In this cohort, genotype G1P[8] strains were the most commonly identified, found in 29% of samples, G12P[8] strains were the 2nd most common genotype, identified in 26% of samples, and G3P[8] strains were the 3rd most common genotype, identified in 14.4% of samples. G2P[4], G4P[8] and G9P[8] all represented minor genotypes in children in this study, and were identified in 2.5%, 1.5% and 6.5% of samples respectively (Table 3).

Analysis of genotypes identified in samples from those over 5 years of age

A total of 253 rotavirus samples were collected from children over the age of 5 years. In this cohort, genotype G12P[8] strains were the most commonly identified, found in 36.4% of samples, G3P[8] strains were the 2nd most common genotype, identified in 15.8% of samples, G1P[8] strains were the 3rd most common genotype, identified in 11.5% of samples and G2P[4] strains were the 4th most common type identified in 10.7% of samples. Genotype G9P[8] strains were identified in 9.5% of samples (Table 4).

Identification of genotypes according to vaccine type

A comparison of the G and P genotyping from the rotavirus samples collected from children under 5 years of age by vaccine usage was undertaken (Figure). Analysis revealed that in

Table 2: Rotavirus G and P genotype distribution in infants, children and adults, 1 January to 31 December 2014

Centre	Type total	G1P[8] %	G1P[8] n	G2P[4] %	G2P[4] n	G3P[8] %	G3P[8] n	G4P[8] %	G4P[8] n	G3P[9] %	G3P[9] n	G9P[8] %	G9P[8] n	G9P[4] %	G9P[4] n	G9P[6] %	G9P[6] n	G12P[8] %	G12P[8] n	G8P[14] %	G8P[14] n	Mix* %	Mix* n	Non-type† %	Non-type† n	Vaccine RotaTeq %	Vaccine RotaTeq n	Neg. n	insuff n
Australian Capital Territory																													
ACT	5	–	0	40	2	20	1	–	0	–	0	40	2	–	0	–	0	–	0	–	0	–	0	–	0			1	0
New South Wales																													
POW	8	13	1	25	2	25	2	–	0	–	0	–	0	–	0	–	0	13	1	–	0	–	0	25	2			1	0
Westmead	58	5	3	16	9	31	18	2	1	–	0	34	20	2	1	2	1	5	3	–	0	–	0	3	2			0	0
John Hunter	3	33	1	–	0	–	0	–	0	–	0	67	2	–	0	–	0	–	0	–	0	–	0	–	0			6	0
Northern Territory																													
Alice Springs	10	80	8	–	0	10	1	–	0	–	0	–	0	–	0	–	0	–	0	–	0	10	1	–	0			6	0
Darwin	12	75	9	–	0	25	3	–	0	–	0	–	0	–	0	–	0	–	0	–	0	–	0	–	0			4	0
Other‡	37	68	25	–	0	22	8	–	0	–	0	5	2	–	0	–	0	–	0	–	0	–	0	5	2			9	0
Queensland																													
Pathology Brisbane	13	8	1	–	0	–	0	–	0	–	0	15	2	–	0	–	0	77	10	–	0	–	0	–	0	–	0	5	0
Qld Regional	23	–	0	–	0	–	0	4	1	–	0	13	3	–	0	–	0	78	18	–	0	–	0	4	1	–	0	31	0
Pathology Townsville	8	13	1	–	0	25	2	–	0	–	0	–	0	–	0	–	0	38	3	–	0	–	0	25	2	–	0	7	0
Pathology (Gold Coast)	13	8	1	–	0	23	3	–	0	–	0	15	2	–	0	–	0	54	7	0	0	–	0	–	0	–	0	13	0
South Australia																													
Adelaide	245	21	51	3	7	5	13	2	4	1	3	1	2	–	0	–	0	44	107	–	0	–	0	10	24	14	34	85	1
Victoria																													
RCH	35	17	6	9	3	9	3	3	1	–	0	9	3	–	0	–	0	40	14	3	1	–	0	9	3	3	1	31	0
Monash	5	–	0	20	1	–	0	–	0	–	0	–	0	–	0	–	0	80	4	–	0	–	0	–	0	–	0	3	0
Western Australia																													
PathWest	239	25	59	6	14	21	51	1	2	–	0	7	16	0.4	1	0.4	1	18	44	–	0	–	0	13	32	8	19	46	2
Perth (P Marg)	19	11	2	5	1	21	4	5	1	–	0	5	1	–	0	–	0	32	6	–	0	–	0	16	3	5	1	38	0
Total	733	23	168	5	39	15	109	1	10	0.4	3	8	55	0.3	2	0.3	2	30	217	0.1	1	0.1	1	10	71	8	55	286	3

* A sample where a multiple G and/or P genotype was identified.

† A sample where no G and/or P genotype was not determined.

‡ Faecal samples from the Northern Territory which were processed in either Western Australia or Adelaide.

Neg Negative for rotavirus

Insuff Insufficient sample available for testing

Non-type and mix samples:

Western Australia

Princess Margaret: 3 x GntP[8]

WAPC 24 x GntP[8], 1x GntP[4], 4x G1P[nt], 1x G12P[nt], 1x GntP[14], 1x G4P[nt]

Queensland

Townsville: 2 x GntP[8]

Regional: 1 x GntP[8]

South Australia: 5 x GntP[8], 3x G1P[nt], 2x G12P[nt], 1x Gnt P[4/8], 3x GntP[4], 5x GntP[nt]

New South Wales:

POW: 1 x G2P[nt], 1X GntP[8]

Westmead: 1x GntP[8]

Northern Territory:

Other 1 x G9P[nt], 1x GntP[8]

Alice Springs 1 x G1/G9 P[8]

Victoria

RCH 3 x GntP[8]

Table 3: Rotavirus G and P genotype distribution in infants under 5 years of age, 1 January to 31 December 2014

Centre	Type total	G1P[8] %	G1P[8] n	G2P[4] %	G2P[4] n	G3P[8] %	G3P[8] n	G4P[8] %	G4P[8] n	G9P[8] %	G9P[8] n	G9P[4] %	G9P[4] n	G12P[8] %	G12P[8] n	Mix* %	Mix* n	Non-type† %	Non-type† n	Vaccine RotaTeq %	Vaccine RotaTeq n	Neg n	insuff n
Australian Capital Territory																							
ACT	1	–	0	–	0	–	0	–	0	100	1	–	0	–	0	–	0	–	0			1	0
New South Wales																							
POW	2	50	1		0	50	1	–	0	–	0	–	0	–	0	–	0	–	0			1	0
Westmead	38	3	1	16	6	32	12	–	0	42	16	3	1	3	1	–	0	3	1			0	0
John Hunter	1	–	0	–	0	–	0	–	0	100	1	–	0	–	0	–	0	–	0			1	0
Northern Territory																							
Alice Springs	10	80	8	–	0	10	1	–	0	–	0	–	0	–	0	10	1	–	0			6	0
Darwin	11	82	9	–	0	18	2	–	0	–	0	–	0	–	0	–	0	–	0			4	0
Other‡	32	66	21	–	0	22	7	–	0	6	2	–	0	–	0	–	0	6	2			6	0
Queensland																							
Pathology Brisbane	5	–	0	–	0	–	0	–	0	20	1	–	0	80	4	–	0	–	0	–	0	3	0
Qld regional	16	–	0	–	0	–	0	4	1	–	0	–	0	88	14	–	0	6	1	–	0	12	0
Pathology Townsville	4	25	1	–	0	50	2	–	0	–	0	–	0	–	0	–	0	25	1	–	0	3	0
Pathology (Gold Coast)	7	14	1	–	0	14	1	–	0	29	2	–	0	43	3	–	0	–	0	–	0	9	0
South Australia																							
Adelaide	151	29	44	–	0	6	9	2	3	–	0	–	0	34	52	–	0	7	10	22	33	48	1
Victoria																							
RCH	24	17	4	4	1	13	3	4	1	13	3	–	0	38	9	–	0	8	2	4	1	16	0
Monash	5	–	0	20	1	–	0	–	0	–	0	–	0	80	4	–	0	–	0	–	0	2	0
Western Australia																							
PathWest	158	30	47	2	3	17	27	1	2	3	5	0.6	1	20	32	–	0	14	22	12	19	23	1
Perth (P Marg)	15	13	2	7	1	27	4	–	0	–	0	–	0	40	6	–	0	7	1	7	1	30	0
Total	480	29	139	3	12	14	69	2	7	7	31	0.4	2	26	125	0.2	1	8	40	11	54	165	2

* A sample where a multiple G and/or P genotype was identified.

† A sample where no G and/or P genotype was not determined.

‡ Faecal samples from the Northern Territory which were processed in either Western Australia or Adelaide.

Neg = Negative for rotavirus.

Insuff = Insufficient sample available for testing.

POW Prince of Wales Hospital

RCH Royal Children's Hospital

Table 4: Rotavirus G and P genotype distribution in children older than 5 years of age, 1 January to 31 December 2014

Centre	Type total	G1P[8] %	G1P[8] n	G2P[4] %	G2P[4] n	G3P[8] %	G3P[8] n	G4P[8] %	G4P[8] n	G3P[9] %	G3P[9] n	G9P[8] %	G9P[8] n	G9P[6] %	G9P[6] n	G12P[8] %	G12P[8] n	G8P[14] %	G8P[14] n	Mix* %	Mix* n	Non–type† %	Non–type† n	Vaccine RotaTeq %	Vaccine RotaTeq n	Neg n	insuff n
Australian Capital Territory																											
ACT	4	–	0	50	2	25	1	–	0	–	0	25	1	–	0	–	0	–	0	–	0	–	0			0	0
New South Wales																											
POW	8	–	0	33	2	17	1	–	0	–	0	–	0	–	0	17	1	–	0	–	0	33	2			0	0
Westmead	20	10	2	15	3	30	6	5	1	–	0	20	4	5	1	10	2	–	0	–	0	5	1			0	0
John Hunter	2	50	1	–	0	–	0	–	0	–	0	50	1	–	0	–	0	–	0	–	0	–	0			5	0
Northern Territory																											
Alice Springs	0	–	0	–	0	–	0	–	0	–	0	–	0	–	0	–	0	–	0	–	0	–	0			0	0
Darwin	1	–	0	–	0	100	1	–	0	–	0	–	0	–	0	–	0	–	0	–	0	–	0			0	0
Other‡	5	80	4	–	0	20	1	–	0	–	0	–	0	–	0	–	0	–	0	–	0	–	0			3	0
Queensland																											
Pathology Brisbane	8	13	1	–	0	–	0	–	0	–	0	13	1	–	0	75	6	–	0	–	0	–	0	–	0	2	0
Qld regional	7	–	0	–	0	–	0	–	0	–	0	43	3	–	0	57	4	–	0	–	0	–	0	–	0	19	0
Pathology Townsville	4	–	0	–	0	–	0	–	0	–	0	–	0	–	0	75	3	–	0	–	0	25	1	–	0	4	0
Pathology (Gold Coast)	6	–	0	–	0	33	2	–	0	–	0	–	0	–	0	67	4	0	0	–	0	–	0	–	0	4	0
South Australia																											
Adelaide	94	7	7	7	7	4	4	1	1	3	3	2	2	–	0	59	55	–	0	–	0	15	14	1	1	37	0
Victoria																											
RCH	11	18	2	18	2	–	0	–	0	–	0	–	0	–	0	45	5	9	1	–	0	9	1	–	0	15	0
Monash	5	–	0	–	0	–	0	–	0	–	0	–	0	–	0	–	0	–	0	–	0	–	0	–	0	1	0
Western Australia																											
PathWest	81	15	12	14	11	30	24	–	0	–	0	14	11	1	1	15	12	–	0	–	0	12	10	–	0	46	2
Perth (P Marg)	4	–	0	–	0	–	0	25	1	–	0	25	1	–	0	–	0	–	0	–	0	50	2	–	0	38	0
Total	253	12	29	11	27	16	40	1	3	1	3	10	24	1	2	36	92	0.4	1	0.4	0	12	31	0.4	1	121	1

* A sample where a multiple G and/or P genotype was identified.

† A sample where no G and/or P genotype was not determined.

‡ Faecal samples from the Northern Territory which were processed in either Western Australia or Adelaide.

Neg Negative for rotavirus.

Insuff Insufficient sample available for testing.

POW Prince of Wales Hospital

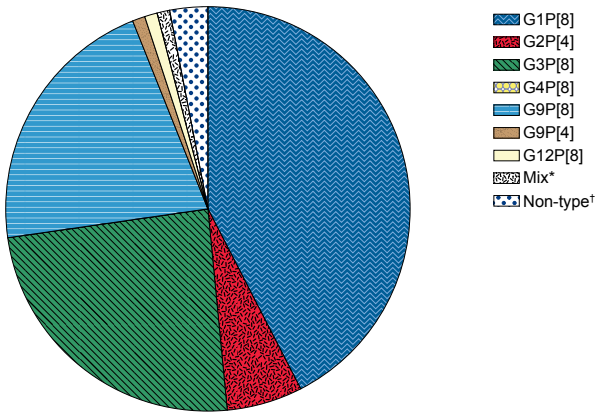
RCH Royal Children's Hospital

states where RotaTeq is in use, G12P[8] was the dominant genotype in children under 5 years of age, identified in 32.2% of samples, while G1P[8] was 2nd most common, identified in 25.7% of strains. G3P[8] strains were the 3rd most common genotype representing 12% of samples. In states using Rotarix, G1P[8] strains were dominant, identified in 42.1% of strains, while genotype G3P[8] and G9P[8] were identified in 24.2% and 21.1% of strains respectively.

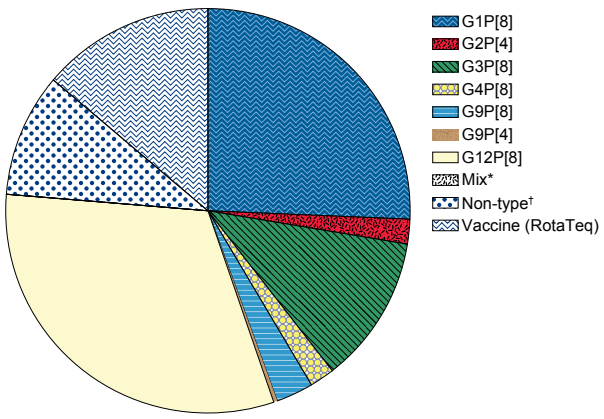
Consistency in genotype distribution within locations using RotaTeq vaccine was observed, with three of the 4 RotaTeq states (Queensland, Victoria and South Australia) having G12P[8] as the dominant genotype. In the remaining location, Western Australia, G12P[8] was the 2nd most common type. However, in locations using Rotarix (New South Wales and the Northern Territory), the dominant genotype differed, with G1P[8] being dominant in the Northern Territory and G9P[8] dominant in New South Wales.

Figure: Overall distribution of rotavirus G and P genotypes identified in Australian children based on vaccine usage for period 1 January to 31 December 2014

Rotarix states



RotaTeq states



* A sample where a multiple G and/or P genotype was identified.

† A sample where no G and/or P genotype was not determined.

Discussion

The Australian Rotavirus Surveillance Program describes the annual distribution of rotavirus genotypes and geographic differences in genotypes causing disease in Australia. This report is for the period 1 January to 31 December 2014. In analysis of all samples collected in 2014 from all age groups, genotype G12P[8] remained the dominant genotype nationally, representing 29.6% of all strains, being the dominant genotype in 3 states; Queensland, Victoria and South Australia. Genotype G1P[8] was the 2nd most common genotype nationally, comprising 22.9% of all strains, but was the dominant genotype in only 1 location, West Australia. Genotype G3P[8] represented the 3rd most common genotype, representing more than 14.9% of strains nationally. In the samples collected from children under 5 years of age, genotype G1P[8] was the dominant genotype nationally identified in 32.6% of samples. The 2nd most common genotype was G12P[8], identified in 29.3% of samples while G3P[8] was identified in 14.4% of samples and represented the 3rd most common genotype.

The unexpected emergence of G12P[8] in 2012, and dominance in 2013 and 2014 is the first time since vaccine introduction that a single genotype has remained dominant for more than a single year in Australia.[17,18] The G12P[8] genotype continued to persist in the same locations in 2014 as were observed in 2013, Queensland, Victoria and South Australia, and continued to emerge in Western Australia during 2014. The first G12 strain was detected in a 2-year-old child in 1987 in the Philippines. More than 10 years later, G12 strains were reported in many settings including Thailand (1998), the United States of America (USA) (1999) and subsequently in several Asian countries, such as India, Japan, and Korea.[19–22] In Europe, G12 strains were identified in 15 of 16 member countries between 2006–2009.[23] Until recently, G12 strains represented a sporadic or rare type in these settings, however, G12 has recently become prevalent in a number of countries in the developed and developing world. In West Africa, G12 strains represented more than 80% of strains in 2011–2012,[24] while in the Basque Country of Spain, G12P[8] was the predominant genotype, causing 65% of rotavirus gastroenteritis.[25] This Spanish outbreak was characterised by a broad geographical distribution (rural and urban) and affected both infants and children.[25] The emerging G12P[8] genotype has also been observed in Uruguay (2011–2012), as well as Italy in 2012–2013.[26–27] The New Vaccine Surveillance Network in the USA, surveyed 7 sites

during the 2009–2010 and 2010–2011 seasons, and G12 viruses accounted for approximately 10% of the typed rotaviruses. It was also found to account for 53% of a small group of rotavirus-positive stool samples from adults in Chicago in 2010.[28] Unlike the other settings, a marked increase in rotavirus hospitalisations was associated with the emergence of G12P[8] strains in Nicaragua, despite having a high vaccine coverage.[29] In Australia, both of the current rotavirus vaccines Rotarix and RotaTeq provided excellent protection against disease caused by the G12P[8] strains.

The predominance of G12P[8] rotaviruses in several locations suggest that they may soon become a major human rotavirus genotype. The emergence or re-emergence of G12P[8] rotavirus could be the result of vaccine pressure exerted on rotavirus types that are more similar to those in currently licensed vaccines or could represent genotype fluctuation occurring independently of the vaccine. Importantly, the presence of the P[8] VP4 protein in the G12 strains suggests that both rotavirus vaccines are likely to be effective against the emergence of G12P[8] strains. This is supported by a vaccine efficacy trial conducted in South Africa and Malawi that showed comparable protection against a range of circulating genotypes including G12 strains.[30]

This report continues to illustrate the diversity of rotavirus genotypes capable of causing disease. In this survey, genotypes G1P[8], G2P[4], G3P[8] and G9P[8] were all identified in more than 5% of strains. Genotype G1P[8] strains were the 2nd most common identified and G3P[8] strains the 3rd most common genotype. In the 6 previous reports G9P[8] strains have been a minor strain identified in less than 3% of strains. However, in 2014 genotype G9P[8] had re-emergence, being identified in 7.5% of strains. This genotype has not been reported as a re-emerging genotype elsewhere, so whether this genotype continues to emerge and cause disease in more Australians will be followed with interest.

The use of different vaccines in Australian states and territories provides a unique opportunity to compare the effect of each vaccine on the circulating wild type strains in cohort of strains identified in children under 5 years of age. In the current survey, G12P[8] strains were the most common in locations using RotaTeq vaccine, however, none were observed in locations using Rotarix vaccine. This represents the 3rd year where this genotype was more prevalent in locations using RotaTeq. Genotype G3P[8] strains were identified in both RotaTeq and Rotarix locations, however, they were more common in RotaTeq states in 2014, and represents the 4th year this has occurred.[17,18,31] In 2014, genotype G9P[8] was more common in locations using Rotarix vaccine. Thus differences in the genotype distribution were again observed based on vaccine usage in 2014, supporting those observed each year since vaccine introduction.[32]

This survey of rotavirus strains causing disease between 1 January and 31 December 2014 highlights the continued emergence of G12P[8] rotavirus as the dominant genotype in Australia. The emergence of G12P[8] and the re-emergence of G9P[8] in 2014 illustrates a unique change to the genotype patterns in Australia. The genotype data highlight the diversity of rotavirus, and illustrates the continual changes in the wild type virus population, suggesting a more dynamic virus population is present in the current post vaccine era. The continued changes in the wild type strain population will continue to challenge vaccine effectiveness. Importantly however, rotavirus vaccines continue to be highly effective and provide excellent protection from disease caused by the circulating rotavirus strains.

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Australian Gonococcal Surveillance Programme annual report, 2014

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Abstract

The Australian Gonococcal Surveillance Programme (AGSP) has continuously monitored antimicrobial resistance in clinical isolates of *Neisseria gonorrhoeae* from all states and territories since 1981. In 2014, 4,804 clinical isolates of gonococci from public and private sector sources were tested for *in vitro* antimicrobial susceptibility by standardised methods. Decreased susceptibility to ceftriaxone (MIC value 0.06–0.125 mg/L) was found nationally in 5.4% of isolates, a lower proportion than that reported in the AGSP 2013 annual report (8.8%). The highest proportions were reported from New South Wales and Victoria (7.1% and 6.6% respectively). The proportion of strains resistant to penicillin in urban and rural Australia ranged from 11% in South Australia to 43% in New South Wales. In rural and remote Northern Territory penicillin resistance rates remained low (1.5%). In remote Western Australia relatively low numbers of strains are available for testing, however there is now widespread molecular testing for penicillin resistance in Western Australia to monitor resistance and inform guidelines and, for first time, these data are included in the AGSP annual report. Quinolone resistance ranged from 27% in the urban and rural areas of the Northern Territory, to 44% in the Australian Capital Territory, and quinolone resistance rates remain comparatively low in remote areas of the Northern Territory (3.1%) and remote areas of Western Australia (5.6%). Azithromycin resistance ranged from 0.5% in South Australia to 5.3% in rural and urban Western Australia. High rates were also reported from the Australian Capital Territory but relatively low numbers were tested. High level resistance to azithromycin (MIC value ≥256 mg/L) was again reported in 2014, in 2 strains from New South Wales. No resistance was reported from the Northern Territory, or remote Western Australia. *Commun Dis Intell* 2015;39(3):E347–E354.

Keywords: antimicrobial resistance; disease surveillance; gonococcal infection; *Neisseria gonorrhoeae*

Introduction

Gonococcal antimicrobial resistance (AMR) was identified as an urgent public health threat by the United States Centers for Disease Control and Prevention in 2013,[1] and the emergence and spread of multidrug-resistant gonorrhoea is predicted to impose significant collateral health and financial costs.[1] For the majority of Australia, and internationally, there is reliance on a dual treatment protocol for gonorrhoea of ceftriaxone and azithromycin, with uncertainty regarding the future direction of gonococcal treatment as the ideal alternative strategy is yet to be identified.[2] In recent years in Australia, there has been a significant increase in rates of gonococcal disease observed in both males and females in the eastern states (Victoria, New South Wales and Queensland), and males in the Australian Capital Territory.[3] In contrast, in Indigenous populations in remote regions of the Northern Territory and Western Australia, gonococcal disease notifications rates are significantly higher but remain relatively stable.[3] The overall Australian age standardised gonorrhoea notification rates in 2012 for Indigenous compared with non-Indigenous Australians were 933 per 100,000 population and 38.5 per 100,000 population, respectively.[3] In these remote areas where disease rates are high, antimicrobial resistance is paradoxically low, and gonorrhoea acquired locally or in an endemic region can still be effectively treated with an oral antibiotics regimen (amoxycillin 3 g, probenecid 1 g and azithromycin 1 g).[4] Importantly though, the remoteness of these regions poses limits in terms of access to diagnostic services. For this reason nucleic acid amplification tests are relied on for diagnosis, and relatively few isolates are available for antimicrobial resistance testing in these regions where monitoring antimicrobial resistance is critical. The development and implementation of an assay to detect penicillinase production[5,6] (the primary cause of penicillin resistance in remote regions) is the first documented use of molecular testing for gonococcal antimicrobial resistance detection and surveillance to monitor AMR and inform local treatment guidelines.[6]

The Australian gonococcal AMR data for 2013 were cause for considerable concern as the proportion of strains with decreased susceptibility to ceftriaxone was reported to be 8.8%, double that of 2012 (4.4%). The highest proportions (11.8%) were reported from New South Wales and Victoria where the greatest increases in gonococcal disease notifications occurred.[7] High level resistance to azithromycin (MIC value >256 mg/L) was also reported in 2013, in 2 strains from Victoria and 2 strains from Queensland.[8] In addition, an

imported multi-drug resistant gonococcal strain, known as the A8806 strain, with a ceftriaxone MIC of 0.5 mg/L, the highest ever reported in Australia, was identified in Australia in 2013.[9] Molecular investigations of this strain showed key genetic similarities to the ceftriaxone-resistant strain H041, which was observed in only a single case in Japan.[9] Enhanced surveillance in the Northern Territory and Queensland has not detected further evidence of the A8806 strain in 2014 (unpublished data from the NNN).

Almost two-thirds of the World Health Organization (WHO) estimated 106 million new *Neisseria gonorrhoeae* infections reported in those aged 15–49 years worldwide each year occur in the Asia–Pacific Region.[10] The WHO gonococcal antimicrobial surveillance data from the Asia–Pacific indicate that there are high levels of gonococcal AMR in the Region, which is densely populated with a disproportionate burden of gonococcal disease. In many countries there is uncontrolled antimicrobial use providing ideal conditions for the development of AMR.[11] This is of continuing concern to Australia where, in urban centres, AMR in *N. gonorrhoeae* has long been influenced by the introduction of multi-resistant strains from overseas.[12] Importation and spread of resistant gonococcal strains and/or resistance developing under selection pressure is an ongoing concern.

Strategies for treating and controlling gonorrhoea are based on regimens effecting cure in a minimum of 95% of cases. Surveillance data derived from continuous monitoring of resistance to the antibiotics in clinical use is therefore critical to monitor AMR, detect imported or novel resistance and to inform treatment guidelines.[13] The WHO has called for enhanced surveillance as a fundamental component of the Global Action Plan to control the spread and impact of gonococcal AMR.[14]

In Australia, the National Neisseria Network (NNN) is a collaboration of reference laboratories in each state and territory that monitors clinical isolates of pathogenic *Neisseria* species nationally from public and private sector laboratories representing as wide a section of the community as possible, for phenotypic and genotypic characteristics, including antimicrobial resistance. The Australian Gonococcal Surveillance Programme (AGSP) is a key activity of the NNN and has continuously monitored the susceptibility of *N. gonorrhoeae* since 1981, making it the longest continually running national surveillance system for gonococcal AMR. In this annual report, for the first time, we provide molecular surveillance data from Western Australia to supplement the AGSP data. This is amid increasing concerns nationally of the status of gonococcal AMR in Australia.

Methods

The NNN AMR data for gonococcal isolates are collated for the AGSP quarterly and annual reports. Gonococcal infection is a notifiable disease in Australia and each confirmed case is notified to the National Notifiable Diseases Surveillance System (NNDSS). The number of isolates tested by the NNN and reported by the AGSP represents a proportion of the number of cases reported to the NNDSS. The NNN tests approximately one-third of the number of notified cases in Australia.

The NNN laboratories test gonococcal isolates for susceptibility to penicillin (representing this group of antibiotics); ceftriaxone (representing later generation cephalosporin antibiotics); ciprofloxacin (representing quinolone antibiotics); azithromycin; spectinomycin; and for high level plasmid mediated resistance to tetracycline using previously described standardised methodology to determine the minimum inhibitory concentration (MIC) values.[15, 16] The MIC value is the least concentration of an antibiotic that inhibits *in vitro* growth under defined conditions. The AGSP conducts a program-specific quality assurance program.[17]

Antibiotic susceptibility data from each jurisdiction are submitted quarterly to the coordinating laboratory (the Neisseria Reference Laboratory and WHO Collaborating Centre for Sexually Transmitted Diseases, Sydney), which collates the results for reporting. Where available, the AGSP collects data on the gender of the patient, country of acquisition, and site of isolation of gonococcal strains. Data from isolates from all jurisdictions is predominantly from urban centres. Data from the Northern Territory and Western Australia are further divided into urban versus rural and remote as therapeutic recommendations differ.

Statistics

Statistical analysis was performed using Prism % version 5.0d. Results were compared using Fisher's exact test for proportional differences.

Results

Number of isolates

There were 4,804 gonococcal isolates tested in NNN laboratories in 2014, representing 31% of the 15,728 cases of gonococcal infection notified to the NNDSS in 2014 (Table 1).[18] This was slightly lower than the proportion tested in 2013 (33%) and lower than the 35%–42% referred between 2008 and 2012.

Table 1: Number of Australian Gonococcal Surveillance Programme gonococcal isolates tested as a proportion of National Notifiable Diseases Surveillance System gonorrhoea notifications, Australia, 2014, by state or territory

State or territory	Number of isolates tested	Number of cases notified*	Number of isolates tested/ Number of cases notified %
Australian Capital Territory	75	120	63
New South Wales	1,672	4,862	34
Northern Territory	229	1,759	13
Queensland	650	2,723	24
South Australia	207	765	27
Tasmania	30	65	46
Victoria	1,440	3,240	44
Western Australia	501	2,194	23
Australia	4,804	15,728	31

Source of isolates

There were 4,009 isolates from men (83%) and 791 (17%) from women (Table 2). Four isolates were from patients of unknown gender. The proportion of gonococcal isolates from males and females tested by the AGSP has remained stable over recent years (2009–2012); ranging between 18% and 20% for women and 80% and 82% for men. The infected site was reported as 'other' or not specified for 58 isolates from males and 25 isolates from females (Table 2). Isolates from urine samples were regarded as genital tract isolates.

Antibiotic susceptibility patterns

As in past years, the patterns of gonococcal antibiotic susceptibility differed between the various states and territories. The data are presented by region as well as aggregated for Australia (Table 3).

Penicillin

Resistance to the penicillin group of antibiotics (penicillin, ampicillin and amoxycillin with or without clavulanic acid) in gonococci is a result of the production of a specific beta lactamase: penicillinase; and/ or by the aggregation of chromosomally-controlled resistance mechanisms. These are denoted respectively, as penicillinase-producing *N. gonorrhoeae* (PPNG); and chromo-

Table 2: Gonococcal isolates tested, Australia, 2014, by sex, site and state or territory

Sex	Site	ACT	NSW	NT	Qld	SA	Tas.	Vic.	WA	Aust.
Male	Genital	21	823	141	354	109	20	620	290	2,378
	Rectal	21	328	1	93	41	3	384	74	945
	Pharynx	22	277	1	40	22	1	210	39	612
	DGI	0	1	3	10	0	0	0	2	16
	Other/NS	1	21	1	2	0	1	30	2	58
	Total	65	1,450	147	499	172	25	1,244	407	4,009
Female	Genital	8	169	73	130	29	2	167	74	652
	Rectal	0	6	1	3	3	0	3	5	21
	Pharynx	0	39	1	6	3	0	13	9	71
	DGI	0	1	5	9	0	0	0	6	21
	Other/NS	2	4	1	3	0	3	13	0	26
	Total	10	219	81	151	35	5	196	94	791
Unknown	Total	0	3	1	0	0	0	0	0	4
Total		75	1,672	229	650	207	30	1,440	501	4,804

DGI: Disseminated Gonococcal Infection; NS: not specified

Table 3: Proportion of gonococcal isolates with resistance to penicillin, ciprofloxacin and azithromycin and decreased susceptibility to ceftriaxone reported, Australia, 2014, by state or territory

State or territory	Number of isolates tested	Decreased susceptibility Ceftriaxone		Resistance Ciprofloxacin		Azithromycin		Penicillin	
		n	%	n	%	n	%	n	%
Australian Capital Territory	75	2	2.7	33	44.0	7	9.3	9	12.0
New South Wales	1,672	119	7.1	726	43.0	33	2.0	725	43.0
Queensland	650	21	3.2	184	28.0	23	3.5	153	24.0
South Australia	207	2	1.0	86	42.0	1	0.5	22	11.0
Tasmania	30	0	0.0	8	27.0	1	3.3	7	23.0
Victoria	1,440	95	6.6	559	39.0	33	2.3	322	22.0
Northern Territory/Urban	99	3	3.0	27	27.0	0	0.0	21	21.0
Northern Territory/Remote & Rural	130	1	0.8	4	3.1	0	0.0	2	1.5
Western Australia/Urban & Rural	393	14	3.6	117	30.0	21	5.3	104	26.0
Western Australia/Remote	108	1	0.9	6	5.6	0	0.0	5	4.6
Australia	4,804	258	5.4	1,750	36.0	119	2.5	1,370	29.0

somally mediated resistant to penicillin (CMRP). Chromosomal resistance is defined as an MIC to penicillin of 1 mg/L or more.

In 2014, 1,370 (29%) isolates were penicillin resistant, which was a proportional decrease from 2012–2013 (3%2–35%); similar to that reported in 2010–2011 (25%–29%); but lower than 2008–2009 (36%–44%). In 2014, there were 652 (14%) isolates with CMRP; and 718 (15%) with PPNG. In comparison in 2013, the proportion of isolates with CMRP was 20%, and 15% were PPNG. Thus the decrease in penicillin resistance nationally in 2014 was due to a decrease in the proportion of isolates with CMRP.

Penicillin resistance in the Northern Territory

In 2014 there were 229 isolates tested from the Northern Territory. Of these, 99 were from Darwin, and 130 were from rural and remote Northern Territory comprising 117 from Alice Springs, 5 isolates from Katherine and 8 isolates from other areas.

Of the isolates tested from the Northern Territory, 21 (21%) from the city of Darwin were penicillin resistant: (3 CMRP and 18 PPNG) (Table 3). Of these, two also had decreased susceptibility to ceftriaxone. In contrast, from the remote regions of the Northern Territory, 2 (1.5%) strains tested were penicillin resistant (both PPNG).

Penicillin resistance in Western Australia

In 2014, there were 501 isolates tested from Western Australia; 108 from remote regions and 393 from rural and urban regions. Of the isolates tested from rural and urban regions, 26% were reported as resistant, whereas of the 108 from remote regions there were 5 isolates (4.6%) that were penicillin resistant (3 PPNG and 2 CMRP).

In addition to the isolate based surveillance for penicillin, specimens that were *N. gonorrhoeae* positive by a nucleic acid amplification test (NAAT) in Western Australia were tested using a PPNG assay now routinely in use at PathWest.[5,6] There were 1,158 gonococcal diagnoses by NAATs at PathWest from across Western Australia and of those, 1,011 (87%) were able to be tested for PPNG. There were high rates of PPNG in Perth (17%); Wheatbelt (14%); Great Southern (33%) and SouthWest (25%). Conversely, the remote regions continue to have lower rates of PPNG positive *N. gonorrhoeae*. There were 4/120 (3%) from the Pilbara and 0/475 (0%) from the Kimberley region. Lower rates of PPNG were also reported from the Midwest and Goldfields (7% and 11% respectively), but these rates are considered less reliable as lower numbers were tested in these regions (58 and 35 respectively). These data support and enhance the isolate based surveillance findings of the AGSP, that PPNG rates remain low in the remote regions of Western Australia. All PPNG positive *N. gonorrhoeae* from remote regions were determined to be in non-local workers or residents in the major regional centres and there was no PPNG positive

N. gonorrhoeae detected from remote Indigenous communities (personal communication, Dr David Speers, PathWest).

Ceftriaxone

From 2001 onwards, gonococcal isolates categorised as having decreased susceptibility to ceftriaxone, by the AGSP criteria (MIC values 0.06–0.125 mg/L), have been reported in Australia. The proportion increased incrementally from 0.6% in 2006, to 4.4% in 2012, then in 2013 doubled to 8.8%. In 2014, the proportion of gonococci with decreased susceptibility to ceftriaxone nationally decreased to 5.4% (Table 4).

Ceftriaxone decreased susceptibility includes the MIC values 0.06 and 0.125 mg/L. The right shift in the distribution of ceftriaxone MIC values over recent years (Table 5), is statistically significant with a sustained increase in the proportion of strains with an MIC value of 0.06 mg/L (2011–2012: [P=0.02, 95% CI: 1.04–.62], and 2012–2013 [P<0.0001, 95% CI: 1.70–2.38]). In 2014, the proportion of strains with an MIC value of 0.06 mg/L decreased to 4.8%.

The proportion of strains with a ceftriaxone MIC 0.125 mg/L also increased from 0.1% in 2010 and 2011, to 0.3% in 2012 to 0.6% in 2013, but was unchanged in 2014 (Table 5). These differences were not significant, which may be attributable to the low number of strains in this MIC category. No isolates of *N. gonorrhoeae* with an MIC value greater than 0.125 mg/L were reported from Australia in 2014.

Azithromycin

Nationally, the proportion of isolates exhibiting any resistance to azithromycin (2.4%) was higher than that reported in 2011–2012 (1.1%–1.3%), and slightly higher than the previous year (2.1%) (Table 3). There were marked increases in the proportion of strains with resistance to azithromycin between 2012 and 2013 from the Australian Capital Territory (from 2.2% to 9.3%) but the number of isolates tested was relatively low; and in Western Australia (from 1.9% to 4.2%); and also New South Wales (from 1.0% to 2.0%). In 2014, there were 2 isolates, both from New South Wales, that exhibited high level resistance to azithromycin (MIC value > 256 mg/L).

Quinolone antibiotics

The AGSP uses ciprofloxacin as the representative quinolone. Quinolone resistant *N. gonorrhoeae* are defined as MICs ≥ 1 mg/L. The resistance mechanism in *N. gonorrhoea* has thus far been mediated only by chromosomal mechanisms so that incremental changes in MIC values are observed.

Table 4: Number and percentage of gonococcal isolates with decreased susceptibility to ceftriaxone (MIC 0.06–0.125 mg/L), Australia, 2010 to 2014, by state or territory

	Decreased susceptibility to ceftriaxone									
	2010		2011		2012		2013		2014	
State or territory	n	%	n	%	n	%	n	%	n	%
Australian Capital Territory	2	6.7	2	3.1	2	3.6	0	0.0	2	2.7
New South Wales	74	5.6	58	4.4	76	4.5	183	11.8	119	7.1
Northern Territory	1	0.2	2	0.4	0	0.0	4	1.5	4	1.7
Queensland	26	3.2	18	2.3	17	2.4	33	4.9	21	3.2
South Australia	19	11.6	1	0.7	1	0.7	4	1.9	2	1.0
Tasmania	0	0.0	0	0.0	0	0.0	11	24.4	0	0.0
Victoria	52	5.7	50	5.3	105	8.4	181	11.8	95	6.6
Western Australia	17	5.2	3	0.7	6	1.2	13	2.7	15	3.0
Australia	191	4.8	134	3.2	207	4.4	429	8.8	258	5.4

Table 5: Proportion of gonococcal isolates tested with MIC values at 0.06 mg/L and 0.125 mg/L, Australia, 2010 to 2014

Ceftriaxone MIC mg/L	2010	2011	2012	2013	2014
0.06	4.8%	3.2%	4.1%	8.2%	4.8%
0.125	0.1%	0.1%	0.3%	0.6%	0.6%

In 2014, 1,750 of the 4,800 gonococci examined (36%) were resistant to ciprofloxacin (Table 3). The proportion reported by the AGSP in 2012 (30%) and 2013 (34%) was lower, however overall there has been a trend of decreasing proportions since 2008, when 54% isolates were reported as ciprofloxacin resistant.

High-level tetracycline resistance

High-level tetracycline resistant *N. gonorrhoeae* (TRNG) is used as an epidemiological marker even though tetracyclines are not a recommended treatment for gonorrhoea and are rarely, if ever used for treatment of gonorrhoea in Australia. The proportion of TRNG detected nationally increased between 2006 and 2011 from 12% to 21% and decreased to 14% in 2012, and was again reported as 14% in 2013. In 2014 the proportion of TRNG was 19%.

TRNG were present in all jurisdictions in 2014, with the highest proportions in remote Northern Territory (44%), New South Wales (26%), and remote Western Australia (23%).

Spectinomycin

In 2014, all isolates from jurisdictions testing (Western Australia; Victoria; South Australia; Queensland; Tasmania) were susceptible to spectinomycin.

Discussion

The WHO recommends that treatment regimens for gonorrhoea are based on epidemiological surveillance of the distribution and extent of AMR, and that a resistance rate of 5% or more is the nominal threshold for change of treatment recommendations.[13] The AGSP has continuously monitored antimicrobial resistance in Australia since 1981, and has established quality assurance and quality control of gonococcal AMR data with the AGSP External Quality Assurance Program and WHO *N. gonorrhoeae* reference strains.[17,19]

The overall number of gonococcal strains examined by the AGSP in 2014 was higher in number but proportionally the same as 2013. The clinical isolates were from both the public and private health sectors, constituting a comprehensive sample of 31% of all notifications nationally. The increasing use of NAATs for diagnosis, both in urban setting and remote settings is of increasing concern for gonococcal AMR surveillance programs worldwide because of the impact on the number of strains for AMR testing, and therefore antimicrobial resistance surveillance data. Whilst NAATs have an advantage over culture in terms of sensitivity, and are more robust and reliable for remote settings where cultures may not survive, the distinct disadvantage is that they cannot test broadly for AMR. However, molecular AMR testing strategies can give targeted and specific information, which is clinically and epidemiologically important.[2] At this stage, however, NAATs are unable to provide definitive data for predicting AMR, thus the continued commitment to the support of surveillance programs such as the AGSP is vital to monitor and detect new resistant strains. However, directed and specific NAATs such as the PPNG assay can contribute to surveillance programs and can be used to inform treatment guidelines.[6] This AGSP report includes for the first time, the PPNG NAAT data from Western Australia, which provides important additional situational data for the AGSP in a region where penicillin based treatment strategies are still in place. Introduction of this assay is planned for the Northern Territory, where penicillin based treatment strategies are also in use, to provide enhanced surveillance data for 2015.

The primary focus for surveillance for the majority of Australia, and in most countries, is the monitoring of ceftriaxone MIC values. Gonococci with decreased susceptibility to ceftriaxone (MIC value in the range 0.06–0.125 mg/L) have been reported in increasing proportions in Australia, with the rate doubling over the period 2012 to 2013, from 4.4% to 8.8%.[7] In 2014, there was a decrease in the proportion of isolates with decreased susceptibility to ceftriaxone in New South Wales, Victoria, South Australia and Queensland, whereas proportions were essentially unchanged in Western Australia and the Northern Territory. Low numbers of isolates were tested in Tasmania and the Australian Capital Territory. There was no evidence of spread of the A8806 strain reported in 2013. Decreased susceptibility to the cephalosporin antibiotics has been accompanied by increasing numbers of reports of treatment failures; and multi-drug resistant strains with high level resistance to ceftriaxone have been reported from Japan, France, Spain and now Australia.[9,20–22] All of these strains with high level resistance to ceftriaxone have been shown to have a mosaic penicillin binding protein 2 (PBP2) encoded by a mosaic *PenA* gene, with as little as a single additional amino acid substitution required to confer resistance.[23]

Of significant concern is that molecular studies have shown that strains harbouring the mosaic PBP2 are present in a significant proportion of circulating strains globally; are critically only one mutation from high level resistance, and are under constant selection pressure. Paradoxically, these strains with a mosaic PBP2 may not have an elevated ceftriaxone MIC value (i.e. decreased susceptibility) so would not be included in phenotypic

surveillance. Given these considerations, the level of concern about the development of ceftriaxone resistance is growing globally.[23]

International surveillance programs define decreased susceptibility to ceftriaxone differently. The absence of an international standard definition of decreased susceptibility to ceftriaxone, and non-uniform methods of AMR testing confound comparison of surveillance data. However in 2012, the WHO Global Action Plan nominated the criteria for decreased susceptibility to ceftriaxone as an MIC value ≥0.125 mg/L.[14] The proportion of strains tested by the AGSP with a ceftriaxone MIC value of 0.125 mg/L also doubled from 0.3% in 2012 to 0.6% in 2013 but was unchanged in 2014.

A dual therapy strategy of ceftriaxone with oral azithromycin for uncomplicated gonococcal infection is recommended in Australia.[4] In 2013, high level resistance to azithromycin in gonococci was reported for the first time in Australia.[8] There were 4 strains reported; two from Victoria and two from Queensland, and of these, two were likely to have been acquired from China).[8] In 2014, there were 2 further strains reported with high level azithromycin resistance in New South Wales. Evidence of coevolving cephalosporin and azithromycin resistance is being observed outside Australia and is of significant concern.[23]

The proportion of gonococci with high-level tetracycline resistance in Australia increased from 2006 to 2008 and stabilised at 21% in 2009 to 2010. The proportion of TRNG decreased to 18% in 2011, then to 14% in 2012 and remained unchanged (14%) in 2013. In 2014, there was an increase to 19%. Outside of the remote regions of Western Australia and the Northern Territory penicillin and ciprofloxacin resistance rates remain high. There was no resistance to spectinomycin reported in the jurisdictions testing for this antibiotic.

The continued emergence and spread of AMR in *N. gonorrhoeae* is widely recognised as a global public health threat. Broad based disease control strategies including the rational use of antibiotics have been called for. The WHO Global Action Plan states that disease control strategies and the understanding of the global scope of AMR need to continue to be informed by surveillance programs of AMR, nationally and internationally.[14] The need for close and enhanced monitoring of gonococcal AMR is patently clear. NAATs can play a role in this; however, isolate based surveillance to monitor *N. gonorrhoeae* with elevated MIC values, coupled with sentinel site surveillance in high risk populations, remains critically important to inform therapeutic strategies and to detect instances of treatment failure. Sentinel site surveillance programs involve patient follow up and test of cure cultures after treatment of *N. gonorrhoeae* infections, in particular those in oropharyngeal sites. This is currently conducted in a very limited number of settings in Australia, and needs to be expanded throughout all jurisdictions as a matter of priority.

In summary, gonococcal disease rates and AMR rates are increasing. In 2013, the proportion of strains with elevated ceftriaxone MIC values doubled from that reported in 2012. In 2014 this declined to 5.4% but there is little reassurance in this. For the second consecutive year in Australia, high level resistance to azithromycin has been reported. The next direction for treatment is uncertain, but what is clear is that additional and renewed efforts for disease prevention and disease control are urgently called for, and that continued monitoring of AMR to inform treatment and monitor interventions is paramount.

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Influenza epidemiology in adults admitted to sentinel Australian hospitals in 2014: 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 2014 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. During the period 3 April to 31 October 2014 (the 2014 influenza season), 1,692 adult patients (>16 years) were admitted with confirmed influenza to one of 15 of 17 FluCAN sentinel hospitals (excluding 2 paediatric hospitals). Of these, 47% were over 65 years of age, 10% were Indigenous Australians, 3.3% were pregnant and 85% had chronic comorbidities. The majority of cases were due to influenza A. Influenza B was detected in 7% of patients. There were a large number of hospital admissions detected with confirmed influenza in this national observational surveillance system in 2014. These are estimated to represent a national annual burden of around 15,000 admissions and almost 100,000 bed-days nationally. *Commun Dis Intell* 2015;39(3):E355–E360.

Keywords: influenza; hospitalisation; morbidity

Introduction

Influenza is a common respiratory viral infection that affects up to 5%–10% of the population each year.[1] Although the proportion of cases requiring hospitalisation is low, because infection with influenza virus is relatively widespread, the incidence of hospitalisation from influenza is of public health significance.[2]

We established a national sentinel surveillance program for severe influenza in 2009 primarily to provide timely information to public health authorities nationally on hospitalisations with laboratory-confirmed influenza. In this report, we describe the epidemiology of hospitalisation in adults with laboratory-confirmed influenza. A report on severe paediatric influenza will be reported separately.

Methods

The Influenza Complications Alert Network (FluCAN) is a national hospital-based sentinel surveillance system.[3] For the 3 most recent influenza seasons including 2014, the participating sites have been The Alfred Hospital (Vic.), Royal Melbourne Hospital (Vic.), Canberra Hospital (ACT), Calvary Hospital (ACT), Monash Medical Centre (Vic.), University Hospital Geelong (Vic.), Royal Perth Hospital (WA), Royal Adelaide Hospital (SA), Royal Hobart Hospital (Tas.), Mater Hospital (Qld), Princess Alexandra Hospital (Qld), Cairns Base Hospital (Qld), Alice Springs Hospital (NT), Westmead Hospital (NSW), and John Hunter Hospital (NSW). In 2014, 2 additional paediatric speciality hospitals also participated but paediatric data will be reported separately. Case numbers vary from previous reports due to exclusion of paediatric cases.[4] Ethical approval has been obtained at all participating sites, at Monash University and the Australian National University.

An influenza case was defined as a patient admitted to hospital with influenza confirmed by nucleic acid testing. Surveillance is conducted from April to November (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. 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 on all pre-disease performance without restriction.[5]

We examined factors associated with ICU admission and the length of hospital stay using

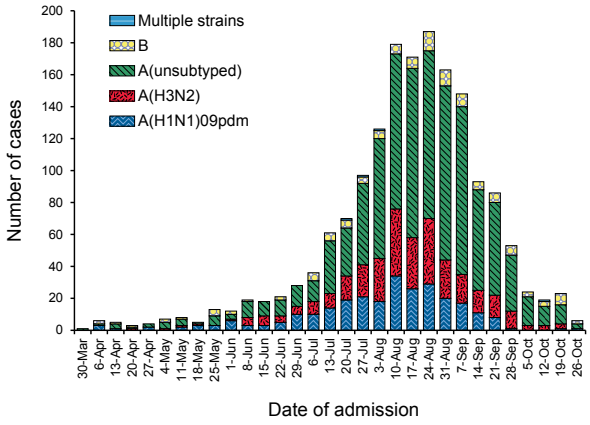
multivariable regression. Factors associated with ICU admission were determined using a logistic regression model, with factors retained in the multivariable model if $P<0.2$. Factors associated with the length of hospital stay were modelled using a linear regression, as the mean duration of stay was the parameter of interest. Standard errors were estimated using bootstrapping (1,000 replicates) to correct for non-normality of residuals due to the skewed distribution of length of stay.

Results

During the period 5 April to 31 October 2014 (the 2014 influenza season), 1,692 patients were admitted with laboratory-confirmed influenza to one of 15 FluCAN non-paediatric sentinel hospitals. The peak rate of admission was highest in early September, and varied by jurisdiction between mid-August and mid-September (Figure). The majority of cases were due to influenza A, but 115 (6.8%) were due to influenza B. The proportion due to influenza B varied by site from 1 of 55 cases (2%) at the University Hospital Geelong, to 32 of 180 cases (18%) at Westmead Hospital.

Of these 1,692 patients, 793 (47%) were more than 65 years of age, 169 (10%) were Indigenous Australians, 56 (3.3%) were pregnant and 1,433 (85%) had chronic co-morbidities (Table 1 and Table 2). Of the 1,283 patients (76%) where influenza vaccination status was ascertained, 636 (50%) had been vaccinated. The most commonly reported co-morbidities were respiratory disease, cardiac disease, immunosuppression and diabetes. Of the total, 684 (40%) patients reported a restriction in functional status and 111 (6.6%) were resident in an aged care facility.

Figure: Date of admission in patients hospitalised with confirmed influenza, 5 April to 31 October 2014



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

Table 1: Demographic characteristics of hospitalised adult patients with confirmed influenza

Characteristic	Influenza type								Total*	
	A/H1N1/09pdm		A/H3N2		A/unsubtyped		B			
Total	271		309		993		115		1,692	
Age group	**n**	**%**	**n**	**%**	**n**	**%**	**n**	**%**	**n**	**%**
16-49 years	126	46.5	67	21.7	276	27.8	37	32.2	507	30.0
5-65 years	79	29.2	53	17.2	234	23.6	26	22.6	392	23.2
65-79 years	51	18.8	107	34.6	250	25.2	24	20.9	434	25.7
80+ years	15	5.5	82	26.5	233	23.5	28	24.3	359	21.2
Male	136	50.2	149	48.2	436	43.9	52	45.2	776	45.9
Indigenous	11	4.1	4	1.3	146	14.7	8	7.0	169	10.0
State or territory										
ACT	39	14.4	100	32.4	9	0.9	10	8.7	158	9.3
NSW	32	11.8	124	40.1	137	13.8	39	33.9	335	19.8
NT	0	0.0	0	0.0	131	13.2	9	7.8	140	8.3
Qld	19	7.0	17	5.5	172	17.3	13	11.3	221	13.1
SA	1	0.4	0	0.0	301	30.3	15	13.0	317	18.7
Tas.	30	11.1	0	0.0	22	2.2	3	2.6	55	3.3
Vic.	107	39.5	46	14.9	221	22.3	16	13.9	391	23.1
WA	43	15.9	22	7.1	0	0.0	10	8.7	75	4.4

* 4 patients with disease with multiple subtypes included in total.

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

	Not admitted to intensive care unit		Admitted to intensive care unit		Total	
Total	1,481		211		1,692	
	n	%	n	%	n	%
Pregnancy	52	3.5	4	1.9	56	3.3
Nursing home resident	109	7.4	2	0.9	111	6.6
Restricted functional status	610	41.2	74	35.1	684	40.4
Medical comorbidities	1,252	84.5	181	85.8	1433	84.7
Chronic cardiac disease	511	34.5	61	28.9	572	33.8
Chronic renal disease	224	15.1	27	12.8	251	14.8
Chronic renal disease	589	39.8	111	52.6	700	41.4
Diabetes	383	25.9	45	21.3	428	25.3
Chronic liver disease	67	4.5	16	7.6	83	4.9
Immunosuppressed	279	18.8	31	14.7	310	18.3
Chronic neurological disease	260	17.6	20	9.5	280	16.5
Influenza vaccination	580/1,132	51.2	56/151	37.1	636/1,283	49.6
Influenza subtype						
A/H1N1/09pdm	216	14.6	55	26.1	271	16.0
A/H3N2	274	18.5	35	16.6	309	18.3
A/unsubtyped	885	59.8	108	51.2	993	58.7
B	102	6.9	13	6.2	115	6.8
Multiple strains	4	0.3	0	0.0	4	0.2
In hospital mortality	23/1,330	1.7	21/186	11.3	44/1,516	2.9

Presentation and treatment

For 1,546 patients with laboratory-confirmed influenza where the duration of symptoms was known, the median duration of symptoms prior to admission was 3 days (interquartile range (IQR): 2, 5 days). Of patients with influenza, 950 (56%) received oseltamivir; of these, 299 were known to have received oseltamivir within 48 hours of symptom onset. The duration of hospital stay was similar in patients that did not receive antivirals (median 4 days, IQR 2, 7 days), received antivirals within 48 hours of symptom onset (4 days, IQR 2, 7 days) or who received antivirals more than 48 hours after symptom onset (5 days, IQR 3, 8 days).

Admissions to intensive care

Of all cases, 179 patients (10.6%) were initially admitted to ICU and a further 32 (2%) were subsequently transferred to ICU after initial admission to a general ward. In a multivariate model, elderly patients and nursing home residents were associated with a lower risk of ICU admission in patients admitted to hospital with laboratory-confirmed influenza Table 3). In this model, medical comorbidities were associated with a higher risk of ICU admission and pregnancy was associated with a low risk of ICU admission, but these differences were not statistically significant. There were no significant differences in the risk of admission by influenza type.

Outcome

The mean length of hospital stay for all patients was 6.6 days. Admission to ICU was associated with an increase in mean hospital length of stay of 8.3 days compared with those not admitted to ICU; other factors associated with a prolonged length of stay included medical comorbidities and restricted pre-morbid functional capacity. Factors associated with a shorter length of stay included pregnancy and Indigenous ethnicity (Table 4).

Of the 1,516 patients where hospital mortality status was documented, 44 (2.9%) patients died, which included 21 patients in ICU. Of all in-hospital deaths, 32 (73%) were patients more than 65 years of age, 43 (98%) had medical comorbidities and 2 (1.9%) were Indigenous Australians. Significant medical comorbidities in patients who died following admission with laboratory-confirmed influenza were recorded as chronic cardiac disease (n=19), chronic respiratory disease (n=23), immunosuppression (n=11), diabetes (n=14) and renal disease (n=9).

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

Variable	Crude odds ratio (95% CI)	*P* value	Adjusted odds ratio (95% CI)	*P* value
Age >65 years	0.43 (0.31, 0.58)	<0.001	0.43 (0.31, 0.59)	<0.001
Medical comorbidities	1.10 (0.73, 1.66)	0.639	1.43 (0.94, 2.17)	0.099
Pregnancy	0.53 (0.19, 1.48)	0.227	0.37 (0.13, 1.05)	0.062
Indigenous Australian	1.12 (0.70, 1.78)	0.637	NI	
Restricted functional status	0.77 (0.57, 1.04)	0.091		
Nursing home resident	0.12 (0.03, 0.49)	0.003	0.16 (0.04, 0.68)	0.013
Influenza type				
Influenza A	1 (referent)			
Influenza B	0.89 (0.49, 1.61)	0.688	NI	

NI Not included in final model.

Table 4: Factors associated with length of hospital stay from presentation or diagnosis with influenza

Variable	Crude coefficient (95% CI)	*P* value	Adjusted coefficient (95% CI)	*P* value
Age >65 years	1.07 (0.19, 1.96)	0.017	0.79 (-0.03, 1.60)	0.059
Medical comorbidities	2.10 (1.28, 2.91)	<0.001	1.20 (0.37, 2.03)	0.004
Indigenous Australian	-1.92 (-2.81, -1.03)	<0.001	-1.44 (-2.19, -0.70)	<0.001
Pregnancy	-3.10 (-4.28, -1.91)	<0.001	-1.69 (-2.56, -0.82)	<0.001
Influenza B (vs Influenza A)	-0.13 (-1.52, 1.26)	0.854	NI	
RACF resident	1.33 (-0.15, 2.80)	0.079		
Restricted functional capacity	2.06 (1.32, 2.80)	<0.001	1.59 (0.95, 2.24)	<0.001
Intensive care admission	8.12 (6.40, 9.84)	<0.001	8.31 (6.53, 10.08)	<0.001

* Bootstrapped linear regression: baseline length of stay 3.7 days (representing mean length of stay in a non-elderly, non-Indigenous patient with no comorbidities or functional restriction, not admitted to intensive care unit).

NI Not included in final model.

RACF Residential aged care facility.

Discussion

Hospital-based sentinel surveillance provides timely and detailed information on the severity of illness, and complements community- and primary care-based surveillance systems that provide information on the extent of spread. Surveillance programs similar to FluCAN are operating in many countries.[6–10] The FluCAN system in Australia includes sites in all jurisdictions with representation from metropolitan and regional hospitals, specialist paediatric hospitals and those in tropical and subtropical regions. By collecting data on patients with acute respiratory illness who test negative for influenza, vaccine coverage (particularly in vulnerable patients) and vaccine effectiveness against severe influenza can be estimated from the same study.[11, 12]

In 2014, we recorded almost 1,700 admissions to the 15 hospitals that participated in this surveillance network, representing the highest number of admissions since surveillance commenced in 2009. Virological surveillance suggested influenza A(H1N1)pdm09 predominated across most jurisdictions throughout the season, however influenza A(H3N2) was predominant in New South Wales and the Australian Capital Territory.[4] Influenza B (B/Yamagata-lineage) was less common in this season than in 2013.[13] Due to differences in the number and size of sentinel hospitals in each jurisdiction, the relative number of cases between jurisdictions may not represent differences in true influenza incidence.

Compared with previous years, the 2014 season was slightly earlier (mid-August to early September

compared with mid-September in 2013). Patients admitted in 2014 (47% were over 65 years) were older than in 2013 (32% over 65 years), but had a similar age profile to those in 2012 (46% over 65 years).

In contrast to other studies, we found that the demographic profile and proportion with chronic comorbidities was similar in patients who were admitted to ICU compared with those admitted to the general wards.[14] Older patients were under-represented in ICU; this is likely to reflect a lower severity of illness, as older patients with mild disease may still require care in hospital. This may also reflect policies discouraging admission of the frail elderly into ICU if deemed futile.

We found that the length of stay was longer in those with more severe illness (ICU admission) and functional impairment. Interestingly, some risk groups (elderly, pregnant, Indigenous Australians) had a shorter length of hospital stay suggesting that the severity of illness is the primary driver for length of stay, rather than underlying risk factors. It may also reflect ascertainment bias in that patients with risk factors may have been more likely to be tested for influenza.

With a mean length of stay of 6.6 days, the patients with laboratory-confirmed influenza detected in this surveillance system represent over 9,870 bed days in the 15 sentinel hospitals. As the hospitals represented in this network represent approximately 12% of the national hospital bed capacity, the cases detected here are likely to represent approximately 15,000 admissions and almost 100,000 bed days nationally. Although the estimate of disease-attributable cost varies widely according to the method of calculation,[15] recent Australian costing data suggest that the direct hospital inpatient cost of admissions with confirmed influenza is approximately A$60 million to A$100 million based on accounting costs.[16] This is likely to represent a minimum estimate due to influenza case under-ascertainment, healthcare costs incurred following discharge and costs borne by other payers.

There are several limitations to this study. 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 season only.

In summary, we detected a large number of hospital admissions with laboratory-confirmed influenza in a national observational study in 2014 compared with previous years. A high proportion of patients with severe influenza, and almost all deaths, occurred in patients with chronic comorbidities. In admitted patients, younger age was associated with ICU admission, highlighting the importance of this under-vaccinated risk group.

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Flutracking weekly online community survey of influenza-like illness: 2013 and 2014

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Abstract

Flutracking is a national online community influenza-like illness (ILI) surveillance system that monitors weekly ILI activity and severity. This article reports on the 2013 and 2014 findings from Flutracking. From 2013 to 2014 there was a 14.0% increase in participants who completed at least 1 survey to 21,021 participants. By the end of the 2013 and 2014 seasons, respectively 59.7% and 59.1% of all participants had received the seasonal influenza vaccine. The 2013 Flutracking national ILI weekly incidence peaked in late August at 4.3% in the unvaccinated group, 1 week earlier than national counts of laboratory confirmed influenza. The 2014 Flutracking national ILI weekly incidence also peaked in late August at 4.7% in the unvaccinated group, in the same week as national counts of laboratory confirmed influenza. A lower percentage of Flutracking participants took 2 or more days off from work or normal duties in 2013 (peak level 1.6%) compared with 2014 (peak level 2.5%) and sought health advice in 2013 (peak level of 1.1%) compared with 2014 (peak of 1.6%). Flutracking ILI surveillance suggests that 2014 was a moderately more intense season than 2013 and similar to 2012. *Commun Dis Intell* 2015;39(3):E361–E368.

Keywords: influenza, surveillance, syndromic surveillance, influenza-like illness, survey, Flutracking

Introduction

There are a number of surveillance methods that contribute to influenza surveillance in Australia each year.[1] Integrating data from each of these systems is vital in creating a timely and accurate picture of influenza activity as each surveillance method has its strengths and limitations.[2] The Flutracking surveillance system makes an important contribution to Australian influenza surveillance by providing weekly community level influenza-like illness (ILI) attack rates that are not biased by health seeking behaviour, clinician testing practices or differences in jurisdictional surveillance methods.[3–6] The Flutracking surveillance system has been incorporated into the weekly Australian Influenza Surveillance Report since 2009.[1]

The main aims of Flutracking are to:

1. compare ILI syndrome rates between vaccinated and unvaccinated participants to detect inter-pandemic and pandemic influenza and provide early confirmation of vaccine effectiveness or failure;
2. provide consistent surveillance of influenza activity across all jurisdictions and over time; and
3. provide year to year comparison of the timing, incidence, and severity of influenza.

This article reports on 2013 and 2014 Flutracking ILI surveillance. We report on participation numbers compared with previous years, participant vaccination uptake for the seasonal influenza vaccine, field vaccine effectiveness (FVE) estimates, weekly ILI estimates and comparison of these estimates with Australian laboratory influenza notifications, and burden of illness estimates.

Methods

The Flutracking surveillance system was in operation for 26 weeks in 2013, from the week ending 28 April to the week ending 20 October 2013 and for 25 weeks in 2014 from the week ending 4 May to the week ending 19 October 2014. Unless Flutracking participants unsubscribe, the cohort of participants is maintained year to year and is boosted by an annual recruitment drive which usually runs from March to May. The recruitment methods in 2013 were similar to those used in 2007 to 2012 except for some enhanced social media promotions.[3] Recruitment in 2014 was focused on email and social media promotions. Recruitment took place from 23 April to 17 May 2013 and from 10 April to 19 May 2014.

The weekly survey questions have evolved from 2007 to 2012.[3,7] No changes were made to the survey questions for 2013 and 2014.

Participation and vaccination rate

Peak weekly participation numbers using the peak week of national ILI for each year were reported for 2013 and 2014 at the national and state or territory level. The participation rate (per 100,000) was calculated using participant number in the national peak ILI week and June 2014 Estimated Resident Population (ERP) for each state and territory from the Australian Bureau of Statistics.[8] Participation numbers were documented from 2006 to 2014.

The percentage of participants reporting fever, cough, fever and cough, and fever and cough and sore throat in the national peak week of ILI for 2013 and 2014 were compared (peak week determined using the number of participants with fever and cough in the unvaccinated group divided by the total number of participants for that week in the unvaccinated group).

Field vaccine effectiveness

FVE analyses for New South Wales participants 18 years of age or older were conducted for 2013 and 2014 using the same method as previously reported.[9]

The FVE was calculated for the peak influenza period defined as the 4 consecutive weeks for New South Wales participants aged 18 years or over with the highest weekly Flutracking ILI percentages for unvaccinated participants. Table 1 shows peak influenza periods used in yearly vaccine effectiveness calculations.

Weekly influenza-like illness prevalence and comparison with national laboratory influenza notifications

Weekly ILI prevalence is defined as the percentages of participants with both fever and cough. The unstratified (by vaccination status) ILI percentages were compared with national laboratory influenza notifications for 2009 to 2014.

Burden of illness (index of severity)

The weekly percentage of participants from 2011 to 2014 who had 1) fever and cough and two or more days off work or normal duties and 2) who visited a general practitioner, emergency department or stayed as a hospital inpatient due to fever and cough was calculated.

Table 1: Peak 4 week influenza periods used in yearly vaccine effectiveness calculations, New South Wales, 2007 to 2014

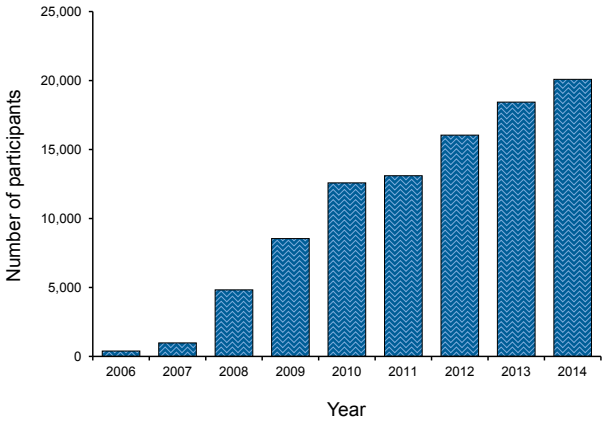
Year	Peak influenza period (week ending) for 18 years or over
2007	29 July – 19 August
2008	17 August – 7 September
2009	5 July – 26 July
2010	15 August – 5 September
2011	22 May – 12 June
2012	27 May – 17 June
2013	18 August – 8 September
2014	10 August – 31 August

Results

Participation and recruitment in 2013 and 2014

Of the 16,831 participants who completed a survey during the first 4 survey weeks of 2013, 43.1% completed all available surveys, and 69.7% of participants completed more than 90% of available surveys. Of the 19,740 participants who completed a survey during the first 4 survey weeks of 2014, 61.1% completed all available surveys, and 79.7% of participants completed more than 90% of available surveys. There were 20,087 participants who completed at least 1 survey in 2014, compared with 18,440 in 2013 (an 8.9% increase) (Figure 1). At the state and territory level, increases in peak week participation were most marked in the Australian Capital Territory, New South Wales and Western Australia. In 2013 and 2014, Tasmania had the highest Flutracking participation rate, followed by the Northern Territory and South Australia (Table 2).

Figure 1: Number of participants who completed at least one survey, Australia, 2006 to 2014, by year



The most successful recruitment strategy in 2013 and 2014 was recruitment through previous participants. On 22 April 2013, a *Welcome Back to Flutracking* email was sent to all active participants with a suggestion that participants invite 3 people to join the survey. On 22, 23 and 24 April, respectively 1,071, 263, and 290 participants enrolled. There were an additional 106 participants recruited on 6 May: this spike corresponds to the date the first Flutracking survey email was sent to participants and a Flutracking media release on 'Man-flu' based on an analysis of severity of ILI symptoms by gender on 7 May (Figure 2).[10]

A total of 529 organisations were invited to participate in Flutracking in 2013. Of these, 206 agreed

Table 2: Recruitment to Flutracking, 2013 and 2014, by state or territory

State or territory	2013		2014		
	Number of respondents (peak week)	Rate of Flutracking participation per 100,000 population	Number of respondents (peak week)	Percentage positive change	Rate of Flutracking participation per 100,000 population
ACT	526	136.3	662	25.9	171.5
NSW	5,163	68.7	6,357	23.1	84.6
NT	669	272.9	801	19.7	326.8
Qld	1,528	32.4	1,726	13.0	36.5
Tas.	1,775	344.8	2,012	13.4	390.8
SA	2,651	157.3	2,840	7.1	168.5
Vic.	2,398	41.0	2,844	18.6	48.7
WA	869	33.8	1,045	20.3	40.6
Total	15,579	66.3	18,287	17.4	77.8

Figure 2: Significant Flutracking recruitment events and impact, 2013

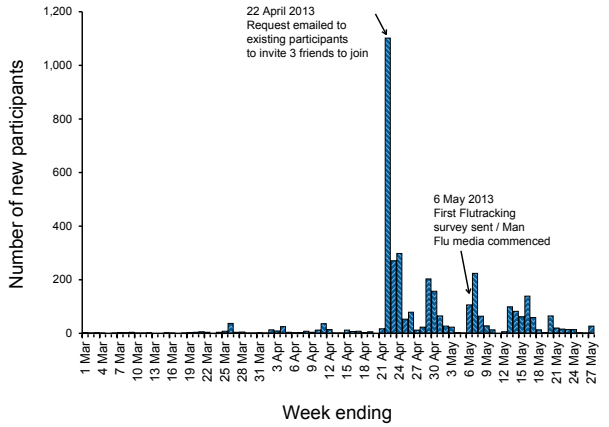
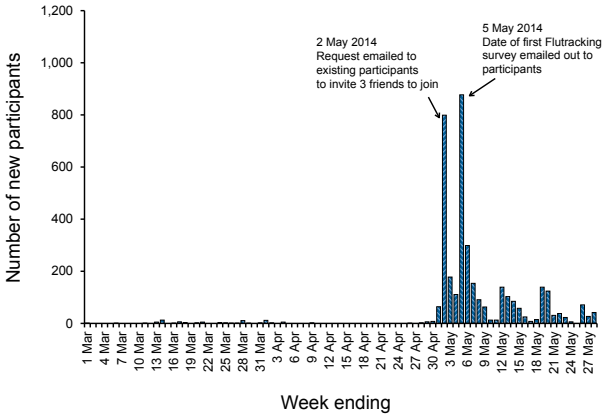


Figure 3: Significant Flutracking recruitment events and impact, 2014



to be emailed additional information regarding employee invitations to the survey, 29 accepted the invitation and agreed to invite their employees and 24 declined.

On 2 May 2014, an email to participants with a request to invite 3 friends (similar to 2013) was sent. On 2, 3, and 4 May, respectively 799, 178, and 111 participants enrolled. There were an additional 877 participants recruited on 5 May 2014: this spike corresponds to the date the first Flutracking survey email was sent to participants (Figure 3).

Facebook advertising and engagement with participants via emailed survey links and the survey website resulted in an increase in page 'likes' from 69 'likes' (4 April 2013) to 992 'likes' (21 May 2013) and an increase from 1,136 'likes' (30 April 2014) to 2,432 'likes' (23 May 2014).

As a result of the above recruitment strategies and media coverage a total of 4,446 participants joined the survey in 2013 (an 11.9% increase from 2012) and 5,085 joined the survey in 2014 (a 14.4% increase from 2013).

Socio-demographic characteristics

Of the participants who completed at least 1 survey and responded to each of the demographic questions, almost two thirds (63% and 61% in 2013 and 2014 respectively) were aged 35–64 years, and almost two-thirds (63% and 61% in 2013 and 2014 respectively) were female. Sixty per cent in 2013 and 59% in 2014 had completed a bachelor degree, graduate diploma/certificate or postgraduate degree, and 1.3% in 2013 and 1.4% in 2014 identified as Aboriginal and/or Torres Strait Islander (Table 3).

Percentage of participants vaccinated

Seasonal vaccination levels were higher in 2013 and 2014 than most prior years. By the end of the 2014 season (week ending October 19, 2014),

Table 3: Socio-demographic characteristics of Flutracking participants, 2012 to 2014

Age (years)	2012		2013		2014	
	Frequency	Per cent	Frequency	Per cent	Frequency	Per cent
0–15	1,854	11.6	2,081	11.3	2,638	12.6
16–34	2,902	18.1	3,258	17.7	3,754	17.9
35–49	4,544	28.3	5,016	27.2	5,405	25.7
50–64	5,623	35.0	6,538	35.5	7,311	34.8
65 or over	1,123	7.0	1,547	8.4	1,909	9.1
Total participants	16,046	100.0	18,440	100.0	21,017	100.0
Gender						
Male	4,882	36.4	6,097	37.0	7,461	38.6
Female	8,516	63.6	10,386	63.0	11,867	61.4
Total reported	13,398	100.0	16,483	100.0	19,328	100.0
Education						
Year 10 or below (or equiv)	918	7.2	1,070	7.3	1,325	7.9
Year 11 (or equivalent)	392	3.1	451	3.1	529	3.1
Year 12 (or equivalent)	912	7.2	1,071	7.3	1,299	7.7
Certificate I/II/III/IV	1,211	9.5	1,347	9.2	1,563	9.3
Advanced diploma/Diploma	1,190	9.3	1,363	9.3	1,538	9.1
Enrolled bachelor degree	428	3.4	494	3.4	612	3.6
Completed bachelor degree	2,871	22.5	3,328	22.8	3,801	22.6
Grad diploma/Grad certificate	1,762	13.8	2,017	13.8	2,265	13.4
Postgraduate degree	3,071	24.1	3,477	23.8	3,921	23.3
Total reported (15 years or over only)	12,755	100.0	14,618	100.0	16,853	100.0
Aboriginal and/or Torres Strait Islander						
Yes	102	1.2	167	1.3	235	1.4
No	8,698	98.8	13,033	98.7	16,309	98.6
Total reported	8,800	100.0	13,200	100.0	16,544	100.0

58.5% (9,742/16,642) of participants had received the 2014 seasonal vaccine, compared with 59.7% (8,939/14,968) of participants by the end of 2013. Of the 3,418 participants who identified as working face-to-face with patients in 2014, 2,697 (78.9%) received the vaccine compared with 77.9% by the end of 2013. In 2014, 14.2% of participants less than 10 years of age whose parents completed a survey on their behalf were vaccinated with the seasonal influenza vaccine by the end of the season, compared with 18.3% in 2013 (Table 4).

Percentage of participants with influenza-like illness symptoms

Of participants who completed a survey in the national peak week of ILI for 2014, 4.7% reported fever and cough compared with 3.6% in 2013 and 4.7% in 2012. Of participants who completed at least 1 survey in the national peak 4 weeks of ILI for 2014 12.4% reported fever and cough, compared with 9.6% in 2013 and 12.1% in 2012 (Table 5).

Field vaccine effectiveness

From 2007 to 2014 our field FVE calculation for New South Wales participants demonstrated that the seasonal vaccine was effective against all reported ILI except in 2009 during the pandemic and in 2014. The FVE calculated for 2014 was less than the 2009 FVE estimate (Figure 4).

Detection of influenza-like illness

Figure 5 shows the 2009 to 2014 weekly ILI prevalence by vaccination status. Peak ILI activity for 2014 was during the week ending 24 August (5.0% in the unvaccinated group). However, divergence between the vaccinated and unvaccinated participants' ILI prevalence was highest during the week ending 29 June. Peak ILI activity for 2013 was during the week ending 25 August (4.3% in the unvaccinated group). This is also when divergence between the vaccinated and unvaccinated participants' ILI prevalence was highest.

Table 4: Number and percentage of participants vaccinated with the seasonal influenza vaccine at the final survey of each year, by participant characteristics

Participant group	2007	2008	2009	2010	2011	2012	2013	2014
All participants								
Received vaccine (%)	52.9	50.8	58.4	54.0	56.1	54.2	59.7	58.5
Number of participants	726	3,921	6,753	9,934	10,899	13,196	14,968	16,642
Participants working face to face with patients								
Received vaccine (%)	73.3	71.3	76.8	68.6	73.1	73.3	77.9	78.9
Number of participants	221	1,159	1,741	2,317	2,588	2,785	3,139	3,418
Participants less than 10 years of age								
Received vaccine (%)	N/A	18.2	18.5	16.1	9.4	12.1	18.3	14.2
Number of participants	N/A	77	200	360	466	730	905	1,151

Table 5: Percentage of participants with influenza-like illness symptoms in the peak week of influenza-like illness and peak 4 weeks of influenza-like illness, Australia, 2012 to 2014

Influenza-like illness symptoms	Peak influenza-like illness week						Peak 4 weeks of influenza-like illness					
	2012*		2013†		2014‡		2012§		2013‖		2014¶	
	n	%	n	%	n	%	n	%	n	%	n	%
Fever	785	5.7	742	4.8	1,067	5.8	2,206	14.9	2,130	12.5	2,920	15.2
Cough	2,254	16.4	2,208	14.2	2,957	16.2	4,814	32.4	4,816	28.3	6,212	32.4
Fever and cough	646	4.7	558	3.6	852	4.7	1,795	12.1	1,634	9.6	2,385	12.4
Fever, cough and sore throat	449	3.3	430	2.8	618	3.4	1,377	9.3	1,263	7.4	1,775	9.3

* Week ending 15 July 2012, N= 13,707

† Week ending 25 August 2013, N=15,579

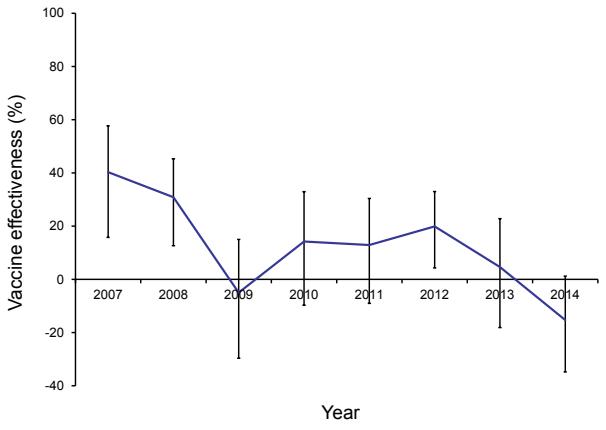
‡ Week ending 24 August 2014, N=18,287

§ Weeks ending 1 to 22 July 2012, N=14,851

‖ Weeks ending 11 August to 1 September 2013, N=16,988

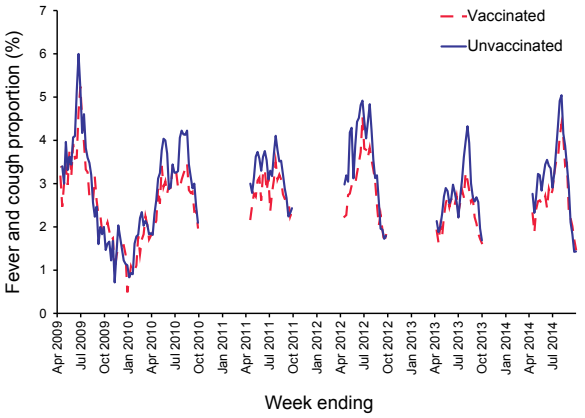
¶ Weeks ending 10 August to 31 August 2014, N=19,188.

Figure 4: Field vaccine effectiveness against all influenza-like illness for peak 4 weeks for participants greater than 18 years of age, New South Wales, 2007 to 2014



Vertical bars represent 95% confidence intervals.

Figure 5: National fever and cough percentage stratified by vaccination status, 2009 to 2014, by week



The levels of ILI seen in 2014 were similar to the 2012 season. The 2013 season was milder than levels of ILI seen in 2014, 2012, and similar to levels of ILI seen in 2011 and 2010.

Comparison with national laboratory influenza notifications

There was an increase in the number of laboratory confirmed cases of influenza from 2,381 notifications in the peak week of 2013 to 7,170 notifications in the peak week of 2014. The peak weekly percentage of Flutracking participants with ILI for 2014, unstratified by vaccination status, was 4.7% compared with 3.6% in 2013 (Figure 6). In 2014 the timing of the peak week of Flutracking ILI levels was the same as the timing of the peak week of laboratory notifications of influenza. However, in 2013 the peak week of Flutracking ILI levels was 1 week earlier than the peak week of laboratory notifications of influenza.

Index of severity

There were higher percentages of participants taking time off work or normal duties in 2014 compared with 2013 (peak level of 2.5% in 2014 compared with a peak level of 1.6% in 2013) and seeking health advice (peak level of 1.6% in 2014 compared with a peak of 1.1% in 2013) (Figure 7).

Discussion

The number of participants completing at least 1 survey during the year continues to increase each year, and in each state and territory. This is welcome as it is resulting in higher population participation rates and broader geographic reach.

Direct marketing of Flutracking via telephone to organisations to invite the participation of their members/employees is becoming less relevant as the size of the cohort increases. It is more efficient to request Flutracking participants to invite their friends and colleagues to join and this has been very successful – Flutracking is now the largest weekly survey of influenza-like illness in the world.

Flutracking participants continue to be predominantly of working age and higher educational status. While a decreased reliance on workplace recruitment will assist to broaden the participant base, requesting existing participants to recruit their friends and colleagues will likely result in them recruiting people from their own demographic base.

The FVE calculated for both 2013 and 2014 demonstrated low vaccine effectiveness compared with 2010, 2011 and 2012. The 2014 FVE estimate

Figure 6: National fever and cough prevalence, April through October (not stratified by vaccination status) compared with national influenza laboratory notifications, 2007 to 2014, by week

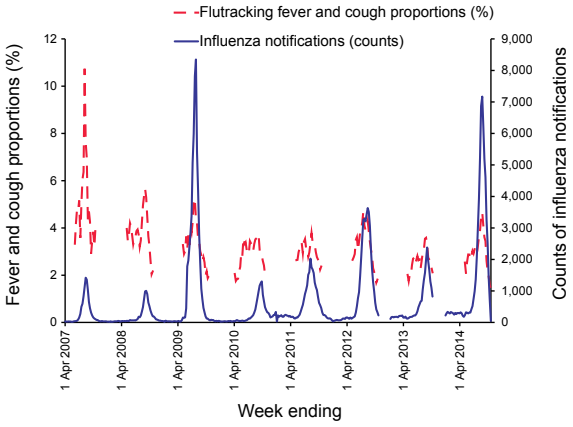
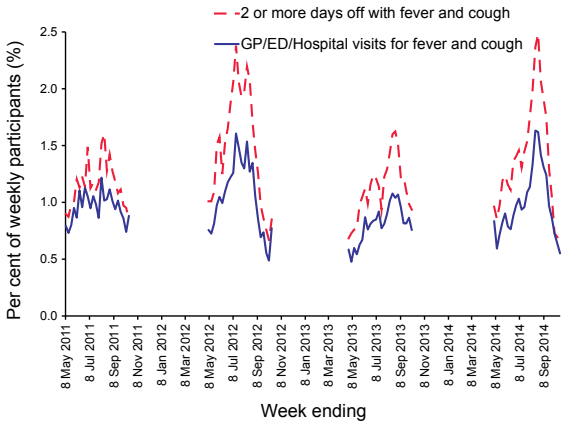


Figure 7: Weekly index of severity: Participants with fever and cough plus two or more days off work, and participants who sought medical advice from a general practitioner, emergency department or as a hospital inpatient, Australia, May 2011 to October 2014



was even lower than that calculated in 2009. New South Wales experienced predominantly A(H3N2) strains of influenza in 2014.[11] The low 2014 FVE estimate may reflect a decreased match between circulating viruses and the 2014 A(H3N2) vaccine strain.[12,13] A symptom based case definition, such as that used by Flutracking, cannot be expected to provide the same quantitative estimates of a laboratory confirmed outcome. Despite its limitations, it appears to concur, at least qualitatively, with conventional test negative vaccine effectiveness methods. The main benefit of Flutracking's FVE calculations may be that of offering a rapid qualitative indication of FVE as was provided during the 2009 influenza pandemic.

Based on Flutracking fever and cough weekly percentages, the community attack rates in the 2014 season were higher than the attack rates in the 2013 season, but similar to the 2012 attack rates. National influenza laboratory notifications also showed an increase in cases of influenza from 2013 to 2014. This increase was much larger than the increase seen in the Flutracking ILI percentages, which may reflect the ongoing increases in laboratory testing occurring year to year. Flutracking burden of illness data suggested higher levels of ILI in the community in 2014 compared with 2013, with higher rates of time off normal duties and health care seeking behaviour. Influenza A(H1N1)pdm09 re-emerged in 2013 and represented over 15% of all influenza notifications nationally, compared with <1% of notifications in 2012, and the proportion of influenza B was higher in 2013 than in recent years.[14] Additionally in 2013, influenza A(H1N1)pdm09 was the predominant subtype in New South Wales and other eastern jurisdictions, where a large proportion of Flutracking participants reside. Influenza A(H1N1)pdm09 also predominated across most states and territories throughout the 2014 season. However, in New South Wales and the Australian Capital Territory A(H3N2) was predominant, and there were late increases in A(H3N2) notifications in Queensland, Western Australia, Tasmania and the Northern Territory.

With an increasing number of participants across each jurisdiction each year, Flutracking is becoming a more reliable comparison measure of ILI timing and severity between Australian jurisdictions. Flutracking is aiming to further improve the representativeness of the data through targeted recruitment strategies to increase the proportions of participants in the 0–15 years age group and 65 years or over age group, Aboriginal and Torres Strait Islander participants, and participants in regional areas of Australia.

Nationally, in 2014 Flutracking contributed to the influenza surveillance by balancing the high levels of laboratory influenza notifications seen in 2014 with community level data showing more moderate levels of ILI. Increased laboratory testing may have been responsible for as much as two-thirds of the reported year over year increase in laboratory notifications.[15] Flutracking and other syndrome surveillance systems can provide situational awareness to assist with the interpretation of the more specific influenza surveillance provided by laboratories

Authors' contributions

Sandra Carlson led the writing of the manuscript, Lisa McCallum and Sandra Carlson both contributed to the statistical analysis, Craig Dalton conceived and designed the project, oversaw the statistical analysis, contributed to and oversaw writing of the manuscript, Michelle Butler also contributed to the statistical analysis, John Fejsa, contributed to the design of the project and had primary responsibility for the online software and database development, as well as questionnaire design, Elissa Elvidge contributed to the daily operational running of the system in 2013, David Durrheim contributed to the design of the project, statistical analysis, and writing of the manuscript.

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Surveillance of adverse events following immunisation in Australia annual report, 2013

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Abstract

This report summarises Australian passive surveillance data for adverse events following immunisation (AEFI) for 2013 reported to the Therapeutic Goods Administration (TGA) for 2013 and describes reporting trends over the 14-year period 1 January 2000 to 31 December 2013. There were 3,161 AEFI records for vaccines administered in 2013. This is an annual AEFI reporting rate of 13.9 per 100,000 population, the 2nd highest since 2000 and an increase of 59% compared with 2012 (1,994 AEFI records; 8.8 per 100,000 population). The increase was partly due to implementation of enhancements to vaccine safety reporting. This included stimulated reporting of AEFI as part of the extension of national human papillomavirus (HPV) vaccination under the National Immunisation Program to males aged 12–13 years, along with a catch-up program for males aged 14 and 15 years in February 2013 (n=785; includes males and females), in which certain events, such as syncope, were closely monitored. Eighty-two per cent (n=341/414) of the syncope reports were following HPV vaccination and of these 57% (n=195) were males and 43% (n=146) were females. In addition, reporting rates for most other the vaccines were higher in 2013 compared with 2012. The majority of AEFI reports described non-serious events while 5% (n=158) were classified as serious. There were 4 reports of death; however, all deaths were investigated by the TGA and no clear causal relationship with vaccination was found. The most commonly reported reactions were injection site reaction (13%), rash (10%), pyrexia (8%), and syncope (7%). *Commun Dis Intell* 2015;39(3):E369–E386.

Keywords: AEFI, adverse events, vaccines, surveillance, immunisation, vaccine safety

Introduction

This report summarises national passive surveillance data for adverse events following immunisation (AEFI) reported to the Therapeutic Goods Administration (TGA) by 28 February 2014. The report focuses on AEFI reported for vaccines administered during 2013 and trends in AEFI reporting over the 14-year period 1 January 2000 to 31 December 2013.

An adverse event following immunisation is defined as any untoward medical occurrence that follows immunisation and which does not necessarily have a causal relationship with the usage of the vaccine.[1] The adverse event may be any unfavourable or unintended sign, abnormal laboratory finding, symptom or disease.[1]

Thus, AEFI may be caused by a vaccine(s) or may be coincidental. Adverse events may also include conditions that occur following the incorrect handling and/or administration of a vaccine(s). The post-marketing surveillance of AEFI is particularly important to detect signals of rare, late onset or unexpected events, which are difficult to detect in pre-registration vaccine trials.

Reports summarising national AEFI surveillance data have been published regularly since 2003.[2–13] Trends in reported adverse events following immunisation are heavily influenced by changes to vaccine funding and availability provided through the National Immunisation Program (NIP). These changes impact on the interpretation of trend data and have been described in detail in previous reports published regularly since 2003.[2–13] These are listed in Table 1 in chronological order. Recent changes that impact on AEFI surveillance data presented in this report are:

1. In February 2013, the National HPV Vaccination Program (quadrivalent HPV vaccine Gardasil® – CSL Biotherapies/Merck & Co. Inc.) was extended to males aged 12–13 years through the school-based program, including a 2 year catch up program for males aged 14–15 years until the end of 2014.
2. From July 2013, the 2nd dose of MMR vaccine, previously given at 4 years, was brought forward to 18 months of age and delivered as a combination MMRV vaccine.

- From July 2013, combined *Haemophilus influenzae* type b (Hib) and meningococcal serogroup C (MenC) vaccine, Menitorix®, was listed on the NIP replacing the separate administration of monovalent meningococcal C conjugate vaccine (MenCCV) and Hib vaccine previously scheduled at 12 months of age.

To assist readers, at the end of this report there is a glossary of the abbreviations of the vaccines referred to in this report.

Table 1: Changes to the Australian Standard Vaccination Schedule (2003–2013)[2–15]

Date	Intervention
2003	Commencement of the meningococcal C conjugate vaccine (MenCCV) immunisation program. 18-month dose of DTPa vaccine removed from the National Immunisation Program.
2004	dTpa funded at 15–17 years of age replacing the diphtheria-tetanus dose.
2005	From January 2005, universal funded infant 7-valent pneumococcal conjugate vaccine (7vPCV) program replaced the previous targeted childhood program, with a catch-up program for children aged <2 years. Universal 23-valent pneumococcal polysaccharide vaccine (23vPPV) for adults aged ≥65 years replaced previous subsidy through the Pharmaceutical Benefits Scheme. From November 2005, universal funded immunisation against varicella at 18 months of age with a school-based catch-up program for children at 10–13 years of age not previously vaccinated and without a history of varicella infection (no funded catch-up for children 2–10 years of age). IPV funded to replace OPV, in combination vaccines.
2007	From April 2007, funded immunisation against human papillomavirus for all Australian girls aged 12–13 years delivered through a school-based program from April 2007, with a temporary catch-up program through schools or primary care providers for females aged 13–26 years until December 2009. From July 2007, universal funded immunisation against rotavirus at 2 and 4 months of age (Rotarix®) or at 2, 4 and 6 months of age (Rotateq®).
2008	Western Australia commenced a seasonal influenza vaccination program for all children aged 6 months to <5 years (born after 1 April 2003). In March 2008, Queensland, South Australia and Victoria changed from using 2 combination vaccines (quadrivalent DTPa-IPV and Hib-HepB) to the single hexavalent DTPa-IPV-HepB-Hib vaccine.
2009	By late 2009, all states and territories were using the single hexavalent DTPa-IPV-Hib-HepB (Infanrix hexa®) vaccine for all children at 2, 4 and 6 months of age, due to an international shortage of *Haemophilus influenzae* type b (Hib) (PedvaxHib® [monovalent] and Comvax® [Hib-HepB]) vaccines. Pandemic H1N1 2009 influenza vaccine (Panvax®) was rolled out across Australia from 30 September 2009 for people aged ≥10 years. From December 2009, the pandemic vaccine was made available to children aged 6 months to 10 years.
2010	Annual vaccination with seasonal trivalent influenza vaccine (TIV, containing 3 influenza strains: A/H1N1, A/H3N2 and B) was funded under the National Immunisation Program (NIP) for people aged ≥6 months with medical risk factors (previously subsidised through the Pharmaceutical Benefits Scheme) and all Indigenous people aged ≥15 years (previously all Indigenous adults ≥50 years and 15–49 years with medical risk factors). On 23 April 2010, the use of the 2010 seasonal TIV in children <5 years of age was suspended by Australia's Chief Medical Officer due to an increased number of reports of fever and febrile convulsions post-vaccination. A subsequent investigation identified that Fluvax® and Fluvax junior® (CSL Biotherapies), but neither of the other 2 available brands registered for use in young children, were associated with an unacceptably high risk of febrile convulsions. The recommendation to resume the use of seasonal influenza vaccine in children aged 6 months to 5 years, using brands other than Fluvax® and Fluvax junior®, was made in August 2010.
2011	From 1 July 2011, Prevenar 13® replaced Prevenar® on the NIP for children at 2, 4 and 6 months of age in all states and territories except the Northern Territory which adopted 13vPCV from 1 October 2011. 1 October 2011 to 30 September 2012 – all children aged between 12–35 months who had completed a primary pneumococcal vaccination course with 7vPCV were eligible to receive a free supplementary dose of Prevenar 13® On 25 March 2011, TGA issued a recall of Batch N3336 of the 23 valent pneumococcal polysaccharide vaccine 23vPPV, Pneumovax® 23. April 2011 – health professionals were advised not to administer a second or subsequent dose of Pneumovax 23 vaccine. December 2011 – Revised recommendations regarding which patients should be re-vaccinated under the NIP were provided.
2012	From 1 October 2012, a fourth dose of Prevenar 13®, (13vPCV, a 13-valent pneumococcal conjugate vaccine) was listed on the NIP for Indigenous children, aged 12–18 months, residing in Queensland, South Australia, Western Australia and the Northern Territory. This replaced the booster dose of Pneumovax23®, (23vPPV, a 23-valent pneumococcal polysaccharide vaccine) administered between 18 and 24 months of age for Indigenous children from these jurisdictions.
2013	From 1 February 2013, 4vHPV was extended to males aged 12–13 years, delivered through a school-based program, with a catch-up program for males aged 14–15 years in 2013 and 2014. From July 2013, the 2nd dose of MMR vaccine, previously given at 4 years, was brought forward to 18 months of age and delivered as a combination MMRV vaccine. From July 2013, combined *Haemophilus influenzae* type b (Hib) and meningococcal serogroup C (MenC) vaccine, Menitorix®, was funded for infants aged 12 months. This combination vaccine replaced the single dose of monovalent meningococcal C conjugate vaccine (MenCCV) and booster dose of monovalent Hib vaccine previously scheduled at 12 months of age.

Abbreviations of vaccine names are defined in the Appendix.

Methods

AEFI are notified to the TGA by state and territory health departments, health professionals, vaccine companies and members of the public.[14, 15] All reports are assessed using internationally consistent criteria[16] and entered into the Australian Adverse Drug Reactions System (ADRS) database. The TGA medical officers review all serious reports for drugs and vaccines. Reports are used in data mining and signal detection activities. Where there is insufficient information in a report to determine causality for a serious adverse event the TGA will contact the reporter on up to 3 occasions to elicit further information.

Adverse events following immunisation data

De-identified information on all AEFI reported to the TGA from 1 January 2000 to 31 December 2013 and stored in the ADRS database were released to the National Centre for Immunisation Research and Surveillance (NCIRS) in March 2014. Readers are referred to previous AEFI surveillance reports for description of the surveillance system.[2,5]

Records*contained in the ADRS database were eligible for inclusion in the analysis if a vaccine was recorded as 'suspected'† of involvement in the reported adverse event and either

(a) the vaccination occurred between 1 January 2000 and 31 December 2013, or

(b) for records where the vaccination date was not recorded, the date of onset of symptoms or signs occurred between 1 January 2000 and 31 December 2013.

Study definitions of adverse events following immunisation outcomes and reactions

AEFI were defined as 'serious' or 'non-serious' based on information in the report sent to the TGA and criteria similar to those used by the World Health Organization[16] and the US Vaccine Adverse Events Reporting System (VAERS).[17] In this report, an AEFI is defined as 'serious' if it meets one or more of the following criteria: (1) results in death; (2) is life-threatening; (3) requires inpatient hospitalisation or prolongation of existing hospitalisation; (4) results in persistent or significant disability/incapacity; (5) is a congenital anomaly/birth defect or; (6) is a medically important event or reaction.

Typically, each record lists several reaction terms that is symptoms, signs and/or diagnoses that have been coded by TGA staff from the reporter's description into standardised terms using the Medical Dictionary for Regulatory Activities (MedDRA®).[18,19]

In reports published previously, in order to analyse the data, MedDRA® coding terms were grouped to create a set of reaction categories that were broadly analogous to the reactions listed in previous *Australian Immunisation Handbooks*.[14,15] However, the methodological framework of reporting of adverse events have been recently reviewed by NCIRS in collaboration with TGA and a revised format for AEFI analyses using MedDRA preferred terms (PTs) was evaluated.[20] For this report, the new format using MedDRA PTs is used for data analysis. Grouping of reactions using PTs is more comparable with data from other countries and internationally accepted.[21–23] In conjunction with the new national vaccine-specific reporting form,[24] the use of PTs will allow better reflection of post-marketing surveillance data on vaccines in Australia.

Data analysis

All data analyses were performed using SAS software version 9.3.[25] Average annual population-based reporting rates were calculated for each state and territory and by age group using population estimates obtained from the Australian Bureau of Statistics.

Reporting rates per 100,000 administered doses were estimated where information was available on the number of doses administered. This was done for vaccines funded through the NIP for children aged less than 7 years.

Denominator data to estimate reporting rates for influenza and 23vPPV for people aged ≥ 18 years were obtained from a national adult coverage survey conducted in 2009.[26] For 23vPPV, the number of people vaccinated in 2013 was derived from the number of people in this survey who reported receipt of the vaccine within the previous 5 years, divided by five. The number of administered doses of each of the childhood vaccines was obtained from the Australian Childhood Immunisation Register (ACIR), a national population-based register of approximately 99% of children aged less than 7 years.[27]

* The term 'AEFI record' is used throughout this report because a single AEFI notification/report to the Office of Product review can generate more than one record in the ADRS database. This may occur if there is a time sequence of separate adverse reactions in a single patient, such as systemic and local reactions.

† Vaccines are classified as 'suspected' if the report contains sufficient information to be valid and the relationship between reported reactions and the vaccine is deemed at least possible.

Notes on interpretation

Caution is required when interpreting the data presented in this report. Due to reporting delays and late onset of some AEFI, the data are considered preliminary, particularly for the 4th quarter of 2013. Data published in previous reports for 2000–2012 may differ from that presented in this report for the same period because this report has been updated to include delayed notifications to the TGA that were not included in prior publications. Data can also differ because reports may be updated and recoded when follow-up information is received or when vaccine-specific analyses are conducted.

The information collated in the ADRS database is intended primarily for signal detection and hypothesis generation. While reporting rates can be estimated using appropriate denominators, they cannot be interpreted as incidence rates due to under-reporting and biased reporting of suspected events, and the variable quality and completeness of information provided in individual notifications.[2–13,28]

It is important to note that this report is based on vaccine information and MedDRA preferred terms collated in the ADRS database and not on comprehensive clinical notes or case reviews. The reported symptoms, signs and diagnoses in each AEFI record in the ADRS database are temporally associated with vaccination but are not necessarily causally associated with a vaccine or vaccines.

Comparison with online Database of Adverse Events Notifications

In August 2012, the TGA made a searchable database; the Database of Adverse Event Notifications (DAEN) available to the public on its website. DAEN contains reports of all adverse event reports for medicines and vaccines.[29] The data in this report have not been downloaded from DAEN. This annual report uses data from the ADRS database sent to NCIRS by TGA in March 2014, and includes more detailed data than are provided by DAEN. The numbers published in this report may be different to the numbers in the DAEN database due to different dates of data extraction and amendment to reports where further information has become available. In addition, this report provides several features that are not available from the DAEN database, including long-term trends and population and dose-based reporting rates, put in the context of changes in vaccine policy and use, and reporting practices.

Results

The ADRS database included a total of 3,161 records where the date of vaccination (or onset of adverse event, if vaccination date was not reported) was between 1 January and 31 December 2013.

In 2013, 88% of AEFI (n=2,768) were reported to the TGA via states and territories (except for Tasmania where all AEFIs are directly reported to TGA), while the rest were reported directly to the TGA by doctors or health care providers (7% n=231), members of the public (3% n=94), hospitals (1% n=30), and vaccine companies (1% n=38).

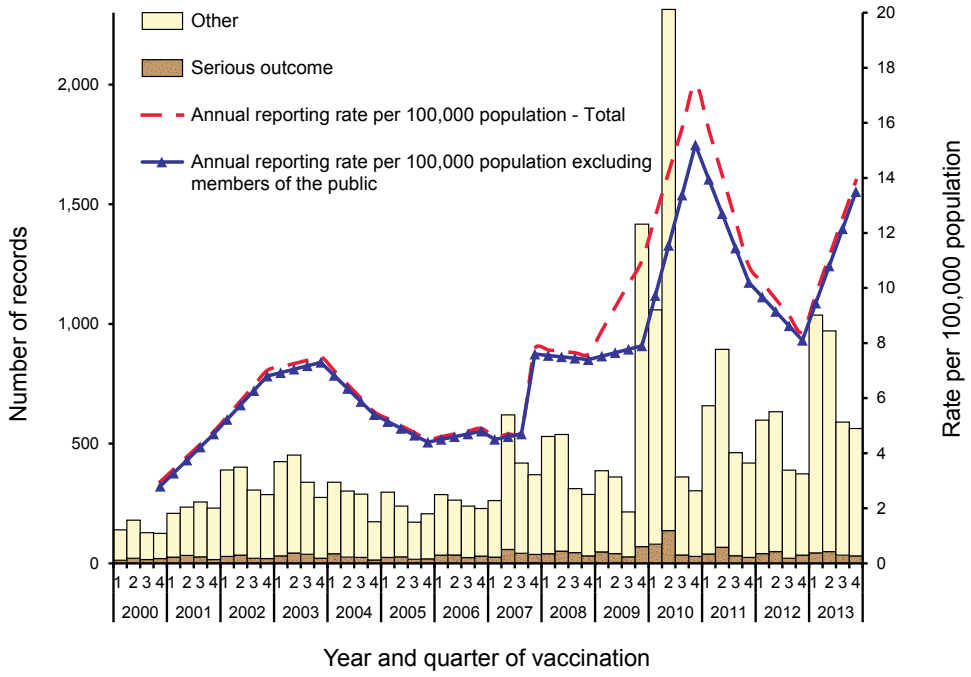
Reporting trends

The overall reporting rate for 2013 was 13.9 per 100,000 population compared with 8.4 per 100,000 in 2012. This was the 2nd highest rate in the 14-year period 2000–2013. The highest peak was observed in 2010 (17.4 per 100,000) predominantly due to reports in children following vaccination with the pandemic and 2010 seasonal trivalent influenza vaccines.[11]

The vast majority of reported events in 2013 (from all reporter types) were of a non-serious nature similar to the previous years (Figure 1).[9,10] Figures 2a, 2b and 2c demonstrate marked variations in reporting levels in association with previous changes to the NIP from 2000 onwards. The increase in reports in 2013 was predominantly due to an increase in reports following HPV vaccines in adolescents, and was associated with the extension of HPV vaccination to males (Figure 2a). An increase was observed in estimated reporting rates for the majority of vaccines in children aged less than 7 years in 2013 compared with 2012, but it was not statistically significant for rotavirus and varicella vaccines (Table 2, Figure 2c). The reporting rates for new NIP vaccines, MMRV and HibMenC, were 75.1 and 73.7 per 100,000 doses, respectively (Table 2, Figure 2b).

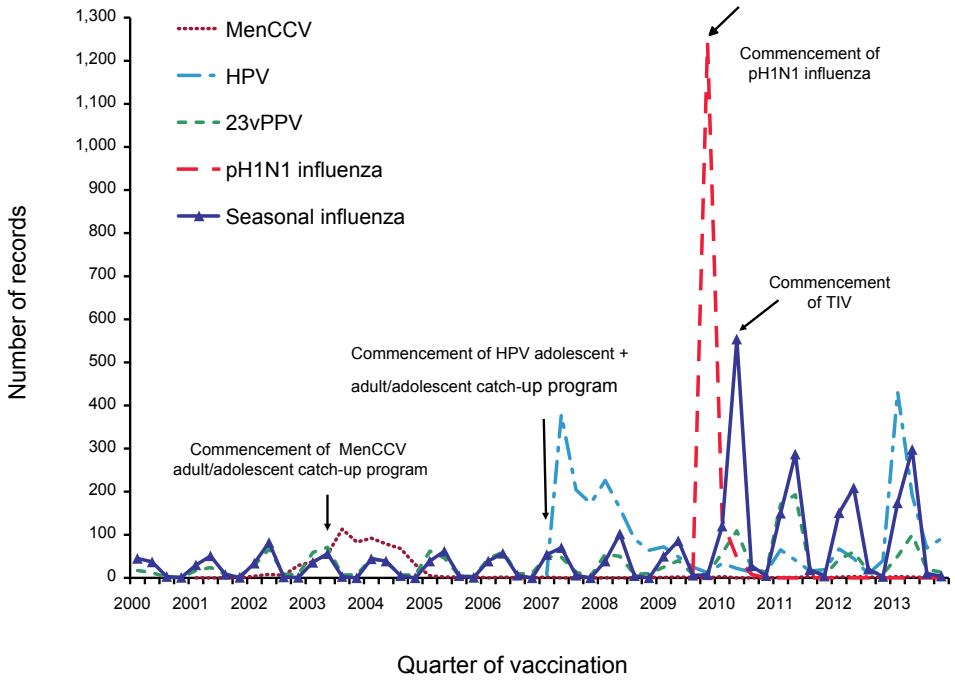
A seasonal pattern of AEFI reporting was apparent in 2013 as in previous years, with the highest number of AEFI notifications for vaccinations administered in the 1st half of the year (Figure 1). This corresponds to the months when influenza vaccine is given and older Australians receive 23vPPV (March to June). However, more AEFI reports following influenza vaccine were received in each of the last 4 years than years prior to 2009 (pre-pandemic era) (Figure 2a).

Figure 1: Adverse events following immunisation, ADRS database, 2000 to 2013, by quarter of vaccination



Note: For reports where the date of vaccination was not recorded, the date of onset or date event was reported to the Therapeutic Goods Administration was used as a proxy for vaccination date.

Figure 2a: Adverse events following immunisation for people aged ≥7 years in frequently reported vaccines, ADRS database, 2000 to 2013, by quarter of vaccination



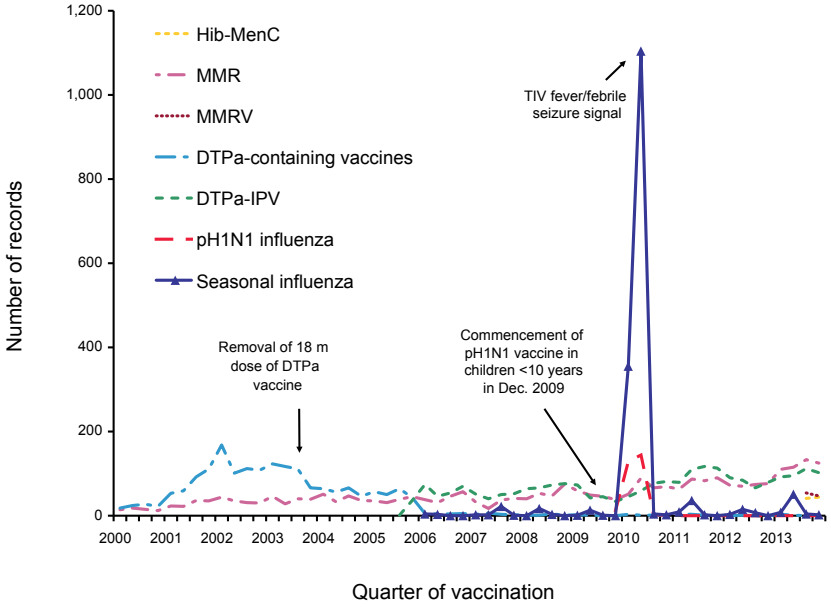
* Safety signal for fever and febrile convulsion found to be due to bioCSL Fluvax 2010 TIV in children.

Meningococcal C conjugate vaccine was introduced onto the National Immunisation Program schedule on 1 January 2003; pH1N1 influenza vaccine for children 6 months to 10 years on December 2009; pH1N1 vaccination for those ≥10 years commenced on 30 September 2009; seasonal trivalent influenza vaccine in 2010, which was an extension of existing adult and Indigenous programs to at-risk populations; and human papillomavirus program extended to boys in February 2013 (Table 1).

Abbreviations of vaccine names are defined in the Appendix.

Note: For reports where the date of vaccination was not recorded, the date of onset or date event was reported to Therapeutic Goods Administration was used as a proxy for vaccination date.

Figure 2b: Adverse events following immunisation for children aged 1 to <7 years in frequently reported vaccines, ADRS database, 2000 to 2013, by quarter of vaccination



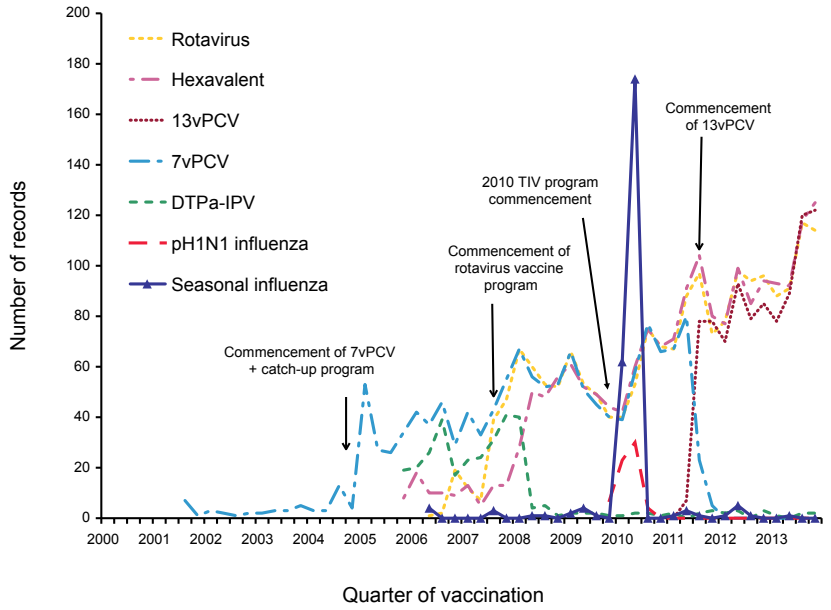
* Safety signal for fever and febrile convulsion found to be due to bioCSL Fluvax 2010 TIV in children.

DTPa-IPV was introduced onto the National Immunisation Program schedule in November 2005 replacing DTPa and OPV; seasonal trivalent influenza vaccine in 2010 which was an extension of existing adult and Indigenous programs to at-risk populations; MMRV and HibMenC vaccines on July 2013, and HPV program extended to boys in February 2013 (Table 1).

Abbreviations of vaccine names are defined in the Appendix.

Note: For reports where the date of vaccination was not recorded, the date of onset or date event was reported to Therapeutic Goods Administration was used as a proxy for vaccination date.

Figure 2c: Adverse events following immunisation for children aged <1 year, ADRS database, 2000 to 2013, by quarter of vaccination



* Safety signal for fever and febrile convulsion found to be due to bioCSL Fluvax 2010 TIV in children.

DTPa-IPV and DTPa-IPV-HepB-Hib (hexavalent) vaccines were introduced onto the National Immunisation Program schedule in November 2005; rotavirus (RotaTeq® and Rotarix®) vaccines on 1 July 2007; pH1N1 influenza vaccine for children 6 months to 10 years on December 2009; seasonal trivalent influenza vaccine in 2010 which was an extension of existing adult and Indigenous programs to at-risk populations; and the 13-valent pneumococcal conjugate vaccine (13vPCV) on 1 July 2011 (Table 1).

Abbreviations of vaccine names are defined in the Appendix.

Note: For reports where the date of vaccination was not recorded, the date of onset or date event was reported to Therapeutic Goods Administration was used as a proxy for vaccination date.

Table 2: Vaccine types listed as 'suspected' in records of adverse events following immunisation for 4 age groups, ADRS database, 2013

Vaccines*	AEFI records† (n)	Vaccine doses‡ (n)	Reporting rate per 100,000 doses§ (95% CI) 2013	2012
<7 years				
DTPa-containing vaccines	869	1,156,146	75.2 (70.3–80.3)	57.6 (53.3–62.1)
Hexavalent (DTPa-IPV-HepB-Hib)	461	857,760	53.7 (49.0–58.9)	40.2 (36.0–44.6)
DTPa-IPV	408	298,386	136.7 (123.8–150.7)	107.1 (95.7–119.5)
Measles-mumps-rubella	502	600,467	83.6 (76.4–91.2)	47.8 (42.4–53.8)
Pneumococcal conjugate - PCV	458	866,915	52.8 (48.1–57.9)	37.2 (33.5–41.2)
Rotavirus vaccine	415	537,413	77.2 (70.0–85.0)	62.9 (56.4–70.0)
Meningococcal C conjugate	107	184,674	57.9 (47.5–70.0)	26.6 (21.0–33.1)
Measles-mumps-rubella-varicella	102	135,832	75.1 (61.3–91.1)	na
Haemophilus influenzae type b	96	170,932	56.2 (45.5–68.6)	23.2 (18.0–29.5)
Hib-MenC	92	124,918	73.7 (59.4–90.3)	na
Seasonal influenza	66	na	na	na
Varicella	60	160,579	37.4 (28.5–48.1)	23.9 (18.5–30.3)
Total (<7 years)‖	1,423	3,937,876	36.1 (34.3–38.1)	23.5 (22.1–25.0)
7–17 years				
HPV	770	na	na	na
Hepatitis B	263	na	na	na
dTpa	198	na	na	na
Varicella	95	na	na	na
Seasonal influenza	41	na	na	na
Total (7–17 years)	961	na	na	na
18–64 years				
Seasonal influenza¶	334	3,170,300	10.5 (9.4–11.7)	8.6 (7.9–9.6)
dTpa	58	na	na	na
23vPPV¶	56	132,520	42.3 (31.9–54.9)	30.2 (21.6–41.1)
Total (18–64 years)**	549	3,302,820	11.8 (10.7–13.0)	9.4 (8.4–10.5)
≥65 years				
Seasonal influenza¶	93	2,176,000	4.3 (3.5–5.2)	3.6 (2.8–4.5)
23vPPV¶	114	317,400	35.9 (29.6–43.1)	24.3 (19.2–30.3)
dTpa	3	na	na	na
Total (≥65 years)**	192	2,493,400	8.3 (7.2–9.5)	6.2 (5.3–7.3)**

Abbreviations of vaccine names are defined in the Appendix.

* Records where at least one of the vaccines shown in the table was suspected of involvement in the reported adverse event.

† Number of AEFI records in which the vaccine was coded as 'suspected' of involvement in the reported adverse event and the vaccination was administered between 1 January and 31 December 2013. More than 1 vaccine may be coded as 'suspected' if several were administered at the same time.

‡ Number of vaccine doses recorded on the Australian Childhood Immunisation Register and administered between 1 January and 31 December 2013.

§ The estimated reporting rate per 100,000 vaccine doses recorded.

‖ Number of AEFI records excluding influenza vaccines.

¶ Number of administered doses of seasonal influenza vaccine estimated from the 2009 AIHW national adult vaccination survey.[22]

** Seasonal influenza and 23vPPV only.

Na Not applicable.

Age distribution

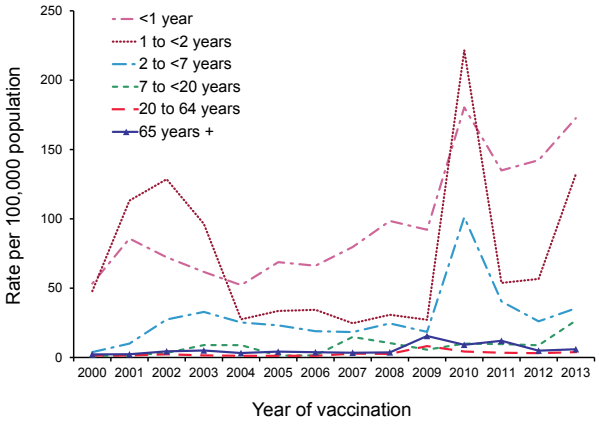
In 2013, the highest population-based AEFI reporting rate occurred in infants less than 1 year of age; the age group that received the highest number of vaccines (Figure 3). Compared with 2012, AEFI reporting rates in children increased substantially in all age groups but the magnitude differed: among the under 1 year age group, it increased 1.2-fold (from 142.3 to 172.6 per 100,000 population); in the 1 to less than 2 years age group it increased approximately 2-fold from 56.7 to 132.1; and in the 2 to less than 7 years age group the increase was 1.4-fold from 26.1 to 35.5 (Figure 3). In the 7 to less than 20 years age group the increase was 3-fold from 8.8 to 26.6 (Figure 3).

Reporting rates per 100,000 doses increased overall and for most individual vaccines in 2013 compared with 2012 (Table 2). This excludes influenza due to the absence of reliable dose data. This should be interpreted with caution due to the lack of recent denominator data and extrapolation of 2009 denominator data.

Geographical distribution

Population-based reporting patterns varied between states and territories during 2013 (Table 3) as in previous years.[2–13] The highest reporting rates were from the Australian Capital Territory, the Northern Territory, Victoria, and Western Australia (40.8, 28.1, 17.9, and 15.2 per 100,000, respectively) while Tasmania had the lowest rate (8.0 per 100,000). Reporting rates increased in most jurisdictions in 2013 compared with 2012 except Tasmania, which experienced a slight drop. The Australian Capital Territory observed a 3-fold increase followed by New South Wales and Queensland, which experienced a 2-fold increase compared with 2012.

Figure 3: Reporting rates of adverse events following immunisation per 100,000 population, ADRS database, 2000 to 2013, by age group and year of vaccination



Note: For reports where the date of vaccination was not recorded, the date of onset or date event was reported to Therapeutic Goods Administration was used as a proxy for vaccination date.

Table 3: Adverse events following immunisation records, ADRS database, 1 January to 31 December 2013, by state or territory

State or territory	AEFI records		Annual reporting rate per 100,000 population*		
	n	%	Overall	'Serious' outcome‡	Aged <7 years
Australian Capital Territory	153	5	40.8 (34.6–47.8)	0.8	8.5
New South Wales	641	20	8.8 (8.1–9.5)	0.3	2.5
Northern Territory	66	2	28.1 (21.7–35.7)	2.6	12.3
Queensland	692	22	15.2 (14.0–16.3)	0.8	6.9
South Australia	201	6	12.1 (10.5–13.9)	0.8	5.9
Tasmania	41	1	8.0 (5.7–10.9)	0.4	2.9
Victoria	1005	32	17.9 (16.8–18.9)	0.7	10.2
Western Australia	317	10	13.0 (11.6–14.5)	0.9	7.2
Other§	45	2	na	na	na
Total	3,161	100	13.9 (13.4–14.4)	0.7	6.3

* Average annual rates per 100,000 population calculated using mid-2013 population estimates (Australian Bureau of Statistics).

† See previous reports[2,3] for criteria used to assign causality ratings.

‡ AEFI records defined as 'serious' (i.e. recovery with sequelae, hospitalisation, life-threatening or death).

§ Records where the jurisdiction in which the AEFI occurred was not reported or was unclear. AEFI records in this category were notified mainly by pharmaceutical companies (n=38), general practitioners (n=5), members of the public (n=1) and nurses (n=1).

Vaccines

Thirty-one different vaccines were included in the 3,161 records received in 2013 (Table 4). The percentage of records where only 1 vaccine was reported as being the suspected vaccine differed by vaccine administered, typically varying according to whether multiple vaccines were routinely co-administered for the patient's age. There were slight variations in the numbers with outcomes defined as 'serious', which have remained low as in previous years.

The most frequently reported individual vaccine was HPV vaccine with 786 records (25%) followed by seasonal influenza vaccine with 552 records (17%), MMR (n=534; 17%), hexavalent DTPa-IPV-HepB-Hib (n=465; 15%) and 13vPCV (n=462; 15%), (Table 4).

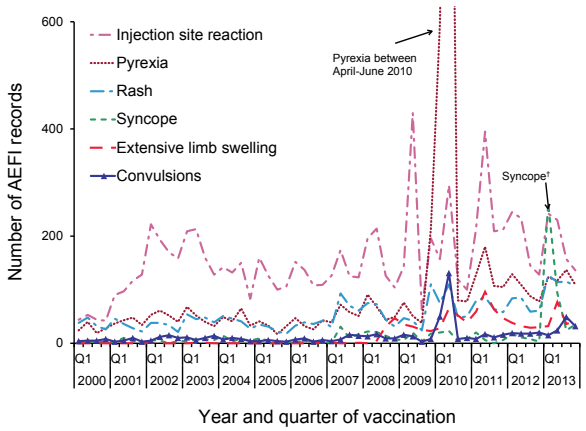
Reactions

In 2013, there was a total of 5,790 events reported for 3,161 AEFI records. The most frequently reported adverse events were injection site reactions (ISRs) (n = 765; 13%), rash (n = 561; 10%), pyrexia (n = 486; 8%), syncope (n = 414; 7%), and extensive swelling of vaccinated limb (n = 187; 3%) (Table 5, Figure 4). Some of the other reactions of interest were convulsions (n = 120; 2%), including 84 cases of febrile convulsion, hypotonic-hyporesponsive episode (HHE) (n=52; 1%), intussusception (n = 11; 0.2%), Guillain-Barré syndrome (GBS) (n = 6; 0.1%), and anaphylaxis (n = 1; 0.02%) (Table 5).

Of the total 765 cases of ISR, 364 (48%) were in children aged less than 7 years. The most commonly suspected vaccines for children aged less than 7 years related to ISR were: DTPa-IPV (n = 237); MMR (n=206); and hexavalent vaccine (n = 56) either given alone or co-administered with other vaccines. For those aged ≥7 years (n = 391), these were seasonal influenza vaccine (n = 158); 23vPPV (n = 70); HPV (n = 66); and dTpa (n = 65), either given alone or co-administered with other vaccines. As expected, reports of ISR associated with 23vPPV were predominantly in the ≥ 65 years age group (74%), while ISR associated with seasonal influenza vaccine was most commonly reported in those 18–64 years of age (69%). The dTpa vaccine was the vaccine most commonly recorded in association with ISR in records from those aged 7–17 years (51%) and 18–64 years (46%).

The number of reports in each reaction category has changed over time (Figure 4). Much of the variation in reporting of ISR related to specific changes in the immunisation schedules for vaccines that are known to have higher rates of ISR, including DTPa-containing vaccines, MenCCV, 23vPPV and HPV vaccine.[2–13,30,31] Increases in reports of fever were largely associated with time periods when new vaccines were added to the NIP in the reporting period, such as PCV 7 and HPV; the extension of seasonal influenza vaccine on the NIP to include persons less than 65 years of age at high risk of influenza in 2010; 13vPCV replaced 7vPCV in July 2011; and the extension of HPV to males in 2013.

Figure 4: Selected frequently reported adverse events following immunisation, ADRS database, 2000 to 2013, by year and quarter of vaccination



* Associated with administration of bioCSL Fluvax 2010 TIV and associated stimulated reporting.

† The peak in syncope coincided with the enhanced human papillomavirus surveillance program in which there was stimulated reporting of syncope for the first 6 months of 2013.

Abbreviations of vaccine names are defined in the Appendix.

Note: For reports where the date of vaccination was not recorded, the date of onset or date event was reported to Therapeutic Goods Administration was used as a proxy for vaccination date. Also, grouping for reactions are different for this report though these reactions have been mapped back to 2000 as mentioned in the Methods section.

The majority of the reports of pyrexia were following vaccination with MMR (n = 157), DTPa/HepB/IPV/Hib (n = 94), PCV (n = 87), and DTPa/IPV (n = 84) either given alone or co-administered.

The most commonly suspected vaccines for syncope were: HPV (n = 341); HepB (n = 151), 76% of which were co-administered with HPV vaccine; and dTpa vaccine (n = 86), 71% of which were co-administered with HPV vaccine.

Severity of outcomes

Summary data on outcomes are presented in Table 6. Eighty-three per cent of reported events

Table 4: Vaccine types listed as 'suspected' in records of adverse events following immunisation, ADRS database, 2013

Suspected vaccine type*	AEFI records	One suspected vaccine or drug only†		'Serious' outcome§		Age group‖			
						<7 years		≥7 years	
	n	n	%¶	n	%¶	n	%¶	n	%¶
HPV	786	396	50	20	3	2	0.3	780	99
Influenza	552	467	85	36	7	66	12	468	85
MMR	534	74	14	29	5	502	94	28	5
DTPa-IPV-HepB-Hib	465	14	3	44	9	461	99	4	1
PCV	462	15	3	43	9	458	99	4	1
Rotavirus	415	32	8	42	10	415	100	0	0
DTPa-IPV	414	170	41	8	2	408	99	6	1
Hepatitis B	312	44	14	9	3	8	3	300	96
dTpa	265	123	46	6	2	3	1	259	98
23vPPV	194	132	68	8	4	11	6	178	92
Varicella	163	66	40	6	4	60	37	103	63
MenCCV	114	6	5	11	10	107	94	5	4
MMRV	104	98	94	3	3	102	98	2	2
HibMenC	92	5	5	7	7	91	99	1	1
Hib	96	0	0	9	9	95	99	0	0
Hepatitis A	33	13	39	3	9	13	39	20	61
Typhoid	25	3	12	1	4	4	16	21	84
DTPa	22	8	36	1	5	9	41	13	59
dT	18	18	100	0	0	0	0	18	100
Rabies	16	10	63	1	6	2	13	14	88
Hepatitis A + B	14	7	50	1	7	0	0	14	100
Q fever	14	14	100	1	7	0	0	12	86
BCG	13	13	100	0	0	13	100	0	0
Hepatitis A-typhoid	13	5	38	1	8	0	0	13	100
Yellow fever	5	5	100	1	20	0	0	5	100
dTpa-IPV	4	1	25	0	0	0	0	4	100
Japanese encephalitis	4	0	0	0	0	0	0	4	100
IPV	3	2	67	2	67	0	0	3	100
Cholera	2	0	0	0	0	0	0	2	100
Men4PV	2	0	0	0	0	0	0	2	100
Tetanus	2	2	100	1	50	0	0	2	100
Total**	3,161	1,739	55	158	5	1,423	45	1,702	54

* Abbreviations of vaccine names are defined in the Appendix.

† Adverse events following immunisation (AEFI) records where only 1 vaccine was suspected of involvement in a reported adverse event.

‡ Causality ratings were assigned to AEFI records using criteria described previously.[2,3]

§ 'Serious' outcomes are defined in the Methods section.

‖ Includes only AEFI records where an age or date of birth has been reported.

¶ Percentages are calculated for the number of AEFI records where the vaccine was suspected of involvement in the AEFI, e.g. HPV was 'suspected' in 785 AEFI records; this was the only suspected vaccine in 50% of the 785 AEFI records, 3% were defined as 'serious' and 99% were for those aged ≥7 years.

** Total number of AEFI records analysed, not the total in each column as categories are not mutually exclusive and an AEFI record may list more than 1 vaccine.

Table 5: Selected reported adverse events and reactions of interest* classified by MedDRA preferred terms in records of adverse events following immunisation, ADRS database, 2013†

MedDRA preferred terms (Adverse events)	AEFI records n	Only reaction reported‡		'Serious' outcome§		Age group‖			
						<7 years		≥7 years	
		n	%¶	n	%¶	n	%¶	n	%¶
Injection site reaction**	765	415	54	15	2	364	48	391	51
Rash††	561	252	45	12	2	371	66	188	34
Pyrexia	486	34	7	33	7	315	65	161	33
Syncope	414	353	85	9	2	14	3	399	96
Vomiting	242	17	7	15	6	139	57	102	42
Extensive swelling of vaccinated limb	187	118	63	1	0.5	117	63	69	37
Headache	171	7	4	12	7	10	6	158	92
Nausea	154	2	1	7	5	11	7	143	93
Diarrhoea	131	17	13	7	5	103	79	28	21
Urticaria	131	67	51	4	3	70	53	60	46
Convulsions‡‡	120	89	74	25	21	107	89	12	10
Dizziness	117	24	21	2	2	2	2	115	98
Presyncope	116	75	65	1	1	8	7	105	91
Malaise	98	4	4	5	5	19	19	79	81
Lethargy	92	1	1	6	7	46	50	44	48
Irritability	70	7	10	3	4	68	97	2	3
Pallor	61	3	5	3	5	42	69	19	31
Myalgia	59	3	5	4	7	2	3	53	90
Erythema	56	10	18	1	2	37	66	19	34
Hypotonic-hyporesponsive episodes	52	44	85	8	15	52	100	0	0
Influenza like illness	52	22	42	2	4	6	12	42	81
Pruritus	49	8	16	0	0	10	20	39	80
Decreased appetite	46	0	0	2	4	28	61	17	37
Abdominal pain	42	2	5	4	10	23	55	19	45
Arthralgia	42	5	12	2	5	1	2	39	93
Chills	38	1	3	3	8	3	8	34	89
Cough	36	1	3	2	6	18	50	18	50
Paraesthesia	32	5	16	4	13	0	0	31	97
Fatigue	31	0	0	1	3	5	16	24	77
Screaming	30	3	10	4	13	30	100	0	0
Somnolence	30	3	10	2	7	25	83	5	17
Pain	29	1	3	1	3	9	31	19	66
Lymphadenopathy	28	8	29	1	4	6	21	22	79
Pain in extremity	28	9	32	2	7	3	11	25	89
Chest discomfort	27	1	4	3	11	0	0	27	100
Crying	27	5	19	0	0	27	100	0	0
Dyspnoea	26	1	4	4	15	6	23	19	73
Hypersensitivity	25	20	80	5	20	3	12	21	84
Hyperhidrosis	24	0	0	3	15	4	17	19	79
Oropharyngeal pain	23	0	0	1	4	5	22	18	78
Rhinorrhoea	23	0	0	0	0	19	83	4	17
Swelling face	22	4	18	3	14	8	36	13	59
Abdominal pain upper	20	2	10	4	20	3	15	17	85

Table 5 (cont'd): Selected reported adverse events and reactions of interest* classified by MedDRA preferred terms in records of adverse events following immunisation, ADRS database, 2013†

MedDRA preferred terms (Adverse events)	AEFI records n	Only reaction reported‡		'Serious' outcome§		Age group‖			
						<7 years		≥7 years	
		n	%¶	n	%¶	n	%¶	n	%¶
Local swelling	20	4	20	0	0	11	55	9	45
Guillain-Barré syndrome	6	5	83	4	67	0	0	5	83
Anaphylactic reaction	1	1	100	1	100	0	0	1	100
Encephalitis	1	0	0	1	100	0	0	1	100

* Selected reported adverse events reported during 1 January to 31 December 2013. Note: for injection site reaction, rash and convulsions, preferred terms (PTs) were grouped as described below.

† A complete list of adverse reactions as classified by individual Preferred Terms is available on request.

‡ Adverse events following immunisation (AEFI) records where only 1 reaction was reported.

§ 'Serious' outcomes are defined in the Methods section.

‖ Includes only AEFI records where an age or date of birth has been reported

¶ Percentages relate to the number of AEFI records in which the specific reaction term was listed, e.g. of 765 AEFI records listing injection site reaction, 54% listed only 1 type of reaction and 48% were for children aged <7 years.

** Injection site reaction includes the following MedDRA PTs: injection site reaction (400), injection site swelling (110), injection site pain (75), injection site mass (63), injection site erythema (63), injection site cellulitis (22), injection site rash (21), injection site induration (18), injection site abscess (11), injection site pruritus (9), injection site nodule (6), injected limb mobility decreased (6), injection site urticaria (6), injection site inflammation (5), injection site bruising (4), injection site infection (1), and injection site warmth (1).

†† Rash includes the following MedDRA PTs: rash (200), rash generalised (84), rash erythematous (60), rash pruritic (38), rash maculopapular (26), rash macular (24), rash vesicular (19), rash papular (13), rash morbilliform (4), and rash pustular (1).

‡‡ Convulsion includes the following MedDRA PTs: febrile convulsion (84), and convulsion (30), grand mal convulsion (5), and partial seizures (1).

Table 6: Outcomes of adverse events following immunisation, ADRS database, 2013

Outcome	AEFI records		Age group†			
			<7 years		≥7 years	
	n	%*	n	%‡	n	%‡
Non-serious	2,627	83	1,169	45	1,426	54
Not known (missing data)	376	12	164	44	210	56
Serious:	158	5	90	57	66	42
recovered with sequelae	3		–		3	
hospital treatment – admission	140		84		54	
life-threatening event	11		2		9	
death	4		4		–	
Total	3,161	100	1,423	45	1,702	54

* Percentages relate to the total number of adverse events following immunisation (AEFI) records (n=3,161).

† Includes only AEFI records where an age or date of birth has been reported

‡ Percentages relate to the number of AEFI records with the specific outcome, e.g. of 2,627 AEFI records with a 'non-serious' outcome, 45% were for children aged less than 7 years.

in 2013 were defined as 'non-serious'; 5% were defined as 'serious'; while in 12% severity could not be determined due to insufficient data (Table 6). This is similar to the proportions of serious AEFI in previous years.[9,11,12]

The reactions classified as 'serious' (n=158) were fever (n=33; 21%); convulsions (n=25; 16%), including 14 febrile convulsions; ISR (n=15; 9%); rash (n=10; 6%); syncope (n=9; 6%); HHE (n=8; 3%); diarrhoea (n=7; 4%); intussusception (n=6; 4%); GBS (n=4; 3%); and anaphylaxis (n=1; 1%). There were 4 reports of death (3%). Other relatively severe reactions which were not classified as 'serious', either because they did not satisfy the criteria, or due to a lack of information about the outcome

and/or hospitalisation status, included: convulsion (n=95; 79%), including 70 febrile convulsions; HHE (n=44; 85%); intussusception (n=5; 45%); and GBS (n=2; 33%).

All the reported cases of HHE (52) were in children aged less than 7 years and 79% (n=41) were in children aged less than 1 year. Of the 52 cases, 56% (n=29) were reported from Victoria. For the majority of the reports of HHE (n=37; 71%) the event followed co-administration of hexavalent, PCV and rotavirus vaccines. Of the 6 reported cases of GBS, three were following seasonal influenza vaccine (Fluvax®), one each was influenza vaccine (not otherwise specified), dTpa (Boostrix) and quadrivalent HPV vaccine (Gardasil) co-administered and MMR vaccine.

The only reported case of anaphylaxis was in a person over 18 years of age following seasonal influenza vaccine (Influvac®). The person recovered.

Of the 11 reports of intussusception, 9 (82%) were in infants (<1 year of age) following hexavalent, 13vPCV and rotavirus vaccines co-administered; 1 report was following rotavirus vaccine administered alone; and one was following typhoid and cholera vaccines co-administered.

Four deaths were recorded as temporally associated with receipt of vaccines.

- A 5-year-old child with multiple underlying medical problems who had received seasonal influenza vaccine (Fluarix®) 2 days prior to death.
- A 15-month-old child who had received Hib, MenC and MMR vaccines 9 days prior to death.
- A one-year-old child who had received Hib, MenC and MMR vaccines 5 days prior to death.
- A 2-month-old child who received hexavalent, 13vPCV and rotavirus vaccines 1 day prior to death.

All deaths were investigated by the TGA and no clear causal relationship with vaccination was found.

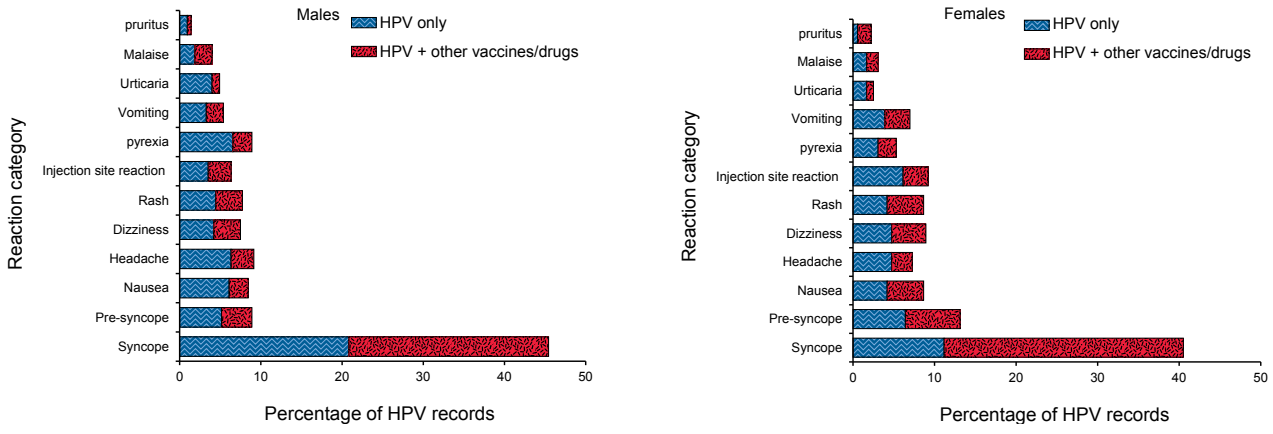
New national immunisation schedule vaccines

Human papillomavirus vaccine

Of the 786 AEFI reports for HPV vaccine in 2013, there were 779 reports for which age was known and all were in the age range 11–30 years. Twenty of these cases were coded as serious. Fifty-four per cent of cases were reported in males with 46% from females. HPV vaccine was the only suspected vaccine in 396 records (50%) (Table 4). Thirty-one per cent (n=245) of cases were reported from New South Wales followed by Victoria (n=188; 24%), Queensland (n=181; 23%), the Australian Capital Territory (n=99; 13%), Western Australia and South Australia (n=28; 4%) each, Tasmania (n=9; 1%), and the Northern Territory (n=7; 1%).

The most commonly reported AEFI were syncope (n=339; 43%), pre-syncope (n=85; 11%), nausea (n=67; 9%), headache (n=65; 8%), dizziness and rash (n=64; 8%) each, injection site reactions (n=60; 8%), pyrexia (n=57; 7%), and urticaria (n=30; 4%). The spectrum of reactions for HPV vaccine was similar in boys and girls, however

Figure 5: Most frequently reported adverse events following immunisation with human papillomavirus vaccine,* 2013, by number of vaccines suspected of involvement in the reported adverse event



* Per cent of 427 adverse events following immunisation records (human papillomavirus males) and 358 records (HPV females) where the vaccine was listed as suspected of involvement in the reported adverse event following immunisation.

Source: Adverse Drug Reactions Reporting System database, Therapeutic Goods Administration.

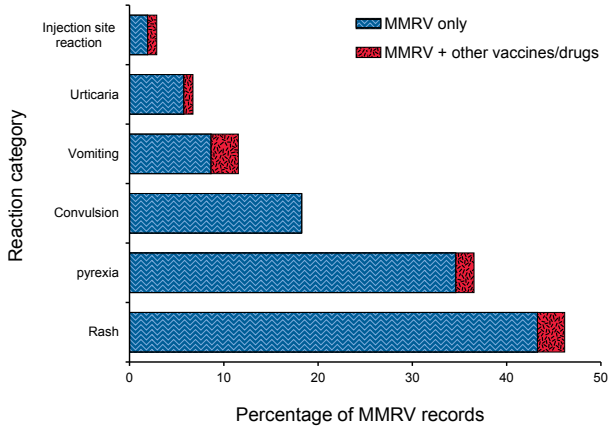
there were more cases in males of pyrexia (67% in males vs 33% in females) and urticaria (70% in males vs 30% in females) (Figure 5).

MMRV vaccine

There was a total of 104 AEFI records for 2013 where MMRV vaccine was recorded (Table 4). In the majority of the records (n=91; 88%), MMRV vaccine was given at 18 months of age. Of all the reported cases, 102 (98%) were in people aged less than 7 years, with three of these cases coded as serious. The reporting rate for children aged less than 7 years was 75.1 per 100,000 doses (Table 2). In 94% of the reports, MMRV (n=98) was administered alone.

The spectrum of reactions for MMRV included 48 (46%) reports of rash; 38 (37%) of pyrexia; 19 cases (18%) of convulsions, including 17 febrile convulsions; 12 cases (12%) of vomiting; 7 cases (7%) of urticaria; 3 cases each of HHE and ISRs (3%); and 2 cases of extensive swelling of the vaccinated limb (Figure 6).

Figure 6: Most frequently reported adverse events following immunisation with measles-mumps-rubella-varicella vaccine,* 2013, by number of vaccines suspected of involvement in the reported adverse event



* Per cent of 104 adverse events following immunisation where measles-mumps-rubella-varicella vaccine was listed as suspected of involvement in the reported adverse event following immunisation.

Source: Adverse Drug Reactions Reporting System database, Therapeutic Goods Administration.

Hib-MenC (Menitorix)

There was a total of 92 AEFI records for 2013 where Hib-MenC vaccine was recorded (Table 4). Of these, 91 (99%) were in people aged less than 7 years, with seven of these cases coded as serious. The reporting rate for children aged less than 7 years was 73.7 per 100,000 doses (Table 2). In 95% of the reports, Hib-MenC vaccine (n=87) was co-administered with MMR vaccine.

The spectrum of reactions for Hib-MenC vaccine included 34 (37%) reports of rash; 30 (32%) of pyrexia; 19 cases (21%) of convulsions, including 16 febrile convulsions; 10 cases (11%) of vomiting; 8 cases (9%) of urticaria; and 6 cases of ISRs (3%).

Discussion

This report uses a different methodology of analysis than that used in previous annual reports for specific AEFIs. The methodological framework used here allows for a clearer reporting of adverse events using MedDRA PTs, as used in the DAEN. This change in methodology needs to be taken into account when comparing with data from previous annual reports on specific reaction terms and categories.

In 2013, there was an increase in both the number of AEFI reports and population-based reporting rates. The increase was predominantly due to the substantial increase in reports in adolescents following HPV vaccination obtained due to a larger vaccine target age group and enhanced safety surveillance, implemented as part of the extension of the National HPV Vaccination Program in February 2013 to males aged 12–13 years, along with a catch-up program for males aged 14 and 15 years.

The TGA, together with state and territory health departments, closely monitored adverse events reported following HPV vaccination as the program was extended to males, through enhanced surveillance using rapid reporting from school-based programs. This aimed to detect 4 acute conditions: a) anaphylaxis; b) generalised allergic reactions; c) loss of consciousness (simple faints [syncope], faints with injury, faints with convulsion); and d) any condition requiring emergency department attendance or hospitalisation. In addition, historical data show that initial high levels of AEFI reporting occur each time a new vaccine is introduced (such as meningococcal C conjugate vaccine in 2003, rotavirus vaccine in 2007, and HPV vaccine in girls in 2007) as immunisation providers are more likely to report milder, less serious AEFIs for vaccines they are not familiar with, followed by a reduction and stabilisation of reporting over time (Weber effect).[26] This enhanced propensity to report events following newer vaccines increases the sensitivity of the system to detect signals of serious, rare or previously unknown events.

The majority of the AEFI reports for HPV vaccine were mild vaccine side effects that had been identified in pre-registration clinical trials.[31] These included injection site reactions, mild allergic reactions, and a range of mild non-specific symptoms including headache, nausea, dizziness, malaise and weakness. A similar range of events has previously been reported to the TGA in secondary school students following receipt of meningococcal C conjugate vaccine as part of the national catch-up program in 2003 and 2004.[4,5] [32,33] The enhanced surveillance implemented in schools in February 2013 resulted in increased reporting of syncope following HPV vaccine. Syncope, usually due to a vasovagal response to having an injection is recognised as a potential AEFI following any immunisation, with highest reporting rates for syncope in adolescents.[34,35]

Reporting rates per 100,000 doses were higher for all individual vaccines in 2013 compared with 2012. The increase in reports for children aged less than 1 years is primarily due to vaccination with hexavalent, 13vPCV and rotavirus vaccines either administered alone or together. The increase in reports for children aged 1 to less than 2 years is primarily due to vaccination with MMR, MMRV, Hib, HibMenC and MenC vaccines either administered alone or together. As two of these were new vaccines on the NIP in 2013, this increased rate of reporting is not unexpected. In addition, active surveillance for febrile seizures following measles-containing vaccines was implemented in paediatric hospitals (in the PAEDS network)[36] as part of enhanced surveillance for the introduction of MMRV to the NIP, and likely contributed to the increased number of reports and awareness of the potential for AEFI following these vaccines. The increase in reports for children aged 2 to less than 7 years is primarily due to ISR following vaccination with DTPa-IPV and MMR co-administered together. The increase was largely seen in Victoria, Queensland and New South Wales.

Conclusion

The total number of reported AEFI in 2013 increased by 59% compared with 2012, due to an increasing trend in propensity to report. The higher reporting rates may also be in response to the activities undertaken by the TGA and the state and territory health departments to encourage and facilitate reporting of AEFI. Reporting rates for the majority of the vaccines were higher than 2012. Increases were most marked in the 7 to under 20 year age group following extension of HPV to boys and associated enhanced surveillance. The majority of AEFIs reported to the TGA were mild transient events. The data reported here are consistent with an overall high level of safety for vaccines included in the NIP schedule.

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Abbreviations of vaccine types

7vPCV	7-valent pneumococcal conjugate vaccine
10vPCV	10-valent pneumococcal conjugate vaccine
13vPCV	13-valent pneumococcal conjugate vaccine
23vPPV	23-valent pneumococcal polysaccharide vaccine
BCG	bacille Calmette-Guérin (i.e. tuberculosis)
dT	diphtheria-tetanus – adolescent and adult formulation
DTPa	diphtheria-tetanus-pertussis (acellular) – paediatric formulation
dTpa	diphtheria-tetanus-pertussis (acellular) – adolescent and adult formulation
dTpa-IPV	combined dTpa and inactivated poliovirus
DTPa-HepB	combined diphtheria-tetanus-pertussis (acellular) and hepatitis B
DTPa-IPV	combined diphtheria-tetanus-pertussis (acellular) and inactivated poliovirus (quadrivalent)
DTPa-IPV-HepB	combined diphtheria-tetanus-pertussis (acellular), inactivated poliovirus and hepatitis B (pentavalent)
DTPa-IPV-HepB-Hib	combined diphtheria-tetanus-pertussis (acellular), inactivated poliovirus, hepatitis B and *Haemophilus influenzae* type b vaccine (hexavalent)
HepB	hepatitis B
Hib	*Haemophilus influenzae* type b
Hib-HepB	combined *Haemophilus influenzae* type b and hepatitis B
HPV	human papillomavirus
IPV	inactivated poliovirus vaccine
Men4PV	meningococcal polysaccharide tetravalent vaccine
MenCCV	meningococcal C conjugate vaccine
MMR	measles-mumps-rubella
MMRV	measles-mumps-rubella-varicella
pH1N1	pandemic H1N1 influenza 2009
TIV	trivalent influenza vaccine

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Australia's notifiable disease status, 2013: Annual report of the National Notifiable Diseases Surveillance System

NNDSS Annual Report Writing Group

Abstract

In 2013, 65 diseases and conditions were nationally notifiable in Australia. States and territories reported a total of 224,434 notifications of communicable diseases to the National Notifiable Diseases Surveillance System, a decrease of 8% on the number of notifications in 2012. In 2013, the most frequently notified diseases were sexually transmissible infections (100,949 notifications, 45% of total notifications), vaccine preventable diseases (59,630 notifications, 26.6% of total notifications), and gastrointestinal diseases (32,536 notifications, 14.5% of total notifications). There were 17,919 notifications of bloodborne diseases; 10,831 notifications of vectorborne diseases; 1,932 notifications of other bacterial infections; 634 notifications of zoonoses and 3 notifications of quarantinable diseases. *Commun Dis Intell* 2015;39(3):E387–E478.

Keywords: Australia, communicable diseases, epidemiology, surveillance

Introduction

Australia's notifiable diseases status, 2013, is an annual surveillance report of nationally notifiable communicable diseases. Communicable disease surveillance in Australia operates at the national, jurisdictional and local levels. Primary responsibility for public health action lies with the state and territory health departments. The role of communicable disease surveillance at the national level includes:

- identifying national trends;
- providing guidance for policy development and resource allocation at the national level;
- monitoring the need for and impact of national disease control programs;
- coordinating the response to national or multi-jurisdictional outbreaks;
- describing the epidemiology of rare diseases that occur infrequently at state and territory levels;
- meeting various international reporting requirements, such as providing disease statistics to the World Health Organization (WHO); and
- supporting quarantine activities, which are the responsibility of the Commonwealth government.

Methods

Australia is a federation of 6 states (New South Wales, Queensland, South Australia, Tasmania, Victoria and Western Australia) and 2 territories (the Australian Capital Territory and the Northern Territory).

State and territory health departments collect notifications of communicable diseases under their respective public health legislation. In September 2007, the *National Health Security Act 2007*[1] received royal assent. This Act provides a legislative basis for, and authorises the exchange of health information, including personal information, between jurisdictions and the Commonwealth. The Act provides for the establishment of the National Notifiable Diseases List,[2] which specifies the diseases about which personal information can be provided. The *National Health Security Agreement*,[3] which was signed by Health Ministers in April 2008, establishes the operational arrangements to formalise and enhance existing surveillance and reporting systems, an important objective of the Act. Under the Agreement, in 2013 states and territories forwarded de-identified notification data on the nationally agreed set of 65 communicable diseases to the Department of Health for the purposes of national communicable disease surveillance, although not all 65 diseases were notifiable in each jurisdiction. Data were electronically updated daily from states and territories. The system was complemented by other surveillance systems, which provided information on various diseases, including four that are not reported to the National Notifiable Diseases Surveillance System (NNDSS), human immunodeficiency virus (HIV), acquired immune deficiency syndrome (AIDS) and the classical and variant forms of Creutzfeldt-Jakob disease (CJD).

The NNDSS core dataset requires the following mandatory data fields: unique record reference number; notifying state or territory; disease code; confirmation status and the date when the jurisdictional health department was notified (notification received date). In addition, the following data fields

were supplied where possible: date of birth; age at onset; sex; Indigenous status; postcode of residence; disease onset date; date when the pathology service authorised a report or a medical practitioner signed the notification form (notification date); death status; date of specimen collection; and outbreak reference number (to identify cases linked to an outbreak). Where relevant, information on the species, serogroups/subtypes and phage types of organisms isolated, and on the vaccination status of the case were collected and reported to NNDSS. Data quality was monitored by the Office of Health Protection and the National Surveillance Committee (NSC) and there was a continual process of improving the national consistency of communicable disease surveillance through the daily, fortnightly and quarterly review of these data.

While not included in the core national dataset, enhanced surveillance information for some diseases (invasive pneumococcal disease, hepatitis B, hepatitis C, tuberculosis, donovanosis, gonococcal infection and syphilis < 2 years duration) were reported from states and territories to NNDSS. With the exception of hepatitis B and hepatitis C these enhanced data are not included in this report. These data, along with influenza enhanced data, are reported in individual annual reports. Additional information concerning mortality and specific health risk factors for some diseases were obtained from states and territories and included in this annual report.

Newly diagnosed HIV infection and AIDS were notifiable conditions in each state or territory health jurisdiction in 2013. These notifications were forwarded to the Kirby Institute for Infection and Immunity in Society. Further information can be found in the Kirby Institute's annual surveillance report.[4]

Surveillance for the classical and variant forms of CJD in Australia has been conducted through the Australian National Creutzfeldt-Jakob Disease Registry (ANCJDR) since its establishment in October 2003. CJD is a nationally notifiable disease and by June 2006, CJD was notifiable in all states and territories. Further surveillance information on CJD can be found in surveillance reports from the ANCJDR.[5]

Information on communicable disease surveillance is communicated through several avenues. The most up-to-date information on topics of interest is provided at the fortnightly teleconferences of the Communicable Diseases Network Australia (CDNA). A summary of these reports is available online from the CDNA website (http://www.health.gov.au/internet/main/publishing.nsf/Content/cdnareport.htm).[6]

The *Communicable Diseases Intelligence* (CDI) quarterly journal publishes surveillance data, annual surveillance reports, short reports, and articles on the epidemiology and control of communicable diseases.

Notification rates for each notifiable disease were calculated using the estimated 2013 December resident population supplied by the Australian Bureau of Statistics (Appendix 1 and Appendix 2).[7] Where diseases were not notifiable in a state or territory, national rates were adjusted by excluding the population of that jurisdiction from the denominator. For some diseases, age adjusted rates were calculated using the direct method of standardisation with 2011 census data as the standard population. All rates are represented as the rate per 100,000 population unless stated otherwise.

Direct age standardised notification rates, using the method described by the Australian Institute of Health and Welfare[8] were calculated for Aboriginal and Torres Strait Islander and non-Indigenous notifications for relevant sexually transmissible infections (STIs) for jurisdictions that had Indigenous status data completed for more than 50% of notifications over the period 2007 to 2012. Where the Indigenous status of a notification was not completed, these notifications were counted as non-Indigenous in the analyses. These data, however, should be interpreted with caution, as STI screening may occur predominately in specific high risk groups, including in remote Aboriginal and Torres Strait Islander populations. Recent studies have suggested that higher rates in Aboriginal and Torres Strait Islander populations may be attributable to higher prevalence and reinfection rates while others have suggested that it may be due to increased testing and contact tracing.[9]

In the national case definitions for chlamydial infection, gonococcal infection and syphilis the mode of transmission cannot be inferred from the site of infection. Infections in children may be acquired perinatally (e.g. congenital chlamydia).[10] Notifications of chlamydial, gonococcal and non-congenital syphilis infections were excluded from analysis of age and sex distribution where the case was aged less than 13 years and the infection was able to be determined as non-sexually acquired through enhanced surveillance data where available.

Notes on interpretation

This present report is based on 2013 data from each state and territory, agreed upon in June 2014, and represents a snap shot of the year after duplicate records and incorrect or incomplete data were removed. Totals in this report may vary slightly from the totals reported in CDI quarterly publications.

Analyses in this report were based on the date of disease diagnosis in an attempt to estimate disease activity within the reporting period. The date of diagnosis is the onset date or where the onset date was not known, the earliest of the following dates, specimen collection date, the notification date, or the notification receive date. In January 2014, the NSC redefined the diagnosis date methodology for hepatitis B (unspecified), hepatitis C (unspecified), leprosy, syphilis (unspecified) and tuberculosis. As a considerable amount of time can elapse between the initial infection, the onset of symptoms and the subsequent diagnosis, the diagnosis date for these 5 diseases is derived from the notification receive date.

When referring to NNDSS notification data throughout the report, the term 'cases' or 'notified cases' are used to identify individuals in whom 'notification' of a condition has been received by NNDSS. These notifications can only represent a proportion (the 'notified fraction') of the total incidence (Figure 1) and this has to be taken into account when interpreting NNDSS data. Moreover, the notified fraction varies by jurisdiction, over time and by disease. This caveat is particularly relevant to STIs, many or most of which are identified through screening programs (Figure 1).

A survey of jurisdictional public health departments was conducted in 2013 to ascertain the source of each notification (Table 1). Whilst most jurisdictions have data on laboratory notifications, the percentage of notifications attributed to doctor only and laboratory and doctor for each state and territory are based on estimates deduced from the data that are available, noting that fields for these data may be incomplete. Only Western Australia and New South Wales maintain data on the source of notifications from laboratories and/or doctors.

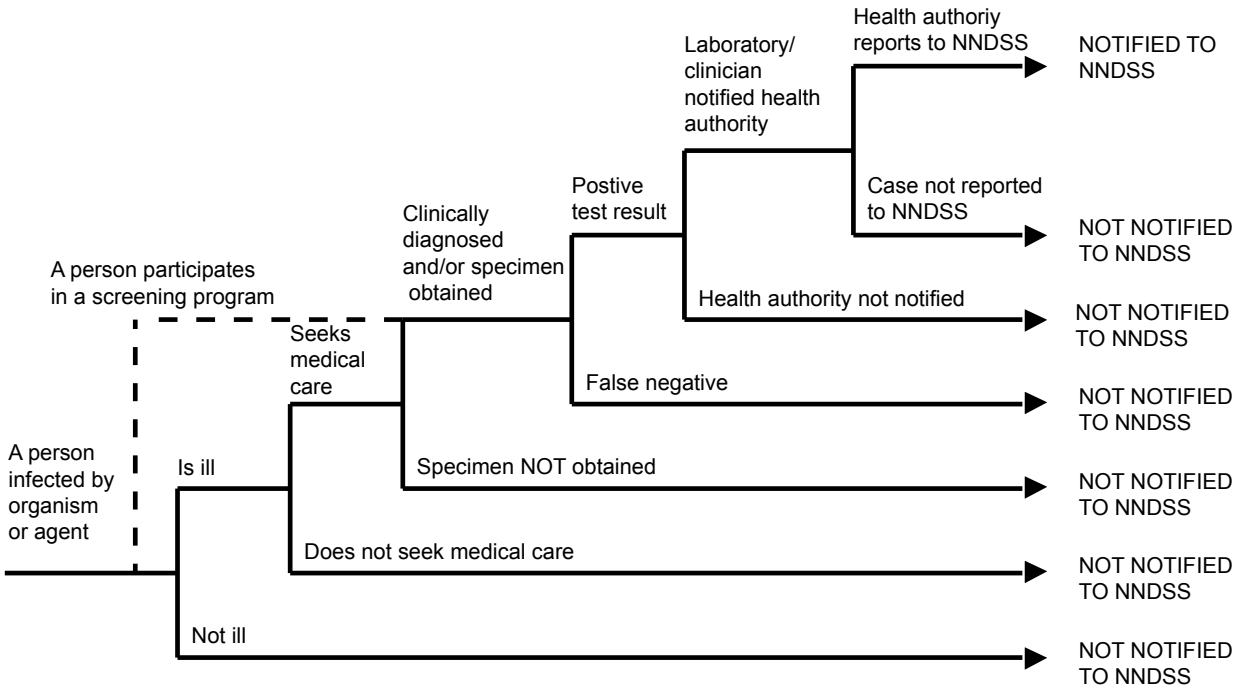
Methods of surveillance vary between states and territories, each having different requirements for notification by medical practitioners, laboratories

Table 1: Percentage of notified cases from different sources in each jurisdiction, 2013*

State or territory	Source of notifications		
	Laboratory only	Doctor only	Laboratory and doctor
ACT	95.0	<1.0	~4.0
NSW	98.2	0.7	0.3
NT	98.0	0.7	1.3
Qld	99.5	0.1	0.3
SA	4.0	3.0	93.0
Tas.	99.0	1.0	<1.0
Vic.	38.0	5.0	52.0
WA	33.4	1.4	65.2

* Not all percentages add up to 100% due to other sources of notifications and/or incomplete data for laboratory and medical notification fields

Figure 1: Communicable diseases notifiable fraction



and hospitals. Although the National Notifiable Diseases List[2] was established, some diseases are not notifiable in all 8 jurisdictions (Table 2).

Changes in surveillance practices may have been introduced in some jurisdictions and not in others, and must be taken into consideration when comparing data between jurisdictions. In this report, some additional information was obtained from states and territories, to assist in the interpretation of the 2013 data. These include changes in surveillance practices, screening practices, laboratory practices, and major disease control or prevention initiatives.

Postcode information usually reflects the residential location of the case, but this does not necessarily represent the place where the disease was acquired.

Data completeness was assessed for cases' sex, age at onset, and Indigenous status, and reported as the proportion of complete notifications. The completeness of data in this report is summarised in the Results.

Table 2: Diseases notified to the National Notifiable Diseases Surveillance System, Australia, 2013

Disease	Data received from
Bloodborne diseases	
Hepatitis B (newly acquired)	All jurisdictions
Hepatitis B (unspecified)	All jurisdictions
Hepatitis C (newly acquired)	All jurisdictions, except Queensland
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
Salmonellosis	All jurisdictions
Shigellosis	All jurisdictions
STEC, VTEC*	All jurisdictions
Typhoid fever	All jurisdictions
Quarantinable diseases	
Cholera	All jurisdictions
Highly pathogenic avian influenza in humans	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 infections	All jurisdictions
Donovanosis	All jurisdictions
Gonococcal infection	All jurisdictions
Syphilis < 2 years duration	All jurisdictions
Syphilis > 2 years or unspecified duration	All jurisdictions
Syphilis – congenital	All jurisdictions

Table 2 (cont'd): Diseases notified to the National Notifiable Diseases Surveillance System, Australia, 2013

Disease	Data received from
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
Poliomyelitis	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	
Arbovirus infection (NEC)	All jurisdictions
Barmah Forest virus infection	All jurisdictions
Dengue virus infection	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 disease (invasive)	All jurisdictions
Tuberculosis	All jurisdictions

* Infection with Shiga toxin/verotoxin-producing *Escherichia coli*.

NEC Not elsewhere classified.

The percentage of data completeness was defined as:

Percentage of data completeness = (total notifications – missing or unknown) / total notifications x 100

The Indigenous status was defined by the following nationally accepted criteria:[11]

1=Indigenous – (Aboriginal but not Torres Strait Islander origin)

2=Indigenous – (Torres Strait Islander but not Aboriginal origin)

3=Indigenous – (Aboriginal and Torres Strait Islander origin)

4=Not Indigenous – (not Aboriginal or Torres Strait Islander origin)

For the purposes of this report, an Indigenous person includes responses 1, 2 or 3 with non-Indigenous including response 4 only.

9=Not stated

In interpreting STI notification data, it is important to note that changes in notifications over time may not solely reflect changes in disease prevalence as changes in screening programs,[12,13] the use of less invasive and more sensitive diagnostic tests[14] and periodic public awareness campaigns[15] may influence the number of notifications that occur over time. Rates for STIs are particularly susceptible to overall rates of testing, with low testing rates resulting in an underestimation of disease and increased testing potentially causing an increase in notifications.[16] For some diseases, changes in surveillance practices may also need to be taken into account when interpreting national trends.

The differences in rates between females and males for STIs should be interpreted with caution, as rates of testing, symptom status, health care-seeking behaviours, and partner notification differ between the sexes.[17]

Notes on case definitions

Each notifiable disease is governed by a national surveillance case definition for reporting to the NNDSS. These case definitions were agreed by CDNA and implemented nationally in January 2004 and were used by all jurisdictions for the first time in 2005. These case definitions are reviewed by the Case Definitions Working Group (CDWG) as required.

The national surveillance case definitions and their review status are available from the Department of Health web site (http://www.health.gov.au/case-definitions).

Results

There were 224,434 communicable disease notifications received by NNDSS in 2013 (Table 3).

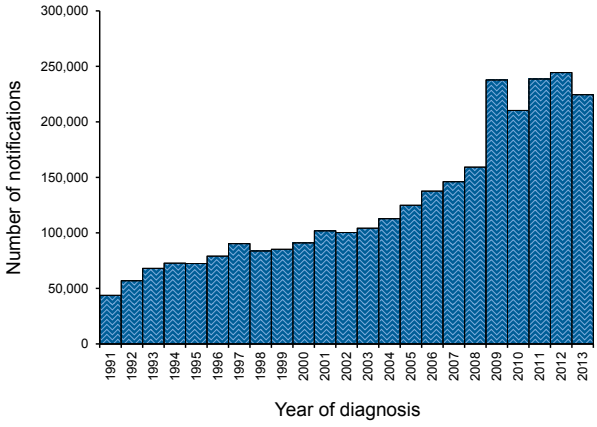
In 2013, the most frequently notified diseases were sexually transmissible infections (100,949 notifications, 45% of total notifications), vaccine preventable diseases (59,630 notifications, 26.6% of total notifications), and gastrointestinal diseases (32,535 notifications, 14.5% of total notifications).

Table 3: Notifications to the National Notifiable Diseases Surveillance System, Australia, 2013, by disease category rank order

Disease category	Number	%
Sexually transmissible infections	100,949	45.0
Vaccine preventable diseases	59,630	26.6
Gastrointestinal diseases	32,536	14.5
Bloodborne diseases	17,919	8.0
Vectorborne diseases	10,831	4.8
Other bacterial diseases	1,932	0.9
Zoonoses	634	0.3
Quarantinable diseases	3	<0.1
Total	224,434	100.0

There was a decrease of 8% compared with the total number of notifications in 2012 (Figure 2). The decrease can largely be attributed to the 2013 influenza season, which commenced later and occurred over a shorter period and was considered a more moderate season when compared with the 2012 season.

Figure 2: Notifications received by the National Notifiable Diseases Surveillance System, Australia, 1991 to 2013



Notifications and notification rates per 100,000 for each disease in 2013, by state or territory, are shown in Table 4 and Table 5 respectively. Notifications and rates per 100,000 for the period 2008 to 2013 are shown in Table 6.

Table 4: Notified cases of communicable diseases, Australia, 2013, by state or territory

Disease	State or territory								
	ACT	NSW	NT	Qld	SA	Tas.	Vic.	WA	Aust.
Bloodborne diseases									
Hepatitis B (newly acquired)*	4	33	6	45	8	3	34	39	172
Hepatitis B (unspecified)†	107	2,506	325	906	286	55	1,850	944	6,979
Hepatitis C (newly acquired)*	14	43	1	NN	62	19	145	123	407
Hepatitis C (unspecified)†,‡	170	3,503	256	2,469	414	210	2,130	1,156	10,308
Hepatitis D	0	9	1	13	4	0	22	4	53
Gastrointestinal diseases									
Botulism	0	2	0	0	0	0	2	0	4
Campylobacteriosis	373	NN	199	3,831	1,719	696	5,953	1,927	14,698
Cryptosporidiosis	39	1,107	89	766	135	74	1,264	372	3,846
Haemolytic uraemic syndrome	0	9	1	2	1	0	2	0	15
Hepatitis A	4	62	0	45	11	0	53	14	189
Hepatitis E	1	16	0	2	0	0	8	4	31
Listeriosis	1	29	3	9	2	2	22	8	76
Salmonellosis	279	3,456	385	3,207	982	249	2,954	1,279	12,791
Shigellosis	10	149	108	73	29	3	115	69	556
STEC,VTEC§	3	25	0	83	53	1	11	4	180
Typhoid fever	5	59	0	24	8	0	44	10	150
Quarantinable diseases									
Cholera	0	2	0	0	0	0	1	0	3
HPAIH	0	0	0	0	0	0	0	0	0
Plague	0	0	0	0	0	0	0	0	0
Rabies	0	0	0	0	0	0	0	0	0
Severe acute respiratory syndrome	0	0	0	0	0	0	0	0	0
Smallpox	0	0	0	0	0	0	0	0	0
Viral haemorrhagic fever	0	0	0	0	0	0	0	0	0
Yellow fever	0	0	0	0	0	0	0	0	0
Sexually transmitted infections									
Chlamydial infection‖,¶	1,269	20,827	2,998	19,497	5,183	1,538	19,467	11,747	82,526
Donovanosis	0	0	0	0	0	0	0	0	0
Gonococcal infection¶	114	4,231	1,955	2,732	855	69	3,014	1,972	14,942
Syphilis – congenital¶	0	3	1	1	0	0	0	2	7
Syphilis < 2 years duration*,¶,**	10	598	22	328	56	19	652	83	1,768
Syphilis > 2 years or unspecified duration†,¶	9	417	94	306	104	11	564	201	1,706
Vaccine preventable diseases									
Diphtheria	0	0	0	1	1	0	0	0	2
Haemophilus influenzae type b	0	9	0	7	0	0	4	0	20
Influenza (laboratory confirmed)	570	8,398	481	5,509	4,825	297	5,854	2,395	28,329
Measles	1	34	0	52	16	0	41	14	158
Mumps	1	90	6	41	5	5	24	45	217
Pertussis	228	2,336	108	3,808	811	513	2,898	1,639	12,341
Pneumococcal disease (invasive)	14	467	58	272	112	37	393	193	1,546
Poliomyelitis	0	0	0	0	0	0	0	0	0
Rubella	1	12	0	6	2	0	3	1	25
Rubella – congenital	0	0	0	0	1	0	1	0	2
Tetanus	0	2	0	0	1	0	0	1	4

Table 4 (cont'd): Notified cases of communicable diseases, Australia, 2013, by state or territory

Disease	State or territory								
	ACT	NSW	NT	Qld	SA	Tas.	Vic.	WA	Aust.
Vaccine preventable diseases (cont'd)									
Varicella zoster (chickenpox)	20	NN	97	280	386	29	871	359	2,042
Varicella zoster (shingles)	52	NN	246	45	1,899	247	1,223	1,305	5,017
Varicella zoster (unspecified)	138	NN	10	5,337	105	89	3,018	1,230	9,927
Vectorborne diseases									
Arbovirus infection (NEC)	0	0	1	20	0	0	0	0	21
Barmah Forest virus infection	6	431	405	2,224	74	3	72	1,024	4,239
Dengue virus infection	10	300	56	489	75	19	414	478	1,841
Japanese encephalitis virus infection	0	0	0	2	1	0	0	1	4
Kunjin virus infection††	0	0	0	3	0	0	0	0	3
Malaria	13	88	22	108	8	11	88	76	414
Murray Valley encephalitis virus infection	0	0	0	1	0	0	0	0	1
Ross River virus infection	4	503	300	1,787	167	8	171	1,368	4,308
Zoonoses									
Anthrax	0	0	0	0	0	0	0	0	0
Australia bat lyssavirus infection	0	0	0	1	0	0	0	0	1
Brucellosis	0	3	0	11	0	0	0	0	14
Leptospirosis	0	12	4	67	2	0	9	1	95
Lyssavirus infection (NEC)	0	0	0	0	0	0	0	0	0
Ornithosis	0	8	0	1	0	0	34	4	47
Q fever	0	167	1	243	17	0	41	8	477
Tularaemia	0	0	0	0	0	0	0	0	0
Other bacterial diseases									
Legionellosis	1	105	6	165	63	6	66	93	505
Leprosy	1	2	2	1	1	0	3	3	13
Meningococcal infection‡‡	3	48	2	33	20	3	25	15	149
Tuberculosis	18	440	41	156	69	8	383	150	1,265
Total	3,493	50,540	8,290	55,005	18,573	4,224	53,943	30,361	224,429

* Newly acquired hepatitis and syphilis < 2 years duration includes cases where the infection was determined to be acquired within 24 months prior to diagnosis.

† Unspecified hepatitis and syphilis includes cases where the duration of infection could not be determined or is greater than 24 months.

‡ In Queensland, includes newly acquired hepatitis C cases.

§ Infection with Shiga toxin/verotoxin producing *Escherichia coli*.

|| Includes *Chlamydia trachomatis* identified from cervical, rectal, urine, urethral, throat and eye samples, except for South Australia, which reports only cervical, urine and urethral specimens; the Northern Territory and Western Australia exclude ocular infections. From 1 July 2013 the case definition changed to exclude all ocular infections.

¶ The national case definitions for chlamydial, 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).

** Data for all states and territories are reported by diagnosis date, except Queensland, which is reported by notification receive date.

†† In the Australian Capital Territory, Murray Valley encephalitis virus infection and Kunjin virus infection are combined under Murray Valley encephalitis virus infection.

‡‡ Only invasive meningococcal disease is nationally notifiable. However the Australian Capital Territory and New South Wales also report conjunctival cases.

HPAIH Highly pathogenic avian influenza in humans.

NEC Not elsewhere classified.

NN Not notifiable.

Table 5: Notification rates per 100,000 of nationally notifiable communicable diseases, Australia, 2013, by state or territory

Disease	State or territory								Aust.
	ACT	NSW	NT	Qld	SA	Tas.	Vic.	WA	
Bloodborne diseases									
Hepatitis B (newly acquired)*	1.0	0.4	2.5	1.0	0.5	0.6	0.6	1.5	0.7
Hepatitis B (unspecified)†	28.0	33.8	134.7	19.5	17.1	10.7	32.2	37.5	30.2
Hepatitis C (newly acquired)*	3.7	0.6	0.4	NN	3.7	3.7	2.5	4.9	2.2
Hepatitis C (unspecified)†,‡	44.6	47.3	106.1	53.0	24.8	40.9	37.1	45.9	44.6
Hepatitis D	–	0.1	0.4	0.3	0.2	–	0.4	0.2	0.2
Gastrointestinal diseases									
Botulism	–	<0.1	–	–	–	–	<0.1	–	<0.1
Campylobacteriosis	97.8	NN	82.5	82.3	102.9	135.6	103.7	76.5	93.5
Cryptosporidiosis	10.2	14.9	36.9	16.5	8.1	14.4	22.0	14.8	16.6
Haemolytic uraemic syndrome	–	0.1	0.4	<0.1	0.1	–	<0.1	–	0.1
Hepatitis A	1.0	0.8	–	1.0	0.7	–	0.9	0.6	0.8
Hepatitis E	0.3	0.2	–	<0.1	–	–	0.1	0.2	0.1
Listeriosis	0.3	0.4	1.2	0.2	0.1	0.4	0.4	0.3	0.3
Salmonellosis	73.1	46.6	159.6	68.9	58.8	48.5	51.5	50.7	55.3
Shigellosis	2.6	2.0	44.8	1.6	1.7	0.6	2.0	2.7	2.4
STEC,VTEC§	0.8	0.3	–	1.8	3.2	0.2	0.2	0.2	0.8
Typhoid fever	1.3	0.8	–	0.5	0.5	–	0.8	0.4	0.6
Quarantinable diseases									
Cholera	–	<0.1	–	–	–	–	<0.1	–	<0.1
HPAIH	–	–	–	–	–	–	–	–	–
Plague	–	–	–	–	–	–	–	–	–
Rabies	–	–	–	–	–	–	–	–	–
Severe acute respiratory syndrome	–	–	–	–	–	–	–	–	–
Smallpox	–	–	–	–	–	–	–	–	–
Viral haemorrhagic fever	–	–	–	–	–	–	–	–	–
Yellow fever	–	–	–	–	–	–	–	–	–
Sexually transmissible infections									
Chlamydial infection‖,¶	332.7	281.1	1243.0	418.8	310.2	299.7	339.2	466.0	356.7
Donovanosis	–	–	–	–	–	–	–	–	–
Gonococcal infection¶	29.9	57.1	810.5	58.7	51.2	13.4	52.5	78.2	64.6
Syphilis – congenital¶	–	<0.1	0.4	<0.1	–	–	–	0.1	<0.1
Syphilis < 2 years duration*,¶,**	2.6	8.1	9.1	7.0	3.4	3.7	11.4	3.3	7.6
Syphilis > 2 years or unspecified duration†,¶	2.4	5.6	39.0	6.6	6.2	2.1	9.8	8.0	7.4
Vaccine preventable diseases									
Diphtheria	–	–	–	<0.1	0.1	–	–	–	<0.1
Haemophilus influenzae type b	–	0.1	–	0.2	–	–	0.1	–	0.1
Influenza (laboratory confirmed)	149.4	113.3	199.4	118.3	288.8	57.9	102.0	95.0	122.5
Measles	0.3	0.5	–	1.1	1.0	–	0.7	0.6	0.7
Mumps	0.3	1.2	2.5	0.9	0.3	1.0	0.4	1.8	0.9
Pertussis	59.8	31.5	44.8	81.8	48.5	100.0	50.5	65.0	53.3
Pneumococcal disease (invasive)	3.7	6.3	24.0	5.8	6.7	7.2	6.8	7.7	6.7
Poliomyelitis	–	–	–	–	–	–	–	–	–
Rubella	0.3	0.2	–	0.1	0.1	–	0.1	<0.1	0.11
Rubella – congenital	–	–	–	–	0.1	–	<0.1	–	<0.1
Tetanus	–	<0.1	–	–	0.1	–	–	<0.1	<0.1

Table 5 (cont'd): Notification rates per 100,000 of nationally notifiable communicable diseases, Australia, 2013, by state or territory

Disease	State or territory								Aust.
	ACT	NSW	NT	Qld	SA	Tas.	Vic.	WA	
Vaccine preventable diseases (cont'd)									
Varicella zoster (chickenpox)	5.2	NN	40.2	6.0	23.1	5.7	15.2	14.2	13.0
Varicella zoster (shingles)	13.6	NN	102.0	1.0	113.7	48.1	21.3	51.8	31.9
Varicella zoster (unspecified)	36.2	NN	4.1	114.7	6.3	17.3	52.6	48.8	63.1
Vectorborne diseases									
Arbovirus infection (NEC)	–	–	0.4	0.4	–	–	–	–	0.1
Barmah Forest virus infection	1.6	5.8	167.9	47.8	4.4	0.6	1.3	40.6	18.3
Dengue virus infection	2.6	4.0	23.2	10.5	4.5	3.7	7.2	19.0	8.0
Japanese encephalitis virus infection	–	–	–	<0.1	0.1	–	–	<0.1	<0.1
Kunjin virus infection††	–	–	–	0.1	–	–	–	–	<0.1
Malaria	3.4	1.2	9.1	2.3	0.5	2.1	1.5	3.0	1.8
Murray Valley encephalitis virus infection	–	–	–	<0.1	–	–	–	–	<0.1
Ross River virus infection	1.0	6.8	124.4	38.4	10.0	1.6	3.0	54.3	18.6
Zoonoses									
Anthrax	–	–	–	–	–	–	–	–	–
Australia bat lyssavirus infection	–	–	–	<0.1	–	–	–	–	<0.1
Brucellosis	–	<0.1	–	0.2	–	–	–	–	0.1
Leptospirosis	–	0.2	1.7	1.4	0.1	–	0.2	<0.1	0.4
Lyssavirus infection (NEC)	–	–	–	–	–	–	–	–	–
Ornithosis	–	0.1	–	<0.1	–	–	0.6	0.2	0.2
Q fever	–	2.3	0.4	5.2	1.0	–	0.7	0.3	2.1
Tularaemia	–	–	–	–	–	–	–	–	–
Other bacterial diseases									
Legionellosis	0.3	1.4	2.5	3.5	3.8	1.2	1.2	3.7	2.2
Leprosy	0.3	<0.1	0.8	<0.1	0.1	–	0.1	0.1	0.1
Meningococcal infection‡‡	0.8	0.6	0.8	0.7	1.2	0.6	0.4	0.6	0.6
Tuberculosis	4.7	5.9	17.0	3.4	4.1	1.6	6.7	6.0	5.5

* Newly acquired hepatitis and syphilis < 2 years duration includes cases where the infection was determined to be acquired within 24 months prior to diagnosis..

† Unspecified hepatitis and syphilis includes cases where the duration of infection could not be determined or is greater than 24 months.

‡ In Queensland, includes newly acquired hepatitis C cases.

§ Infection with Shiga toxin/verotoxin producing *Escherichia coli.*

|| Includes *Chlamydia trachomatis* identified from cervical, rectal, urine, urethral, throat and eye samples, except for South Australia, which reports only cervical, urine and urethral specimens; the Northern Territory and Western Australia exclude ocular infections. From 1 July 2013 the case definition changed to exclude all ocular infections.

¶ The national case definitions for chlamydial, 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).

** Data for all states and territories are reported by diagnosis date, except Queensland, which is reported by notification receive date.

†† In the Australian Capital Territory, Murray Valley encephalitis virus infection and Kunjin virus infection are combined under Murray Valley encephalitis virus infection.

‡‡ Only invasive meningococcal disease is nationally notifiable. However the Australian Capital Territory and New South Wales also report conjunctival cases.

HPAIH Highly pathogenic avian influenza in humans.

NEC Not elsewhere classified.

NN Not notifiable.

Table 6: Notified cases and notification rate for communicable diseases, Australia, 2008 to 2013

Disease	Number of notified cases								Notification rate per 100,000					
	2008	2009	2010	2011	2012	2013	5-year mean	Ratio (2013: 5-year mean)	2008	2009	2010	2011	2012	2013
Bloodborne diseases														
Hepatitis B (newly acquired)*	261	253	230	193	198	172	227.0	0.8	1.2	1.2	1.0	0.9	0.9	0.7
Hepatitis B (unspecified)†	6,377	7,127	6,957	6,559	6,538	6,979	6,711.6	1.0	29.7	32.9	31.6	29.4	28.8	30.2
Hepatitis C (newly acquired)*	364	399	400	412	486	407	412.2	1.0	2.1	2.3	2.3	2.3	2.7	2.2
Hepatitis C (unspecified)†‡	10,801	11,104	11,086	9,882	9,641	10,308	10,502.8	1.0	50.2	51.2	50.3	44.2	42.4	44.6
Hepatitis D	41	35	36	39	31	53	36.4	1.5	0.2	0.2	0.2	0.2	0.1	0.2
Gastrointestinal diseases														
Botulism	0	1	0	2	0	4	0.6	6.7	–	<0.1	–	<0.1	–	<0.1
Campylobacteriosis	15,561	16,104	16,990	17,725	15,655	14,698	16,407.0	0.9	107.4	110.0	114.1	117.2	101.5	93.5
Cryptosporidiosis	2,001	4,624	1,481	1,811	3,142	3,846	2,611.8	1.5	9.3	21.3	6.7	8.1	13.8	16.6
Haemolytic uraemic syndrome	32	13	9	13	20	15	17.4	0.9	0.1	0.1	<0.1	0.1	0.1	0.1
Hepatitis A	276	563	267	145	166	189	283.4	0.7	1.3	2.6	1.2	0.6	0.7	0.8
Hepatitis E	44	33	37	41	35	31	38.0	0.8	0.2	0.2	0.2	0.2	0.2	0.1
Listeriosis	68	92	71	70	93	76	78.8	1.0	0.3	0.4	0.3	0.3	0.4	0.3
Salmonellosis	8,297	9,503	11,912	12,276	11,256	12,791	10,648.8	1.2	38.6	43.8	54.1	55.0	49.5	55.3
Shigellosis	830	616	552	493	548	556	607.8	0.9	3.9	2.8	2.5	2.2	2.4	2.4
STEC,VTEC§	98	128	80	95	111	180	102.4	1.8	0.5	0.6	0.4	0.4	0.5	0.8
Typhoid fever	106	115	96	135	124	150	115.2	1.3	0.5	0.5	0.4	0.6	0.5	0.6
Quarantinable diseases														
Cholera	4	5	3	6	5	3	4.6	0.7	<0.1	<0.1	<0.1	<0.1	<0.1	<0.1
HPAIH	0	0	0	0	0	0	0.0	–	–	–	–	–	–	–
Plague	0	0	0	0	0	0	0.0	–	–	–	–	–	–	–
Rabies	0	0	0	0	0	0	0.0	–	–	–	–	–	–	–
Severe acute respiratory syndrome	0	0	0	0	0	0	0.0	–	–	–	–	–	–	–
Smallpox	0	0	0	0	0	0	0.0	–	–	–	–	–	–	–
Viral haemorrhagic fever	0	0	0	0	0	0	0.0	–	–	–	–	–	–	–
Yellow fever	0	0	0	2	0	0	0.4	–	–	–	–	<0.1	–	–

Table 6 (cont'd): Notified cases and notification rate for communicable diseases, Australia, 2008 to 2013

Disease	Number of notified cases						5-year mean	Ratio (2013: 5-year mean)	Notification rate per 100,000					
	2008	2009	2010	2011	2012	2013			2008	2009	2010	2011	2012	2013
Sexually transmissible infections														
Chlamydial infection[‖,¶]	58,456	63,013	74,320	80,918	82,903	82,526	71,922.0	1.1	271.9	290.5	337.3	362.2	364.8	356.7
Donovanosis	2	1	1	0	1	0	1.0	–	<0.1	<0.1	<0.1	–	<0.1	–
Gonococcal infection[¶]	7,678	8,279	10,324	12,100	13,842	14,942	10,444.6	1.4	35.7	38.2	46.9	54.2	60.9	64.6
Syphilis – congenital[¶]	6	3	3	7	1	7	4.0	1.8	<0.1	<0.1	<0.1	<0.1	<0.1	<0.1
Syphilis < 2 years duration[*,¶,**]	1,323	1,314	1,118	1,318	1,570	1,768	1,328.6	1.3	6.2	6.1	5.1	5.9	6.9	7.6
Syphilis > 2 years or unspecified duration[†,¶]	1,359	1,445	1,329	1,315	1,381	1,706	1,365.8	1.2	6.8	7.2	6.5	6.4	6.1	7.4
Vaccine preventable diseases														
Diphtheria	0	0	0	4	0	2	0.8	2.5	–	–	–	<0.1	–	<0.1
Haemophilus influenzae type b	25	19	24	13	15	20	19.2	1.0	0.1	0.1	0.1	0.1	0.1	0.1
Influenza (laboratory confirmed)	9,173	59,028	13,466	27,228	44,571	28,329	30,693.2	0.9	42.7	272.1	61.1	121.9	196.1	122.5
Measles	65	104	70	194	199	158	126.4	1.3	0.3	0.5	0.3	0.9	0.9	0.7
Mumps	286	166	98	155	200	217	181.0	1.2	1.3	0.8	0.4	0.7	0.9	0.9
Pertussis	14,286	30,163	34,821	38,727	24,074	12,341	28,414.2	0.4	66.5	139.1	158.0	173.4	105.9	53.3
Pneumococcal disease (invasive)	1,626	1,557	1,640	1,883	1,823	1,546	1,705.8	0.9	7.6	7.2	7.4	8.4	8.0	6.7
Poliomyelitis	0	0	0	0	0	0	0.0	–	–	–	–	–	–	–
Rubella	36	27	44	58	37	25	40.4	0.6	0.2	0.1	0.2	0.3	0.2	0.1
Rubella – congenital	0	0	0	0	1	2	0.2	10.0	–	–	–	–	<0.1	<0.1
Tetanus	4	3	2	3	7	4	3.8	1.1	<0.1	<0.1	<0.1	<0.1	<0.1	<0.1
Varicella zoster (chickenpox)	1,807	1,796	1,792	2,100	1,977	2,042	1,894.4	1.1	19.7	12.3	12.0	13.9	12.8	13.0
Varicella zoster (shingles)	2,341	2,779	3,047	4,025	4,507	5,017	3,339.8	1.5	25.6	19.0	20.5	26.6	29.2	31.9
Varicella zoster (unspecified)	4,410	6,761	7,269	7,689	8,437	9,927	6,913.2	1.4	48.2	46.2	48.8	50.8	54.7	63.1
Vectorborne diseases														
Arbovirus infection (NEC)	12	6	14	18	9	21	11.8	1.8	0.1	<0.1	0.1	0.1	<0.1	0.1
Barmah Forest virus infection	2,080	1,473	1,470	1,863	1,730	4,239	1,723.2	2.5	9.7	6.8	6.7	8.3	7.6	18.3
Dengue virus infection	561	1,402	1,228	821	1,540	1,841	1,110.4	1.7	2.6	6.5	5.6	3.7	6.8	8.0
Japanese encephalitis virus infection	1	0	0	0	1	4	0.4	10.0	<0.1	–	–	–	<0.1	<0.1
Kunjin virus infection[††]	1	2	2	2	0	3	1.4	2.1	<0.1	<0.1	<0.1	<0.1	–	<0.1
Malaria	529	504	404	418	345	414	440.0	0.9	2.5	2.3	1.8	1.9	1.5	1.8

Table 6 (cont'd): Notified cases and notification rate for communicable diseases, Australia, 2008 to 2013

Disease	Number of notified cases						5-year mean	Ratio (2013: 5-year mean)	Notification rate per 100,000					
	2008	2009	2010	2011	2012	2013			2008	2009	2010	2011	2012	2013
Murray Valley encephalitis virus infection††	2	4	0	16	1	1	4.6	0.2	<0.1	<0.1	–	0.1	<0.1	<0.1
Ross River virus infection	5,612	4,742	5,129	5,136	4,686	4,308	5,061.0	0.9	26.1	21.9	23.3	23.0	20.6	18.6
Zoonoses														
Anthrax	0	0	1	0	0	0	0.2	–	–	–	<0.1	–	–	–
Australia bat lyssavirus infection	0	0	0	0	0	1	0.0	–	–	–	–	–	–	<0.1
Brucellosis	46	32	21	38	30	14	33.4	0.4	0.2	0.1	0.1	0.2	0.1	0.1
Leptospirosis	111	141	131	215	115	95	142.6	0.7	0.5	0.7	0.6	1.0	0.5	0.4
Lyssavirus infection (NEC)	0	0	0	0	0	0	0.0	–	–	–	–	–	–	–
Ornithosis	102	65	61	91	76	47	79.0	0.6	0.5	0.3	0.3	0.4	0.3	0.2
Q fever	378	313	336	350	362	477	347.8	1.4	1.8	1.4	1.5	1.6	1.6	2.1
Tularaemia	0	0	0	2	0	0	0.4	–	–	–	–	<0.1	–	–
Other bacterial diseases														
Legionellosis	272	301	306	360	383	505	324.4	1.6	1.3	1.4	1.4	1.6	1.7	2.2
Leprosy	11	5	10	10	7	13	8.6	1.5	0.1	<0.1	<0.1	<0.1	<0.1	0.1
Meningococcal infection‡‡	287	260	228	242	222	149	247.8	0.6	1.3	1.2	1.0	1.1	1.0	0.6
Tuberculosis	1,217	1,306	1,367	1,384	1,323	1,265	1,319.4	1.0	5.7	6.0	6.2	6.2	5.8	5.5
Total	159,271	237,729	210,283	238,606	244,424	224,429								

* Newly acquired hepatitis and syphilis < 2 years duration includes cases where the infection was determined to be acquired within 24 months prior to diagnosis.

† Unspecified hepatitis and syphilis includes cases where the duration of infection could not be determined or is greater than 24 months.

‡ In Queensland, includes newly acquired hepatitis C cases.

§ Infection with Shiga toxin/verotoxin producing Escherichia coli (STEC/VTEC).

|| Includes Chlamydia trachomatis identified from cervical, rectal, urine, urethral, throat and eye samples, except for South Australia, which reports only cervical, urine and urethral specimens; the Northern Territory and Western Australia exclude ocular infections. From 1 July 2013 case definition changed to exclude all ocular infections.

¶ The national case definitions for chlamydial, 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).

** Data for all states and territories are reported by diagnosis date, except Queensland which is reported by notification receive date.

†† In the Australian Capital Territory, Murray Valley encephalitis virus infection and Kunjin virus infection are combined under Murray Valley encephalitis virus infection.

‡‡ Only invasive meningococcal disease is nationally notifiable. However the Australian Capital Territory and New South Wales also report conjunctival cases.

HPAIH Highly pathogenic avian influenza in humans.

NEC Not elsewhere classified.

NN Not notifiable.

Data completeness

In 2013, sex and age at onset was complete for 99.8% of notifications in NNDSS (Table 7).

Indigenous status

Indigenous status is usually obtained from medical notification and completeness varies by disease and by state and territory. This reflects differences in notification requirements (i.e. depending on the jurisdiction, some diseases are primarily or completely notified by pathology laboratories rather than clinicians) and the fact that it is not possible to follow up all cases for diseases with a large volume of notifications and/or not requiring specific case based public health action.

Indigenous status was complete in 47.6% of all notifications reported to NNDSS in 2013. Indigenous status was complete in 94.6% of data reported in the Northern Territory, 92% in Western Australia and 90.7% in South Australia. In the remaining jurisdictions, Indigenous status completeness ranged from 18.2% to 47.3% (Table 8).

Data completeness on Indigenous status also varied by disease as summarised in Appendix 3. In 2013, CDNA set target thresholds of 95% completeness for 18 priority diseases (17 notifiable to NNDSS and one, HIV, which is notified to the Kirby Institute) (Table 9) and 80% completeness for the remainder of the notifiable diseases. Of all diseases there were 33 diseases that equalled or exceeded 80% completeness for Indigenous status and 33% (11/33) were priority diseases.

In 2013, 7 of the 17 priority diseases notified to NNDSS had an Indigenous completeness that exceeded 95% (hepatitis A, meningococcal infection, pneumococcal disease < 5 years, pneumococcal disease ≥ 50 years, syphilis – congenital, leprosy, and tuberculosis).

Bloodborne diseases

In 2013, the bloodborne viruses reported to the NNDSS were hepatitis B, C, and D. Both hepatitis B and C cases were notified to the NNDSS as either 'newly acquired', where evidence was available that the infection was acquired in the 24 months prior to diagnosis; or 'greater than 2 years or unspecified' period of infection. These categories were reported from all states and territories except Queensland where all cases of hepatitis C, including newly acquired, were reported as being 'greater than 2 years or unspecified'. The determination of a

Table 7: Completeness of National Notifiable Diseases Surveillance System data, Australia, 2013, by state or territory

	State or territory								
	ACT	NSW	NT	Qld	SA	Tas.	Vic.	WA	Aust.
Total notifications	3,493	50,541	8,290	55,009	18,573	4,224	53,943	30,361	224,434
Sex									
Unknown/ missing	0	59	0	0	76	0	169	1	305
Per cent complete	100.0	99.8	100.0	100.0	99.0	100.0	99.7	>99.9	99.8
Age at onset*									
Unknown/ missing	0	20	0	0	76	0	182	1	279
Per cent complete	100.0	>99.9	100.0	100.0	99.0	100.0	99.7	>99.9	99.8

* For many diseases onset date in unknown, but is calculated using the diagnosis date derived by the diagnosis date algorithm.

Table 8: Indigenous status completeness of National Notifiable Diseases Surveillance System data, Australia, 2013, by state or territory

	State or territory								
	ACT	NSW	NT	Qld	SA	Tas.	Vic.	WA	Aust.
Total notifications	3,493	50,541	8,290	55,009	18,573	4,224	53,943	30,361	224,434
Indigenous status									
Unknown/ missing	2,507	41,325	446	29,741	1,720	2,227	37,139	2,442	117,547
Per cent complete	28.2	18.2	94.6	45.9	90.7	47.3	31.2	92.0	47.6

Table 9: Percentage completeness of priority diseases for Indigenous status of National Notifiable Diseases Surveillance System data, Australia, 2013, by state or territory

Priority disease	ACT	NSW	NT	Qld	SA	Tas.	Vic.	WA	Aust.
Dengue virus (locally acquired)	No cases	100.0	No cases	91.4	100.0	No cases	100.0	50.0	91.5
Donovanosis	No cases	No cases	No cases	No cases	No cases	No cases	No cases	No cases	No cases
Gonococcal infection	100.0	56.0	98.1	62.0	90.9	95.7	61.0	99.9	71.9
Haemophilus influenzae type b	No cases	100.0	No cases	85.7	No cases	No cases	75.0	No cases	90.0
Leprosy	100.0	100.0	100.0	100.0	100.0	No cases	100.0	100.0	100.0
Measles	100.0	100.0	No cases	76.9	100.0	No cases	92.7	100.0	90.5
Meningococcal disease (invasive)	100.0	100.0	100.0	97.0	100.0	100.0	84.0	100.0	96.6
Pertussis <5 years	87.5	94.6	100.0	67.2	100.0	100.0	78.8	98.7	85.6
Shigellosis	100.0	85.9	99.1	76.7	100.0	33.3	91.3	98.6	90.6
Tuberculosis	94.4	100.0	100.0	100.0	100.0	100.0	100.0	100.0	99.9
Hepatitis A	100.0	100.0	No cases	82.2	100.0	No cases	98.1	100.0	95.2
Hepatitis B (newly acquired)	100.0	81.8	100.0	55.6	87.5	100.0	94.1	100.0	83.1
Hepatitis C (newly acquired)	100.0	81.4	100.0	No cases	95.2	89.5	66.2	100.0	84.8
Syphilis – congenital	No cases	100.0	100.0	100.0	No cases	No cases	No cases	100.0	100.0
HIV	NDP	NDP	NDP	NDP	NDP	NDP	NDP	NDP	NDP
Pneumococcal disease <5 years	100.0	100.0	100.0	97.7	85.7	100.0	89.7	100.0	96.8
Pneumococcal disease ≥50 years	100.0	99.7	100.0	88.8	97.0	100.0	89.7	100.0	95.1
Syphilis < 2 years	100.0	92.5	100.0	94.5	80.4	100.0	85.9	100.0	90.6

NDP – No data provided

case as 'newly acquired' is outlined in the national surveillance case definitions.[18] The determination of a case as newly acquired is heavily reliant on public health follow-up, with the method and intensity of follow-up varying by jurisdiction and over time.

In interpreting these data it is important to note that changes in notified cases over time may not solely reflect changes in disease prevalence or incidence. National testing policies developed by the Australian Society for HIV Medicine[19,20] and screening programs, including the preferential testing of high risk populations such as prisoners, injecting drug users and persons from countries with a high prevalence of hepatitis B or C, may contribute to these changes.

Information on exposure factors relating to the most likely source(s) of, or risk factors for, infection for hepatitis B and C was reported in a subset of diagnoses of newly acquired infections. The collection of enhanced data is also dependent on the level of public health follow-up, which is variable by jurisdiction and over time.

Notifications of HIV and AIDS diagnoses were reported directly to The Kirby Institute, which maintains the National HIV Registry. Information on national HIV and AIDS surveillance can be obtained from the Kirby Institute web site (http://www.kirby.unsw.edu.au/).

Hepatitis B

- 7,151 cases of hepatitis B were notified in 2013.
- Over the past 11 years, notifications of newly acquired hepatitis B have declined.

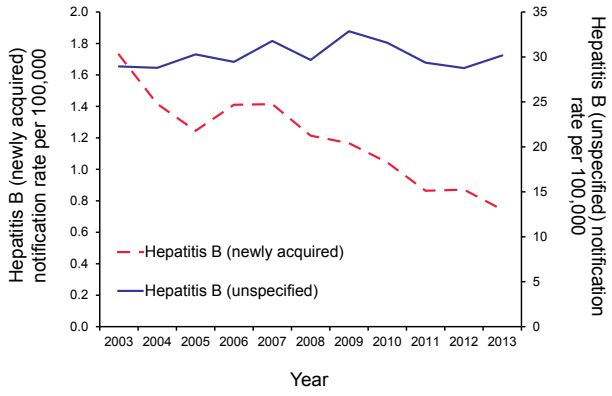
Infection with hepatitis B virus causes inflammation of the liver.[21] Notifications of acute hepatitis B are classified as 'newly acquired' and chronic infections as 'unspecified'.

Epidemiological situation in 2013

In 2013, there were 7,151 notified cases of hepatitis B (both newly acquired and unspecified), equating to a rate of 30.9 cases per 100,000 (Figure 3).

Between 2003 and 2013, newly acquired hepatitis B rates decreased 57% from 1.7 to 0.7 per 100,000 (Figure 3). The continued decline in newly acquired hepatitis B notifications may be attributed to the hepatitis B vaccination program, which was introduced nationally for infants in 2000, and the adolescent hepatitis B vaccination program, which was introduced in 1997.[22] As at 30 June 2014, approximately 92% of children 12–15 months of age in Australia were assessed as being fully immunised for hepatitis B.[23] A 2007 study showed significant improvements in immunity for the 12–17 years age range in jurisdictions with established school-based programs, compared with those jurisdictions without such programs.[24]

Figure 3: Notification rate for newly acquired hepatitis B* and unspecified hepatitis B,† Australia, 2003 to 2013, by year



* Data for newly acquired hepatitis B for the Northern Territory (2003–2004) includes some unspecified hepatitis B cases.

† Data for unspecified hepatitis B for all states and territories, excluding the Northern Territory between 2003 and 2004.

In Australia, hepatitis B vaccination was also recommended for certain at-risk adults from the 1980s, with the list of groups and occupations identified as at-risk varying over time.[25] Some jurisdictions implemented vaccination programs to target identified at-risk adults in a variety of settings and at various times.[22] The full impact of Australian vaccination programs from the 1990s should be reflected in trends in chronic infection and reductions in hepatitis B related complications in the near future.[26]

Between 2003 and 2013, unspecified hepatitis B rates remained relatively stable, increasing slightly by 4.2% from 29.0 to 30.2 per 100,000. It is important to note the significant impact of immigration on rates for unspecified hepatitis B. In 2011, an Australian study found that more than 95% of new cases of chronic hepatitis B virus infection entered the population through migration.[27] While many cases of unspecified hepatitis B go undiagnosed, there is also the potential for duplication, with the National Hepatitis B Testing Policy encouraging clinicians to use patient records to prevent duplication of testing for people from culturally and linguistically diverse backgrounds.[19]

Newly acquired hepatitis B

- 172 cases of newly acquired hepatitis B were notified in 2013.
- The highest rates were in males aged 25–44 years.

Epidemiological situation in 2013

In 2013, 172 newly acquired hepatitis B notifications (0.7 per 100,000) were reported to the NNDSS, a 15% decrease compared with the 198 cases (0.9 per 100,000) reported in 2012 and a continuation of the downward trend in notification rates (Figure 3).

Geographical distribution

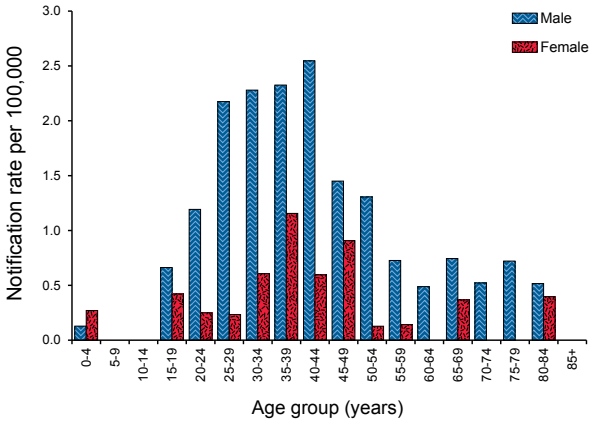
The highest rates were reported from the Northern Territory (2.5 per 100,000) and Western Australia (1.5 per 100,000).

Age and sex distribution

Overall, notification rates were higher among males than females, with a male to female ratio of 3.3:1. In 2013, the highest rates of newly acquired hepatitis B infection were observed among males aged 40–44 years, 30–39 years and 25–29 years (2.5, 2.3 and 2.2 per 100,000 respectively) (Figure 4).

Exposure to hepatitis B may be more common in certain high risk groups, including men who have sex with men; injecting drug users; Aboriginal and Torres Strait Islander peoples; prisoners; and immigrants from endemic regions.[21,27] The greater representation of males in some of these groups may contribute to the higher notification rates among males.

Figure 4: Notification rate for newly acquired hepatitis B, Australia, 2013, by age group and sex*



* Excludes 1 notification where sex was not reported.

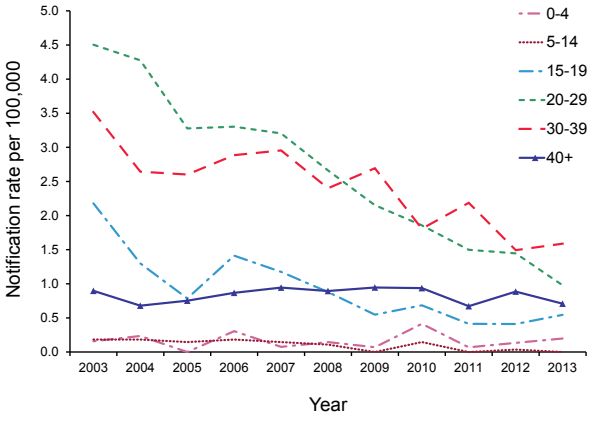
Between 2003 and 2013, most age group specific notification rates were trending downwards. The most marked decreases occurred among those aged 15–19 years and 20–29 years. During this period, notification rates among the 20–29 years age group declined by 78% from 4.5 to 1.0 per 100,000 and notification rates among the 15–19 years age group declined by 75% from 2.2 to 0.5 per 100,000 (Figure 5). These declines are likely to be attributable in part to the adolescent hepatitis B vaccination program.[28]

Risk groups

Enhanced data on risk factors and country of birth was provided by the Australian Capital Territory, New South Wales, South Australia, Tasmania, Victoria and Western Australia* (Table 10). In 2013, 44% (n=76) of these cases had at least 1 risk factor recorded, with a potential source of exposure not recorded or unable to be determined for the remainder. Injecting drug use was the most frequently reported potential source of infection (47%), followed by skin penetration procedures (20%), which includes tattoos, ear or body piercing and acupuncture. Of the 106 cases for which country of birth was reported, 82 were in Australian born persons (77.4%) and 24 cases were born overseas.

* Prior to 2009 enhanced hepatitis B surveillance data were reported to the Kirby Institute from health authorities in the states and territories.

Figure 5: Notification rate for newly acquired hepatitis B,* Australia, 2003 to 2013, by year and selected age groups



* Data for newly acquired hepatitis B for the Northern Territory (2003–2004) includes some unspecified hepatitis B cases.

Unspecified hepatitis B

- 6,979 cases of unspecified hepatitis B were notified in 2013.
- The highest rates were in males aged 30–34 years.

Epidemiological situation in 2013

In 2013, 6,979 cases of unspecified hepatitis B infection were notified to the NNDSS, a rate of 30.2 per 100,000, compared with 6,538 cases (28.8 per 100,000) reported in 2012.

Geographical distribution

In 2013, the Northern Territory had the highest rate of unspecified hepatitis B infection (134.7 per 100,000) (Table 5).

Age and sex distribution

In 2013, the overall male rate (34.9 per 100,000) was higher than for females (25.2 per 100,000), a rate ratio of 1.4:1. Notification rates were higher among males in most age groups, peaking in males aged 30–34 years. For females, the peak notification rate occurred among those aged 25–34 years (Figure 6).

Table 10: Notifications of newly acquired hepatitis B, selected jurisdictions,* 2013, by sex and risk factors†,‡

Exposure category	Number of exposure factors reported			Percentage of total cases (n=76)‖
	Male	Female	Total§	
Injecting drug use	27	9	36	47
Imprisonment	2	0	2	3
Skin penetration procedure	12	3	15	20
Tattoos	8	1	9	12
Ear or body piercing	3	2	5	7
Acupuncture	1	0	1	1
Healthcare exposure	1	2	3	4
Surgical work	1	0	1	1
Major dental surgery work	0	2	2	3
Sexual exposure	8	1	9	12
Sexual contact (hepatitis B positive partner) – opposite sex	3	1	4	5
Sexual contact (hepatitis B positive partner) – same sex	5	0	5	7
Other	22	4	28	37
Household contact	1	0	1	1
Needlestick/biohazardous injury¶	2	0	2	3
Perinatal transmission	1	1	3	4
Other – not further categorised	18	3	22	29
Cases with at least 1 exposure	54	15	69	91
Undetermined	5	2	7	9
Unknown*	12	6	18	–
Total exposure factors reported	77	21	100	–
Total number of cases	59	17	76	–

* Cases from the Australian Capital Territory, New South Wales, South Australia, Tasmania, Victoria and Western Australia. While these jurisdictions provided enhanced data on risk factors, not all cases had this information recorded.

† More than 1 exposure category for each case could be recorded.

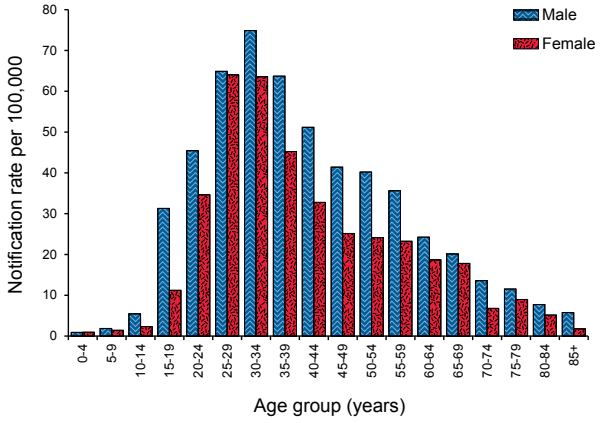
‡ Analysis and categorisation of these exposures are subject to interpretation and may vary.

§ Total includes cases where no sex was reported.

‖ The denominator used to calculate the percentage is based on the total number of cases from all jurisdictions that provide enhanced data (Australian Capital Territory, New South Wales, South Australia, Tasmania, Victoria and Western Australia). As more than 1 exposure category for each notification could be recorded, the total percentage does not equal 100%.

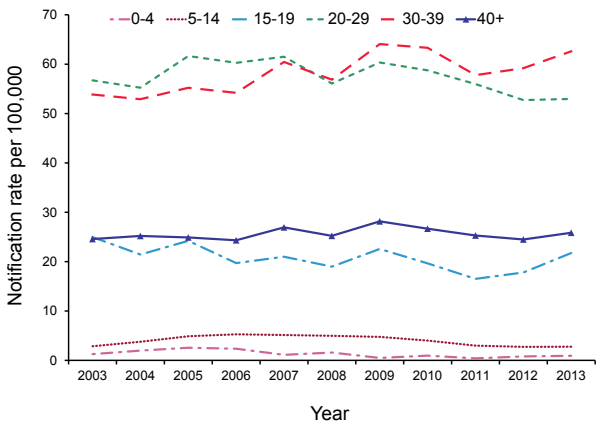
¶ Includes both occupational and non-occupational exposures.

Figure 6: Notification rate for unspecified hepatitis B, Australia, 2013, by age group and sex*



* Excludes 41 cases where age and/or sex were not reported.

Figure 7: Notification rate for unspecified hepatitis B,* Australia, 2003 to 2013, by year and age group†



* Data for hepatitis B (unspecified) from all states and territories except the Northern Territory between 2003–2004.

† Excludes 43 cases where age was not known.

Between 2003 and 2013, notification rates for unspecified hepatitis B remained relatively stable for most age groups. However, there has been a slight upward trend in the notification rate for the 15–19 years age group (from 16.5 to 21.7 per 100,000) and the 30–39 years age groups (from 53.9 to 62.6 per 100,000) (Figure 7).

Hepatitis C

- 10,715 cases of hepatitis C were notified in 2013.
- Over the past 11 years, notifications of hepatitis C have declined by 33%.

Infection with hepatitis C virus causes inflammation of the liver. In more than 90% of cases initial infection with hepatitis C virus is asymptomatic or mildly symptomatic. Approximately 50%–80% of cases go on to develop a chronic infection. Of those who develop a chronic infection, half will eventually develop cirrhosis or cancer of the liver.[21]

Hepatitis C notifications are classified as being either 'newly acquired' (evidence that infection was acquired within the 24 months prior to diagnosis) or 'unspecified' (infection acquired more than 24 months prior to diagnosis or not able to be specified). Ascertaining a person's hepatitis C serostatus and clinical history usually requires active follow-up by public health units.

Epidemiological situation in 2013

Between 2003 and 2013, hepatitis C notifications declined by 33% from 13,748 (69.7 per 100,000) to 10,715 (46.8 per 100,000). This declining trend is reflected in both newly acquired and unspecified hepatitis C notifications (Figure 8).

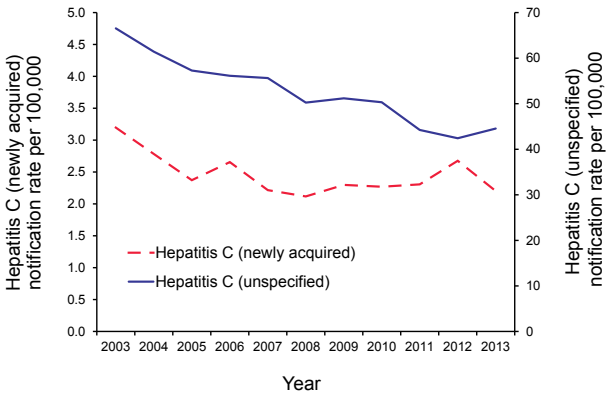
Newly acquired hepatitis C

- 407 cases of newly acquired hepatitis C were notified in 2013.
- The majority of newly acquired cases in 2013 had a history of injecting drug use.
- The highest notification rates in 2013 were among males in the 20–24 years age group.

Epidemiological situation in 2013

Cases of newly acquired hepatitis C were reported from all states and territories except Queensland, where all cases of hepatitis C are reported as unspecified. Nationally, there were 407 notifications in 2013 (2.2 per 100,000) compared with 486 notifications in 2012 (2.7 per 100,000).

Figure 8: Notification rate for hepatitis C (newly acquired* and unspecified†), Australia, 2003 to 2013, by year



* Data for newly acquired hepatitis C from all states and territories except Queensland 2003–2013 and the Northern Territory 2003–2004.

† Data for unspecified hepatitis C provided from Queensland (2003–2013) and the Northern Territory (2003–2004) includes both newly acquired and unspecified hepatitis C cases.

Geographical distribution

The highest rates of newly acquired hepatitis C infection were reported in Western Australia (4.9 per 100,000), South Australia, Tasmania and the Australian Capital Territory (all 3.7 per 100,000). The identification and classification of newly acquired hepatitis C is reliant upon public health follow-up to identify testing and clinical histories.

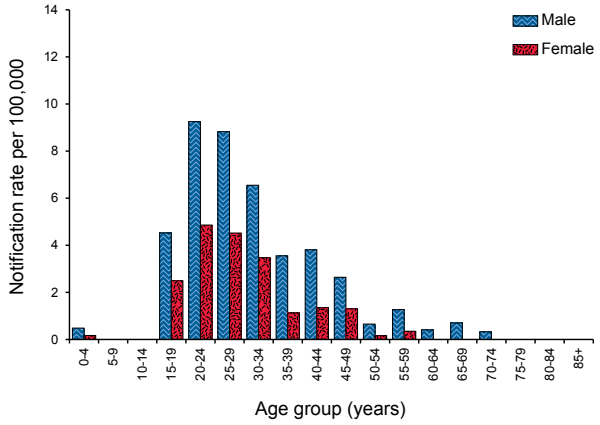
Age and sex distribution

Nationally in 2013, the notification rate for newly acquired hepatitis C in males was 3.0 per 100,000 and in females was 1.4 per 100,000, a male to female ratio of 2.2:1. Notification rates in males exceeded those in females across all age groups for which there were cases. The highest notification rates among males and females were in the 20–24 years (9.3 and 4.3 per 100,000 respectively), 25–29 years (8.8 and 4.5 per 100,000 respectively), and 30–34 years (6.5 and 3.5 per 100,000 respectively) age groups (Figure 9).

Between 2003 and 2013, notification rates for newly acquired hepatitis C have declined overall among those in the 15–39 years age groups The largest decreases from 2003 to 2013 occurred in the 15–19 years age groups (from 6.5 to 3.5 per 100,000), and the 20–29 years age groups (from 11.7

to 6.9 per 100,000). A recent survey suggested there has been a decrease in the prevalence of injecting drug use among young people in Australia.[28] Notification rates in the 0–4 and the 40 years or over age groups have remained low and relatively stable over this time (Figure 10).

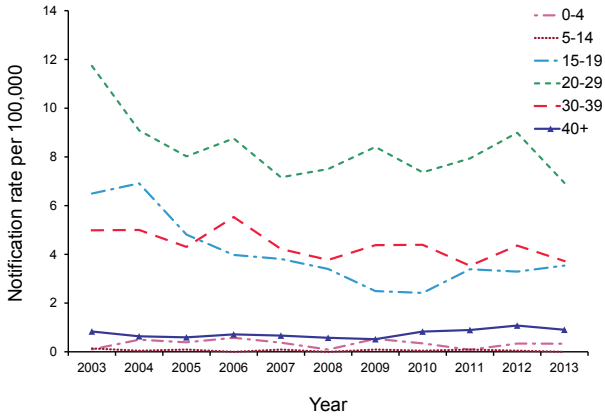
Figure 9: Notification rate for newly acquired hepatitis C, Australia,* 2013, by age group and sex†



* Data from all states and territories except Queensland.

† Excludes 2 cases where age and/or sex were not reported.

Figure 10: Notification rate for newly acquired hepatitis C, Australia,* 2003 to 2013, by year and selected age groups



* Data from all states and territories except Queensland (2003–2013) and the Northern Territory (2003–2004).

Risk groups

Exposure histories for newly acquired hepatitis C cases reported in 2013 were analysed for all jurisdictions except Queensland (notified as unspecified hepatitis C), Western Australia (no exposure data notified) and the Northern Territory (data not available at time of analysis) (Table 11). In 2013, 71.5% of cases had at least 1 risk factor recorded, with the potential source of exposure not recorded or unable to be determined for the remainder. Of the cases for which exposure history was reported, approximately 67% had a history of injecting drug use and approximately 32% reported skin penetration procedures.

Approximately 38% (n=111) of cases with exposure history had reported imprisonment. Of these cases, approximately 47% (n=52) had also reported a history of injecting drug use. However, it is important to note that screening rates are generally higher in the prison entry population than the general population. A screening survey of prison entrants conducted over a two-week period found that the prevalence of hepatitis C, based on hepatitis C antibody detection, was 22% in 2012, a decrease from 35% in 2007.[29]

Unspecified hepatitis C

- 10,308 cases of unspecified hepatitis C were notified in 2013.
- The highest notification rates in 2013 were among males in the 30–39 years age groups.

Epidemiological situation in 2013

In 2013, 10,308 cases of unspecified hepatitis C infections were notified to the NNDSS (44.6 per 100,000) compared with 9,641 cases in 2012 (42.4 per 100,000). Apart from the slight rise from 2012 to 2013, notification rates have decreased annually since 2003, with an overall decline of 33% between 2003 (66.5 per 100,000) and 2013 (44.6 per 100,000) (Figure 11).

Several factors may account for the decrease including changes in surveillance practices, removal of duplicate notifications and a gradual decline in the prevalent group of hepatitis C cases accumulated prior to the introduction of hepatitis C testing in the early 1990s.[24,30] The continuing decline in the notification rate may also be attributable to an apparent decrease in the prevalence of injecting drug use among young people in Australia.[28]

Geographical distribution

In 2013, the Northern Territory continued to have the highest notification rate (106.1 per 100,000).

Age and sex distribution

Nationally in 2013, the notification rate for unspecified hepatitis C in males was 58.5 per 100,000

Table 11: Notified cases of newly acquired hepatitis C, selected jurisdictions,* 2013, by sex and risk factors†,‡

Exposure category	Number of exposure factors reported			Percentage of total cases (n=291)§
	Male	Female	Total	
Injecting drug use	142	53	195	67
Imprisonment	100	11	111	38
Skin penetration procedure	71	22	93	32
Tattoos	54	13	67	23
Ear or body piercing	17	9	26	9
Health care exposure	9	7	16	6
Surgical work	5	6	11	4
Major dental surgery work	3	1	4	1
Haemodialysis	1	0	1	<1
Sexual exposure	21	20	41	14
Sexual contact (hepatitis B positive partner) – opposite sex	9	19	28	10
Sexual contact (hepatitis B positive partner) – same sex	12	1	13	5
Other	68	35	103	35
Household contact	14	10	24	8
Needlestick/biohazardous injury‖	10	3	13	5
Perinatal transmission‡	17	15	32	11
Other – not further specified‡	27	7	34	12
Cases with at least 1 exposure	202	81	283	97
Undetermined	2	6	8	3
Unknown‡	3	4	7	–
Total exposure factors reported	411	148	559	–
Total number of cases	204	87	291	–

* Includes data from all states and territories except Queensland (not notified), the Northern Territory (data not available at time of analysis) and Western Australia (no enhanced data on risk factors). While 5 jurisdictions provided enhanced data on risk factors, not all cases had this information recorded.

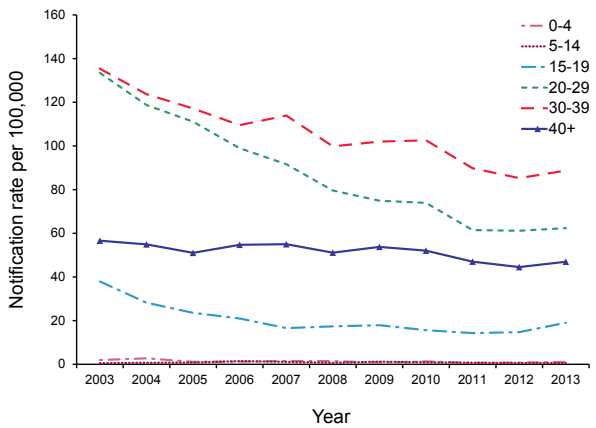
† More than 1 exposure category for each notification could be recorded.

‡ Analysis and categorisation of these exposures are subject to interpretation and may vary.

§ The denominator used to calculate the percentage is based on the total number of notified cases from all jurisdictions, except Queensland (notified as unspecified hepatitis C), Northern Territory (n=0) and Western Australia (no exposure data notified, n=125). As more than 1 exposure category for each case could be recorded, the total percentage does not equate to 100%.

‖ Includes both occupational and non-occupational exposures.

Figure 11: Notification rate for unspecified hepatitis C, Australia,* 2003 to 2013, by selected age groups†



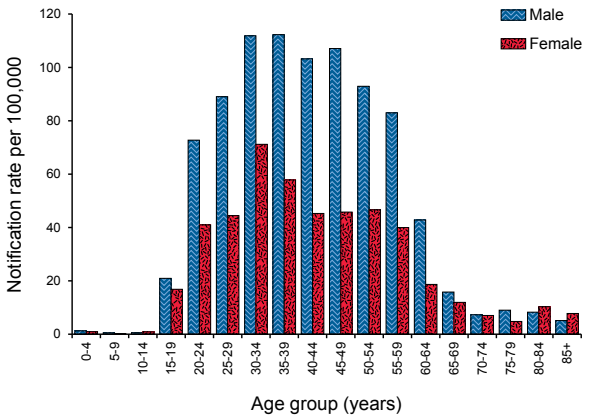
* Data provided from Queensland (2003–2013) and the Northern Territory (2003–2004) includes both newly acquired and unspecified hepatitis C cases.

† Excludes 80 cases where age was not reported.

and in females 30.4 per 100,000, a male to female ratio of 1.9:1. Notification rates in males exceeded those in females across almost all age groups. The highest notification rates were among males in the 35–39 year (112.3 per 100,000) and 30–34 year (111.8 per 100,000) age groups. The highest notification rates among females were for those in the 30–34 years (71.2 per 100,000) and 35–39 years (57.8 per 100,000) age groups (Figure 12).

Between 2003 and 2013, notifications rates for unspecified hepatitis C have declined overall across all age groups, except for the 0–4 years, 5–14 year and 40+ years age groups for which rates have remained relatively stable (Figure 11). The largest decreases have occurred in the 20–29 years (from 133.5 to 62.4 per 100,000), 30–39 years (135.4 to 88.7 per 100,000) and 15–19 years (37.9 to 19.0 per 100,000) age groups.

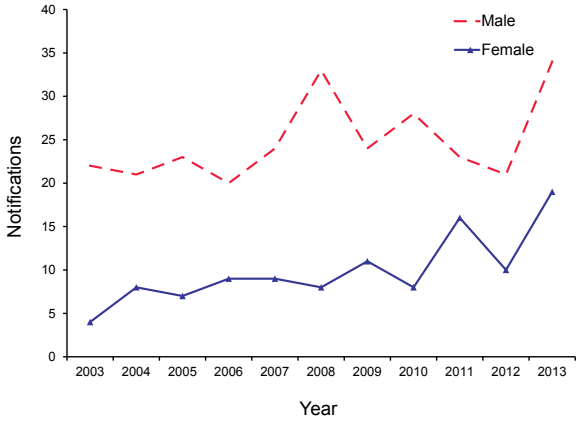
Figure 12: Notification rate for unspecified hepatitis C,* Australia, 2013, by age group and sex†



* Data provided from Queensland includes both newly acquired and unspecified hepatitis C cases.

† Excludes 38 cases where age and/or sex was missing or unknown.

Figure 13: Notified cases of hepatitis D, Australia, 2003 to 2013, by year and sex



Hepatitis D

- 53 cases of hepatitis D were notified in 2013.
- Hepatitis D is always associated with hepatitis B co-infection.

Hepatitis D is a defective single-stranded RNA virus that replicates in the presence of the hepatitis B virus. Hepatitis D infection can occur as either an acute co-infection with hepatitis B or as a super-infection with chronic hepatitis B infection. The modes of hepatitis D transmission are similar to those for hepatitis B.[21]

Epidemiological situation in 2013

In Australia, the notification rate for hepatitis D remains low. In 2013, there were 53 notified cases of hepatitis D, a rate of 0.2 per 100,000. Over the preceding 10 years, notifications of hepatitis D remained relatively low with an average of almost 35 cases notified per year (range: 26 to 53).

Geographical distribution

In 2013, Victoria reported the highest number of cases (22) followed by Queensland (13), New South Wales (9), South Australia and Western Australia (both 4) and the Northern Territory (1). No cases were reported from the Australian Capital Territory or Tasmania during this period.

Age and sex distribution

The male to female ratio in 2013 was 1.8:1. This was less than the average ratio of 2.7:1 over the preceding 5 years (Figure 13).

Gastrointestinal diseases

Overview

In 2013, gastrointestinal diseases notified to NNDSS and discussed in this section were: botulism, campylobacteriosis, cryptosporidiosis, haemolytic uraemic syndrome (HUS), hepatitis A, hepatitis E, listeriosis, salmonellosis, shigellosis, Shiga toxin-producing *Escherichia coli* (STEC) infections and typhoid fever.

Overall, notified cases of gastrointestinal diseases increased from 31,155 in 2012 to 32,535 in 2013. Notifications for salmonellosis, typhoid fever and STEC were at the highest levels since NNDSS records began in 1991.

Surveillance systems overview

The Australian Government established OzFoodNet—Australia's enhanced foodborne disease surveillance system—in 2000 as a collaborative network of epidemiologists and microbiologists who conduct enhanced surveillance, epidemiological outbreak investigations and applied research into foodborne disease across Australia. OzFoodNet's mission is to apply concentrated effort at the national level to investigate and understand foodborne disease, to describe its epidemiology more effectively and to identify ways to minimise foodborne illness in Australia. The data and results summarised in the following sections will be reported in more detail in the OzFoodNet annual report 2013.

Botulism

- 4 cases of botulism were notified in 2013.

Botulism is a rare but extremely serious intoxication resulting from toxins produced by *Clostridium*

botulinum (commonly toxin types A, B and E). Four forms of botulism are recognised; infant, foodborne, wound and adult intestinal toxaemia.[21]

Epidemiological situation in 2013

There were 4 notified cases of botulism in 2013; all four were infant botulism but no links between them were identified. This compares with no notified cases in 2012 and 2 infant botulism cases in 2011.

Campylobacteriosis

- 14,698 cases of campylobacteriosis were notified in 2013.
- This was the most frequently notified enteric infection in 2013.

The bacterium *Campylobacter* is a common cause of foodborne illness (campylobacteriosis) in humans. The severity of this illness varies and is characterised by diarrhoea (often bloody), abdominal pain, fever, nausea and or vomiting.[21] Campylobacteriosis is notifiable in all Australian states and territories except New South Wales.

Epidemiological situation in 2013

There were 14,698 notified cases of campylobacteriosis in 2013 making it the most frequently notified enteric infection (93.5 per 100,000 not including New South Wales). This was a decrease of 6.1% on the number of notifications received for 2012 (n=15,655) and a 10.4% decrease on the 5-year mean (n=16,407). Notification rates ranged from 76.5 per 100,000 in Western Australia to 135.6 per 100,000 in Tasmania.

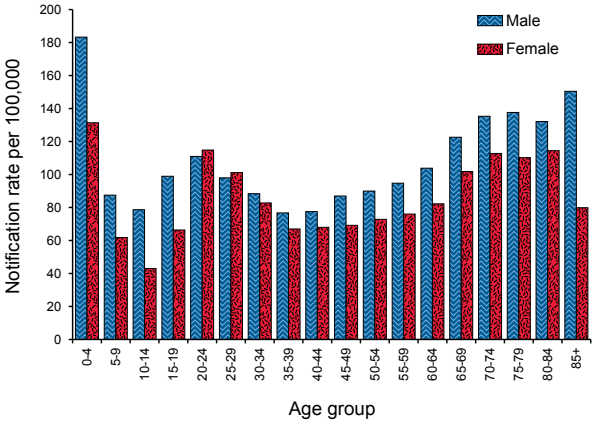
Age and sex distribution

Campylobacteriosis was most frequently notified among the 0–4 age group for both males (183.3 per 100,000) and females (131.3 per 100,000). The median age of notified cases was 28 years and 54.8% (n= 8,056) were male. Notification rates were highest among males in nearly all age groups (Figure 14).

Cryptosporidiosis

- 3,846 cases of cryptosporidiosis were notified in 2013.
- There was 1 outbreak in New South Wales.

Figure 14: Notification rate for campylobacteriosis, Australia, 2013, by age group and sex



Cryptosporidiosis is a parasitic infection characterised by abdominal cramping and usually large-volume watery diarrhoea. Ingesting contaminated water, typically from a recreational source like a community swimming pool or lake is a major risk factor for infection.[21]

Epidemiological situation in 2013

There were 3,846 notified cases of cryptosporidiosis in 2013 (16.6 per 100,000). This represents a 23.1% increase over the 3,124 cases reported in 2012 and a 47.2% increase over the 5-year mean of 2,612 cases. Notification rates ranged from 8.1 per 100,000 in South Australia to 36.9 per 100,000 in the Northern Territory.

Age and sex distribution

In 2013, notified cases of cryptosporidiosis were most frequently reported among the 0–4 years age group (36.6%, n=1,407) and of these, 58.8% (n=828) were male. This was consistent with 2012 figures when notifications of cryptosporidiosis were also most frequent in the 0–4 years age group (45.8%, n=1,437), and the majority of these were male (59.0%, n=848).

Outbreaks

An outbreak of cryptosporidiosis occurred in New South Wales in the 1st quarter of 2013, associated with community swimming pools across New South Wales and particularly in north-eastern Sydney.[31]

Haemolytic uraemic syndrome

- 15 cases of haemolytic uraemic syndrome were notified in 2013.
- Cases were most frequently notified among the 0–4 years age group.

HUS is a rare but serious illness that is characterised by acute renal impairment; with 50% of patients requiring dialysis and about 5% dying.[21] Not all diagnoses of HUS are related to enteric pathogens, but Australian cases are commonly associated with STEC infection.[32] In 2013, 66.7% (10/15) of HUS cases were positive for STEC.

Epidemiological situation in 2013

There were 15 notified cases of HUS in 2013 compared with 20 in 2012 and a mean of 17.2 cases per year between 2008 and 2012. Five of these cases were associated with STEC.

Age and sex distribution

In 2013, HUS was most frequently notified among the 0–4 years age group (27%, n=4), with a median age of 18 years (range 1–82 years). One-third of notified cases were in males (n=5) compared with 70% (n=14) in 2012.

Hepatitis A

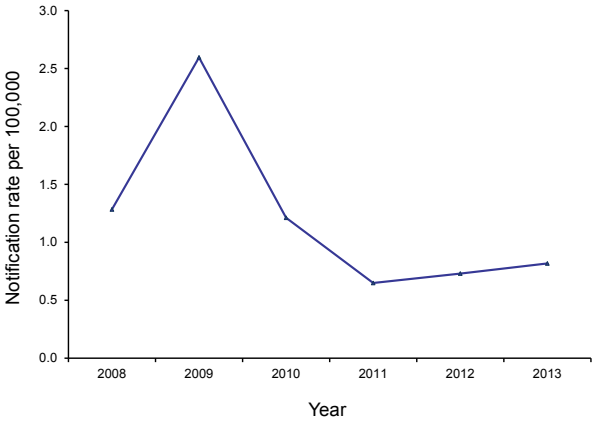
- 189 cases of hepatitis A were notified in 2013.
- Overseas travel was the primary risk factor for notified cases.

Hepatitis A is an acute viral infection primarily of the liver characterised by fever, malaise, anorexia, nausea and abdominal discomfort followed by jaundice. The disease varies from a mild illness to a severely disabling disease lasting several months. Infection is usually spread from person to person via the faecal-oral route but can also be foodborne or waterborne.[21]

Epidemiological situation in 2013

There were 189 notified cases of hepatitis A in 2013 (0.8 per 100,000). This was a 13.8% increase on the number of cases in 2012 (n=166), and a 33.3% decrease on the 5-year mean (n=283.4). The historical mean reflects the impact of a 2009–2010 outbreak of hepatitis A associated with the consumption of semi-dried tomatoes (Figure 15).

Figure 15: Notification rate for hepatitis A, Australia, 2008 to 2013



Age and sex distribution

Hepatitis A was most frequently notified among the 5–9 years age group (n=23) in 2013. The median age of notified cases was 25 years (range 1–79 years), and 59.3% (n=112) of all cases were male compared with 48% in 2012 (n=80).

Indigenous status

Indigenous status was known for 95.2% (n=180) of cases of hepatitis A. Of these, 3 were identified as being Indigenous.

Place of acquisition

Overseas travel was the primary risk factor for notified cases. In 2013, 61.4% (n=116) reported overseas travel during their incubation period for hepatitis A and were considered to have been overseas acquired. Travel to India, the Philippines and Vanuatu were most frequently reported.

In 2013, 20.6% (n=39) of notified cases were locally acquired. This was an increase from 2012 where 18% (n=30) of notified cases were locally acquired (Table 12). A 2009–2010 outbreak associated with

Table 12: Notified cases of hepatitis A, Australia, 2008 to 2013, by place of acquisition

	Locally acquired		Overseas acquired		Unknown		
Year	n	%	n	%	n	%	Total
2008	64	23	121	44	91	33	276
2009	304	54	184	33	75	13	563
2010	112	42	144	54	11	4	267
2011	39	27	97	67	9	6	145
2012	30	18	111	67	25	15	166
2013	39	21	116	61	34	18	189

the consumption of semi-dried tomatoes contributed to an increase in locally acquired hepatitis A cases in those years.[33]

Hepatitis E

- 31 cases of hepatitis E were notified in 2013.
- The first 3 confirmed locally acquired infections occurred in 2013.

Hepatitis E is an acute viral infection primarily of the liver that is transmitted by the faecal-oral route, most often via food or water. [21] The infection is usually acquired overseas among travellers to endemic areas.

Epidemiological situation in 2013

There were 31 notified cases of hepatitis E in 2013, compared with a 5-year mean of 38 cases.

Age and sex distribution

Hepatitis E was most frequently notified among the 25–39 years age group (n=8), the median age of cases was 32 years (range 4–72 years), and 68% (n=21) of total cases were male.

Place of acquisition

Hepatitis E in Australia has traditionally been associated with overseas travel. In 2013, 87% of cases (n=27) reported overseas travel during their incubation period and were considered to have been acquired overseas, of these, 41% (n=11) reported travel to India. The first 3 confirmed cases of locally acquired hepatitis E occurred in New South Wales in the last quarter of 2013.

Listeriosis

- 76 cases of listeriosis were notified in 2013.
- Notified cases were highest in the 80+ years age group.

Invasive listeriosis is caused by a bacterial infection that commonly affects the elderly or immunocompromised, and typically occurs among people with serious underlying illnesses. Listeriosis can also affect pregnant women and infect their unborn baby. Laboratory-confirmed infections in a mother and her unborn child or neonate are notified separately in the NNDSS.

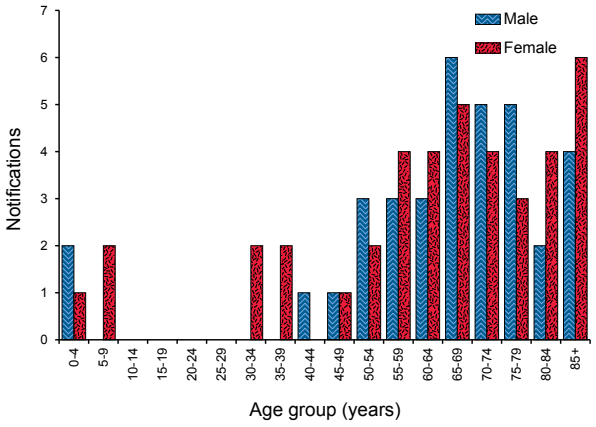
Epidemiological situation in 2013

There were 76 notified cases of invasive *Listeria monocytogenes* infection in 2013 (0.3 per 100,000) compared with a 5-year mean of 78.8 cases.

Age and sex distribution

Notifications for listeriosis were highest in the 80+ years age group (21%, n=16), with 53% (n=40) of all notified cases being female (Figure 16).

Figure 16: Notified cases of listeriosis, Australia, 2013, by age group and sex



Enhanced surveillance datasets

In 2010 OzFoodNet started collecting enhanced surveillance data on all notified cases of listeriosis in Australia. The information collected on cases includes the characterisation of *Listeria monocytogenes* isolates by molecular subtyping methods, food histories and exposure data. The overall aim of this enhanced surveillance is to enable timely detection of outbreaks and subsequent public health response.[32] Further information on OzFoodNet's enhanced *Listeria* surveillance system can be found in OzFoodNet annual reports (http://www.ozfoodnet.gov.au/internet/ozfoodnet/publishing.nsf/Content/reports-1).

Salmonellosis (non-typhoidal)

- 12,791 cases of salmonellosis were notified in 2013.
- Cases were most frequently notified among the 0–4 years age group.

Salmonellosis is a bacterial disease characterised by the rapid development of symptoms including abdominal pain, fever, diarrhoea, muscle pain,

nausea and/or vomiting. People can become infected via faecal-oral transmission, ingesting contaminated food, through animal contact and from environmental exposures. The predominant mode of transmission is contaminated food, mainly of animal origin.[21]

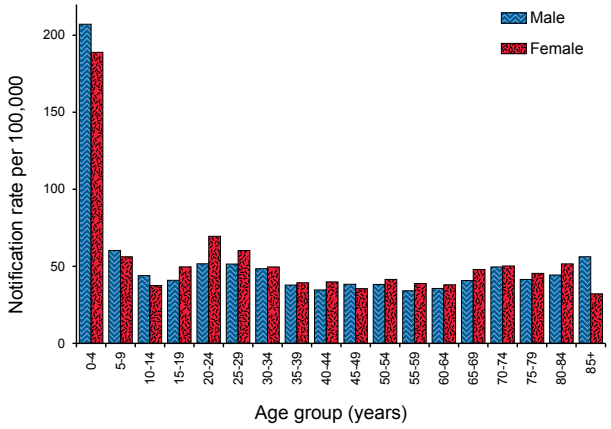
Epidemiological situation in 2013

There were 12,791 notified cases of salmonellosis in 2013 (55.3 per 100,000) with the number of cases being 20.1% higher than the 5-year mean of 10,649 cases. This represents a 13.5% increase in cases compared with 2012 (n=11,265). The number of cases for 2013 was the highest recorded in NNDSS since 1991. Rates ranged from 46.6 per 100,000 in New South Wales to 159.6 per 100,000 in the Northern Territory.

Age and sex distribution

Salmonellosis was most frequently notified among the 0–4 years age group (23.6%, n=3,015), the median age of notified cases was 27 years (range 0–93 years) and 50.5% (n=6,461) of cases where sex was stated were females (Figure 17).

Figure 17: Notification rate for salmonellosis, Australia, 2013, by age group and sex*



* Sex and/or age were not reported for 39 cases.

Shigellosis

- 556 cases of shigellosis were notified in 2013.
- 38% of notified cases were acquired overseas.

Epidemiological situation in 2013

There were 556 notified cases of shigellosis in 2013 (2.4 per 100,000), which was fewer than the 5-year mean of 608 cases. Notification rates ranged from 0.6 per 100,000 in Tasmania to 44.8 per 100,000 in the Northern Territory.

Shigellosis is a bacterial disease characterised by acute abdominal pain and fever, small-volume loose stools, vomiting and tenesmus. *Shigella* is transmitted via the faecal-oral route, either directly (such as male-to-male sexual contact) or indirectly through contaminated food or water.[21]

Age and sex distribution

Notifications for shigellosis were highest in the 0–4 years age group (19.8%, n=110). In 2013, the median age of notified cases was 29 years (range 0–87 years) and 51.5% (n=286) were male.

Indigenous status

Information on Indigenous status was available for 90.6% (n=504) of shigellosis cases. This proportion varied by state or territory, with Queensland and Tasmania being less than 80% complete. Among states and territories with greater than 80% completeness, the proportion of notified cases who identified as being of Aboriginal or Torres Strait Islander origin was 23.9% (115/481).

Place of acquisition

Thirty-nine per cent (n=216) of notified cases of shigellosis were reported as being acquired overseas. The most frequently reported countries of acquisition for imported cases were India (22.2%, n=48) and Indonesia (17.3%, n=38). The place of acquisition for 35.6% (n=198) was inadequately described or unknown, down from 64% (n=530) in 2008 (Table 13).

Shiga toxin-producing *Escherichia coli*

- 180 cases of Shiga toxin-producing *Escherichia coli* were notified in 2013.

Shiga toxin-producing *Escherichia coli* is a common cause of diarrhoeal illness in humans. People can become infected via faecal-oral transmission, ingesting contaminated food, through animal contact and from environmental exposures. Severe illness can progress to HUS. Children under 5 years of age are most frequently diagnosed with infection and are at greatest risk of developing HUS.[21]

Epidemiological situation in 2013

There were 180 notified cases of STEC in 2013 (0.8 per 100,000) compared with a 5-year mean of 102 cases. Of these, 5 cases developed HUS.

Table 13: Notified cases of shigellosis, Australia, 2008 to 2013, by place of acquisition

	Locally acquired		Overseas acquired		Unknown		
Year notified	n	%	n	%	n	%	Total
2008	207	25	93	11	530	64	830
2009	205	33	55	9	356	58	616
2010	153	28	163	30	236	43	552
2011	152	31	133	27	208	42	493
2012	137	25	173	32	238	43	548
2013	142	26	216	39	198	36	556

Detection of STEC infection is strongly influenced by jurisdictional practices regarding the screening of stool specimens.[32] South Australia continues to test all bloody stools for STEC using polymerase chain reaction (PCR) and subsequently has the highest notification rate in the country (3.2 cases per 100,000 compared with between 0.2 and 1.8 cases per 100,000 in other states and territories reporting cases). In addition, Victoria notified cases of HUS caused by STEC as HUS only, whereas all other jurisdictions notify each case as both organisms. These differences in testing practice mean that meaningful comparison of notification data by jurisdiction and over time are not valid.

Age and sex distribution

In 2013, 53% (n=95) of notified STEC cases were female. The median age of notified cases was 23 years (range 0–91 years).

Typhoid

- 150 cases of typhoid were notified in 2013.
- 94% of notified cases were acquired overseas.

Typhoid is a bacterial disease caused by *Salmonella enterica* serotype Typhi. Symptoms include sustained fever, marked headache, malaise and constipation more often than diarrhoea in adults. The transmission mode is the same as for salmonellosis, however typhoid differs in that humans are the reservoir for the bacterium.[21]

Epidemiological situation in 2013

There were 150 notified cases of typhoid in 2013 (0.6 per 100,000), compared with the 5-year mean of 115.2 cases. This was a 21% increase on the number of cases in 2012 (n=124).

Age and sex distribution

Typhoid was most frequently notified among the 10–14 years age group (15%, n=22), the median age of notified cases was 23 years (range 1–58 years), and 51% (n=77) were female.

Place of acquisition

As in previous years, overseas travel was the primary risk factor for notified cases. In 2013, 94% (n=141) reported overseas travel during their exposure period and were considered overseas acquired. India continues to be the most frequently reported country of acquisition, accounting for 61% (n=86) of overseas-acquired cases in 2013. Eight cases (5%) were locally acquired and the place of acquisition was unknown for 1 case (1%).

Quarantinable diseases

Human diseases covered by the *Quarantine Act 1908,* and notifiable in Australia and to the WHO in 2013 were cholera, plague, rabies, yellow fever, smallpox, highly pathogenic avian influenza in humans (HPAIH), severe acute respiratory syndrome (SARS) and 4 viral haemorrhagic fevers (Ebola, Marburg, Lassa and Crimean–Congo). These diseases are of international public health significance.

Travellers are advised to seek information on the risk of contracting these diseases at their destinations and to take appropriate measures. More information on quarantinable diseases and travel health can be found on the Travel Health Information web site (www.health.gov.au/internet/main/publishing.nsf/Content/health-pubhlth-strateg-quaranti-index.htm) and from the Smartraveller website (www.smartraveller.gov.au/).

There were no cases of plague, rabies, smallpox, yellow fever, SARS, HPAIH or viral haemorrhagic fevers reported in Australia in 2013. While there were 3 cases of cholera, Australia remains free of all the listed quarantinable diseases (Table 14).

Table 14: Australia's status for human quarantinable diseases, 2013

Disease	Status	Date of last record and notes
Cholera	Free	Small number of cases reported annually related to overseas travel. Very rare instances of local acquisition as described under the section 'Cholera'.
Plague	Free	Last case recorded in Australia in 1923[34]
Rabies	Free	Last case (overseas acquired) recorded in Australia in 1990[35]
Smallpox	Free	Last case recorded in Australia in 1938, last case worldwide in 1977, declared eradicated by the World Health Organization 1980[36,37]
Yellow fever	Free	Two cases in 2011 were the first recorded, related to overseas travel[38]
SARS	Free	Last case recorded in Australia in 2003[39]
HPAIH	Free	No cases recorded[40]
Viral haemorrhagic fevers		
Ebola	Free	No cases recorded
Marburg	Free	No cases recorded
Lassa	Free	No cases recorded
Crimean–Congo	Free	No cases recorded

Cholera

- 3 cases of cholera were notified in 2013.

Cholera is an infection of the digestive tract (or gut) caused by certain strains of the bacterium *Vibrio cholerae* that produce toxins (poisons) and is most commonly acquired in parts of Africa, Asia, South America, the Middle East and the Pacific islands. *V. cholerae* is found in the faeces of infected people, and is spread by drinking contaminated water, eating food washed with contaminated water or prepared with soiled hands or eating fish or shellfish caught in contaminated water. Person-to-person spread of cholera is less common. Most people do not develop symptoms or have only mild illness but a small proportion of people will develop severe symptoms. Symptoms typically start between 2 hours and 5 days (usually 2–3 days) after ingesting the bacteria. Symptoms can include characteristic 'rice water' faeces (profuse, watery diarrhoea), nausea and vomiting, signs of dehydration, such as weakness, lethargy and muscle cramps. Only toxigenic *V. cholerae* O1 or O139 are notifiable in Australia.

Epidemiological situation in 2013

In 2013, there were 3 notifications of cholera in Australia. There were 23 cases of cholera in total in Australia between 2008 and 2012. The following details are available about the relevant exposures or place of acquisition for the 3 cases in 2013:

- all cases were aged between 20 and 40 years, 2 cases were male and one was female;
- two were reported by New South Wales and one by Victoria;
- the country of acquisition was reported as Bangladesh (2 cases) and Australia;
- the case acquired in Australia was laboratory-acquired.

All cases of cholera reported since the commencement of the NNDSS in 1991 to 2012 have been acquired outside Australia except for 1 case of laboratory-acquired cholera in 1996[41] and 3 cases in 2006 linked to imported whitebait.[42]

Sexually transmissible infections

Overview

In 2013, the STIs reported to the NNDSS were chlamydial infection, donovanosis, gonococcal infection and syphilis. Other national surveillance systems that monitor STIs in Australia include the Australian Gonococcal Surveillance Programme (AGSP), which is a network of specialist laboratories monitoring antimicrobial susceptibility patterns of gonococcal infection; and the Kirby Institute for Infection and Immunity in Society.

Chlamydial infection

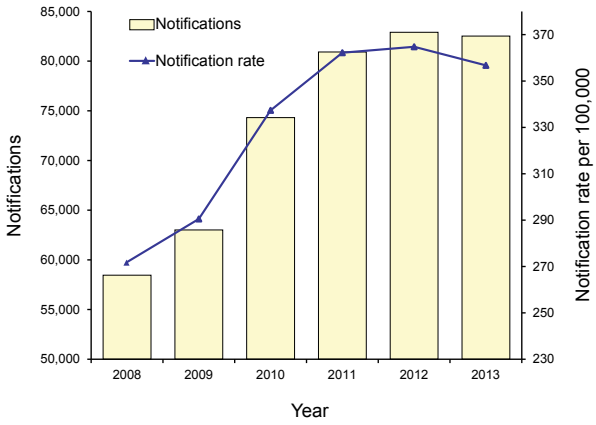
- 82,526 cases of chlamydial infection were notified in 2013.
- 2013 notification rates were similar to 2012.
- Women under 25 years of age and Aboriginal and Torres Strait Islander people were disproportionately represented in the notifications of chlamydial infection.

Genital chlamydial infection is caused by the bacterium *Chlamydia trachomatis* serogroups D–K. Screening is important in detecting chlamydial infections, as a large proportion of infections are asymptomatic. Chlamydial infection is highly treatable, although reinfection is common.[43] If left untreated, complications such as epididymitis in males and infertility and pelvic inflammatory disease in females can arise.[21]

Epidemiological situation in 2013

Chlamydial infection was the most frequently notified disease to the NNDSS (37% of all notifications in 2013), with 82,526 cases (357 per 100,000) notified in 2013. Between 2008 and 2010, notification rates for chlamydial infection increased by 24% (from 272 to 337 per 100,000) but remained relatively stable between 2010 and 2013 (from 337 to 357 per 100,000) (Figure 18).

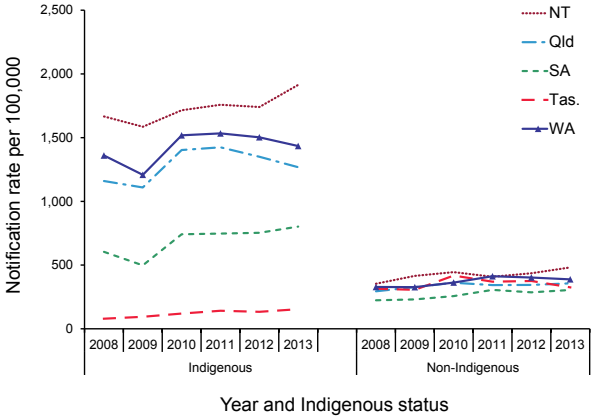
Figure 18: Notified cases and notification rate for chlamydial infection, Australia, 2008 to 2013, by year



Geographical distribution

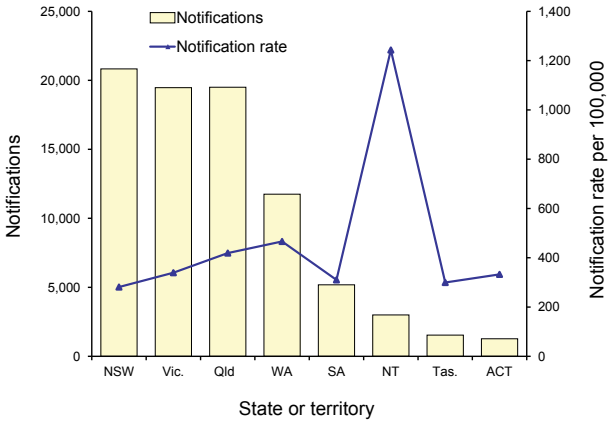
In 2013, the notification rate for chlamydial infection was almost 3.5 times higher in the Northern Territory (1,243 per 100,000) than nationally (357 per 100,000). This variation is mostly explained by the relatively large number of Aboriginal and Torres Strait Islander people in the Northern Territory, who have higher notification rates for chlamydial infection than the general population (Figure 19). In the remaining jurisdictions notification rates ranged between 281 per 100,000 in New South Wales and 466 per 100,000 in Western Australia (Figure 20).

Figure 19: Age standardised notification rate for chlamydial infection, selected states and territories,* 2008 to 2013, by year and Indigenous status



* Includes the states and territories where Indigenous status was reported for more than 50% of cases between 2008 and 2013: excludes New South Wales.

Figure 20: Notified cases and notification rate for chlamydial infection, Australia, 2013, by state or territory



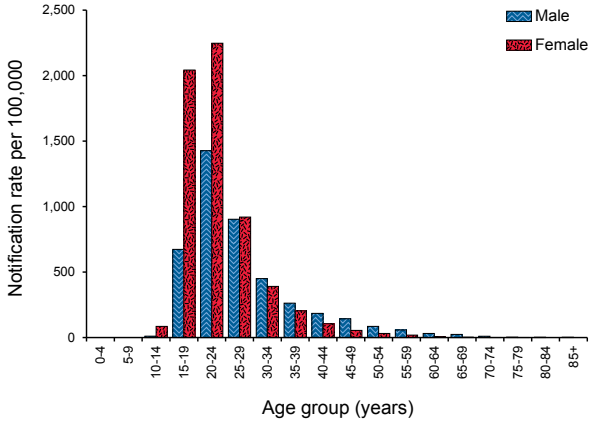
Age and sex distribution

Nationally in 2013, the notification rate for chlamydial infection was 302 per 100,000 in males, and 410 per 100,000 in females. The notification rates for males and females remained relatively stable over the past year, from 307 and 419 per 100,000 respectively in 2012. From 2008 to 2013, notification rates increased 37% for males and 28% for females. In 2013, chlamydial infection occurred predominately among those in the 15–29 years age range, accounting for 79% of notified cases.

In 2013, notification rates in females exceeded those in males for those under the age of 30 years, especially for those in the 15–19 years age group (F:M, 3.03:1) and in the 20–24 years age group

(F:M, 1.58:1). However, in the 30+ years age groups males had higher rates than females (Figure 21). The overall higher rate among females may be partly attributable to preferential testing of women attending health services compared with men.[9,44]

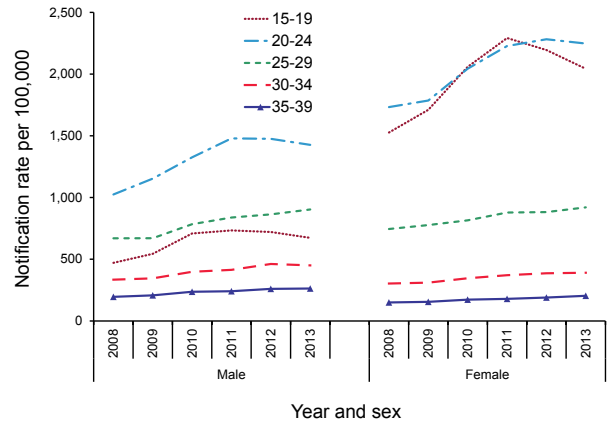
Figure 21: Notification rate for chlamydial infection, Australia, 2013, by age group and sex*



* Excludes notifications where age and/or sex were not reported and notifications where the case was aged less than 13 years.

When considering notification rates over time in the high risk age groups (15–39 years), they have increased overall since 2008, with slight declines from 2011 to 2013 for females (from 1,771 to 1,707 per 100,000) and from 2012 to 2013 for males (from 1,028 to 1,011 per 100,000) (Figure 22).

Figure 22: Notification rate for chlamydial infection in persons aged 15 to 39 years, Australia, 2008 to 2013, by year and sex* and age group



* Excludes notifications for whom age and/or sex were not reported and notifications where the case was aged less than 13 years.

Indigenous population

The completeness of Indigenous status identification in the notification data varies by year and by jurisdiction. Nationally in 2013, data on Indigenous status were complete for 38% of notifications, which is markedly lower than the preceding 5-year average of 50% (range: 49%–51%). Five jurisdictions had greater than 50% completeness of the Indigenous status field across the 2008 to 2013 period: the Northern Territory, Queensland, South Australia, Tasmania, and Western Australia. Among these jurisdictions, the combined age-standardised notification rate ratio between Indigenous and non-Indigenous populations in 2013 was 3.0:1. Overall, the ratio has varied little over the previous 5 years (range: 2.8–3.2).

Among the Indigenous population, the age-standardised notification rate declined from 2008 to 2009 (from 1,195 to 1,116 per 100,000), increased from 2010 and 2011 (from 1,360 to 1,383 per 100,000), which was followed by another decline from 1,339 per 100,000 in 2012 to 1,327 per 100,000 in 2013. Overall, the age-standardised rates in 2013 were 11% higher than in 2008 (1,195 per 100,000).

Age-standardised notification rates among the non-Indigenous population have decreased by 23% from 2008 (294 per 100,000) to 2013 (226 per 100,000). Between 2012 and 2013, age-standardised notification rates for chlamydial infection in the Indigenous population decreased in Queensland by 6% (from 1,350 to 1,268 per 100,000) and in Western Australia by 5% (from 1,503 to 1,435 per 100,000). Conversely, rates increased in the Northern Territory by 10% (from 1,740 to 1,915 per 100,000), in South Australia by 6% (from 753 to 802 per 100,000) and in Tasmania by 15% (from 134 to 154 per 100,000).

Between 2012 and 2013, the age-standardised notification rates for chlamydial infection in the non-Indigenous population increased by 10% in the Northern Territory (from 436 to 482 per 100,000), by 4% in Queensland (from 344 to 357 per 100,000), and by 7% in South Australia (from 286 to 305 per 100,000). In the same period, age-standardised notification rates decreased 13% in Tasmania (from 375 to 325 per 100,000) and by 4% in Western Australia (from 403 to 388 per 100,000) (Figure 19).

Donovanosis

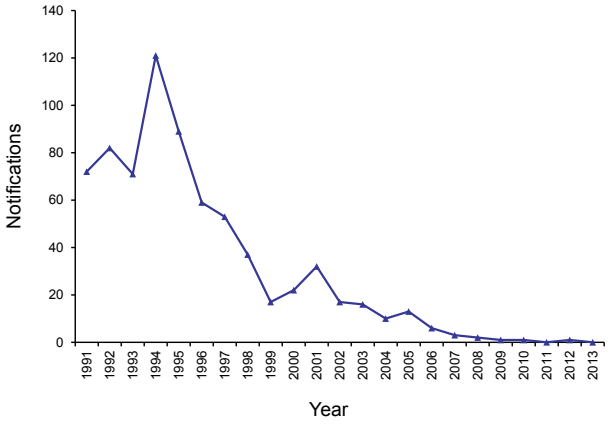
- No cases of donovanosis were notified in 2013.
- This disease is now rare in Australia.

Donovanosis, caused by the bacterium *Klebsiella granulomatis*, is a chronic, progressively destructive infection that affects the skin and mucous membranes of the external genitalia, inguinal and anal regions.[45] Donovanosis was targeted for elimination in Australia through the National Donovanosis Elimination Project 2001–2004.[46] The disease predominantly occurred in Aboriginal and Torres Strait Islander females in rural and remote communities in central and northern Australia. It is now rare, with fewer than 17 cases notified each year since 2002, and fewer than 6 cases notified each year since 2006.

Epidemiological situation in 2013

In 2013, no cases of donovanosis were notified in Australia (Figure 23).

Figure 23: Notified cases of donovanosis, Australia, 1991 to 2013, by year



Gonococcal infection

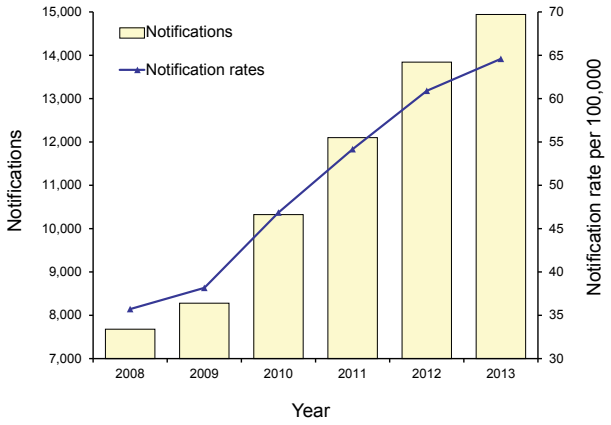
- 14,942 cases of gonococcal infection were notified in 2013.
- Notification rates for gonococcal infection continue to increase.
- Notifications in 2013 occurred predominately in males aged 20–39 years.

Gonorrhoea is caused by the bacterium *Neisseria gonorrhoeae,* which affects the mucous membranes causing symptomatic and asymptomatic genital and extra-genital tract infections.[21] If left untreated, it can lead to pelvic inflammatory disease in women and infertility in both men and women. Gonococcal infection also increases the risk of both acquisition and transmission of HIV.[45]

Epidemiological situation in 2013

In 2013, there were 14,942 cases of gonococcal infection reported to the NNDSS, a notification rate of 65 per 100,000. This was a 6% increase compared with the rate reported in 2012 (61 per 100,000). Overall, gonococcal infection notification rates increased by 81% from 2008 (36 per 100,000) to 2013 (65 per 100,000), at an average of 13% each year (range: 6%–23%) (Figure 24).

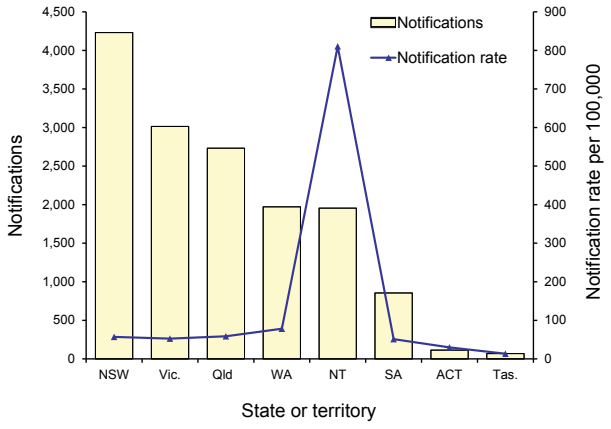
Figure 24: Notified cases and notification rate for gonococcal infection, Australia, 2008 to 2013, by year



Geographical distribution

In 2013, the notification rate for gonococcal infection was 12.5 times higher in the Northern Territory (811 per 100,000) than nationally (65 per 100,000) (Figure 25). This variation is partly explained by the relatively large number of Aboriginal and Torres Strait Islander people in the

Figure 25: Notified cases and notification rate for gonococcal infection, Australia, 2013, by state or territory



Northern Territory, who have higher notification rates for gonococcal infection than the general population (Figure 26).

Age and sex distribution

Nationally in 2013, the notification rate for gonococcal infection was 91 per 100,000 in males and 38 per 100,000 in females, which represented a slight increase from 2012 (84 per 100,000 in males and 36 per 100,000 in females). In 2013, gonococcal infection occurred predominately among those aged 15–34 years, who accounted for 72% of notified cases.

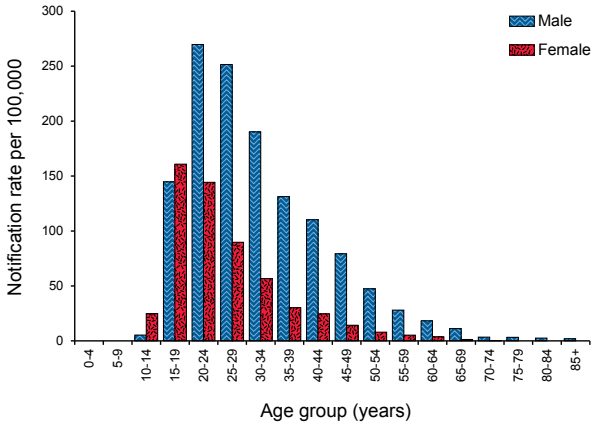
Overall, the male to female ratio was 2.4:1 in 2013, which has not changed from 2012. In 2013, notification rates in females exceeded those in males in the under 20 years age groups, but was the reverse for all age groups above 20 years (Figure 27).

When considering trends over time in those aged 15–49 years, notification rates increased from 2008 to 2013 in all age groups across both sexes, with the exception of females in the 15–19 years age group, where rates declined between 2011 and 2013 (Figure 28).

Indigenous population

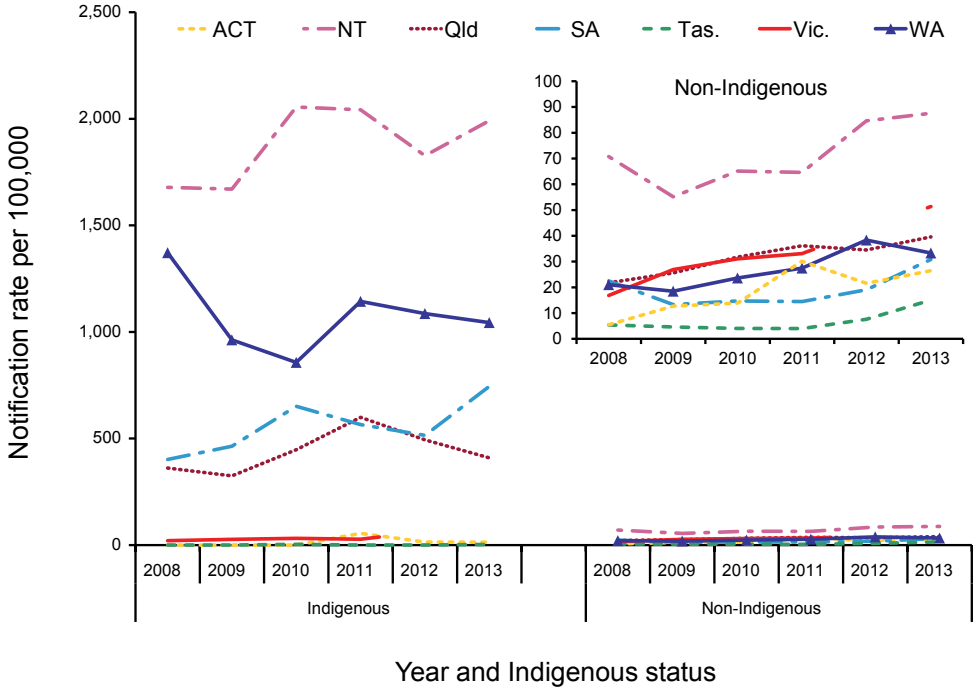
The completeness of Indigenous status identification in the notification data varies by year and by jurisdiction. Nationally in 2013, data on Indigenous status were complete for 72% of notifications, which was higher than the preceding 5–year average of 68% (range: 66%–73%). All states and territories except New South Wales had greater than

Figure 27: Notification rate for gonococcal infection, Australia, 2013, by age group and sex*



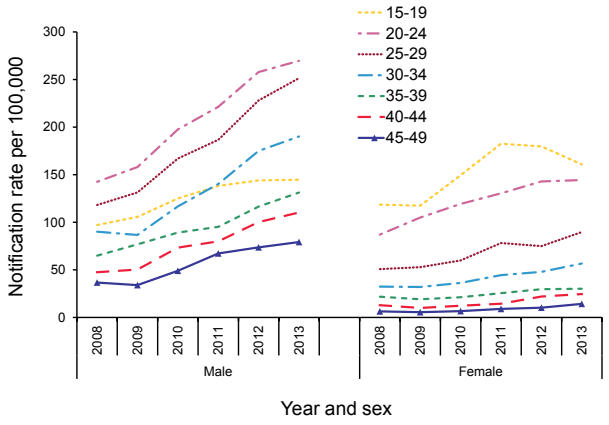
* Excludes notifications where age and/or sex were not reported and notifications where the case was aged less than 13 years and the infection was able to be determined as non-sexually acquired.

Figure 26: Age-standardised notification rate for gonococcal infection, selected states and territories,* 2008 to 2013, by year and Indigenous status



* Includes the states and territories where Indigenous status was reported for more than 50% of cases between 2008 and 2013: excludes New South Wales.

Figure 28: Notification rate for gonococcal infection in persons aged 15 to 49 years, Australia, 2008 to 2013, by year and sex and age group*



* Excludes notifications where age and/or sex were not reported.

50% completeness of the Indigenous status field across the 2008 to 2013 period. Among these states and territories, the combined age-standardised notification rate ratio between Indigenous and non-Indigenous populations in 2013 was 18.5:1, declining from 21.3:1 in 2012. Overall, the rate ratio has declined by 51% from 2008 to 2013 (from 37.6:1 to 18.5:1).

Among the Indigenous population, the age-standardised notification rate increased by less than 1% from 2012 to 2013 (from 770 to 773 per 100,000). Rates in 2013 were 4% lower than in 2008 (745 per 100,000).

The age-standardised notification rate among the non-Indigenous population has more than doubled from 2008 to 2013 (20 and 42 per 100,000 respectively). The average annual increase over this period was 16% (range: 11%–19%).

In terms of geographical trends, age-standardised notification rates for gonococcal infection in the Indigenous population between 2012 and 2013 both increased and decreased among the states and territories in which Indigenous status was more than 50% complete; rates decreased in the Australian Capital Territory by 6% (from 15 to 14 per 100,000), in Queensland by 17% (from 494 to 410 per 100,000), in Victoria by 28% (63 to 45 per 100,000) and in Western Australia by 4% (from 1,086 to 1,044 per 100,000). Conversely, notification rates increased in the Northern Territory by 9% (from 1,827 to 1,991 per 100,000), and in South Australia by 45% (from 515 to 745 per 100,000). Tasmania reported no cases in 2012 but a notification rate of 2.75 in 2013 (Figure 26).

Between 2012 and 2013, the age-standardised rates for gonococcal infection in the non-Indigenous population increased by 23% in the Australian Capital Territory (from 22 to 26 per 100,000), by 4% in the Northern Territory (from 85 to 88 per 100,000), by 15% in Queensland (from 35 to 40 per 100,000), by 62% in South Australia (from 19 to 31 per 100,000), by 98% in Tasmania (from 8 to 15 per 100,000), and by 21% in Victoria (from 42 to 51 per 100,000). Conversely, notification rates decreased in Western Australia by 13% (from 38 to 33 per 100,000) (Figure 26).

Microbiological trends

The AGSP is the national surveillance system for monitoring the antimicrobial resistance of *N. gonorrhoeae* isolates. These results are published in more detail in the AGSP annual report in CDI.[47]

In 2013, the AGSP reported that a total of 4,896 gonococcal isolates were referred for antibiotic susceptibility testing, representing 33% of gonococcal infection notified to the NNDSS. This was slightly lower than the proportion of NNDSS cases tested in 2012 (35%), and a further decrease from the 40%–42% referred in 2008 to 2010.

Eighty-two per cent of the isolates (n=4,032) were from males and 18% (n=862) were from females (M:F, 4.7:1). There were 2 isolates for which gender was unknown. The proportion of gonococcal isolates from males and females tested by the AGSP has remained similar over recent years (<1% variation).

Syphilis (non-congenital categories)

- 3,474 cases of syphilis (non-congenital categories) were notified in 2013, a rate of 15.0 per 100,000.
- In 2013, the notification rate for infectious syphilis was 7.6 per 100,000 and the notification rate for syphilis of more than 2 years or unspecified duration was 7.4 per 100,000.

Syphilis, caused by the bacterium *Treponema palladium*, is characterised by a primary lesion, a secondary eruption involving skin and mucous membranes, long periods of latency and late lesions of skin, bone, viscera, cardiovascular and nervous systems.[21]

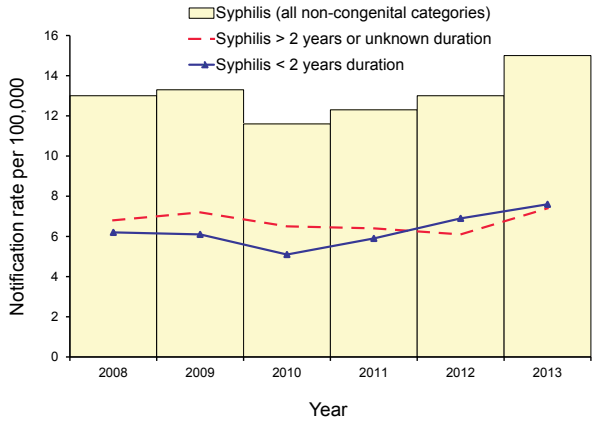
In 2004, all jurisdictions except South Australia began reporting non-congenital syphilis infections to the NNDSS, separately categorised as: infectious syphilis (primary, secondary or early latent) of less than 2 years duration; and syphilis of more than 2 years or unknown duration. From 2004 to 2011,

South Australia reported only cases of infectious syphilis, and then in 2012 commenced reporting syphilis of more than 2 years or unknown duration. Data for all states and territories are reported by diagnosis date, except Queensland, which is reported by notification receive date.

Epidemiological situation in 2013

In 2013, a total of 3,474 cases of syphilis (non-congenital) were reported. This represents a rate of 15.0 per 100,000, a 15% increase compared with 2012 (13.0 per 100,000) (Figure 29). In 2013, 49% of syphilis notifications were categorised as greater than 2 years or unknown duration, and 51% of cases were categorised as less than 2 years duration.

Figure 29: Notification rate for non-congenital syphilis infection (all categories),*,† Australia, 2008 to 2013, by year and category



* For infectious syphilis, excludes notifications where the case was aged less than 13 years and the infection was able to be determined as non-sexually acquired. For syphilis of more than 2 years or unknown duration, excludes all notifications where the case was aged less than 13 years.

† For syphilis of more than 2 years or unknown duration, excludes South Australia from 2008–2011.

Syphilis – infectious (primary, secondary and early latent), less than 2 years duration

- 1,768 cases of infectious syphilis were notified in 2013.
- In 2013, 79% of all notifications occurred in males aged 20–54 years. Notification rates in males exceeded those in females in almost all age groups.
- Cases of infectious syphilis were almost completely in men who have sex with men.

Epidemiological situation in 2013

In 2013, 1,768 notified cases of infectious syphilis (primary, secondary and early latent), less than 2 years duration, were reported to the NNDSS, representing a rate of 7.6 per 100,000. This was an 11% increase compared with the rate reported in 2012 (6.9 per 100,000). The notification rate for infectious syphilis increased overall by 23% from 2008 to 2013 (from 6.2 to 7.6 per 100,000) (Table 6).

Geographical description

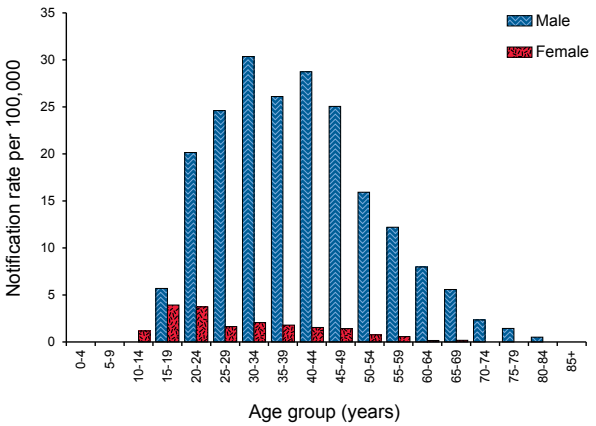
In 2013, notification rates for infectious syphilis (less than 2 years duration) were highest in Victoria, the Northern Territory and New South Wales (11.4, 9.1 and 8.1 per 100,000 respectively) (Table 5).

Age and sex distribution

Nationally in 2013, the notification rate for infectious syphilis was 14.0 per 100,000 in males and 1.3 per 100,000 in females, a male to female rate ratio of 11.0:1. In males, this was an increase of 12% when compared with the 2012 rate (12.4 per 100,000) and in females this was a decrease of 5% compared with the 2012 rate (1.3 per 100,000). In 2013, 79% of all notifications occurred in males aged 20–54 years, and notification rates for males exceeded those for females in almost all age groups (Figure 30). Diagnoses of infectious syphilis in 2013 were almost completely confined to men who have sex with men.[28]

Notification rates for males aged 15 years or over increased overall among most age groups from

Figure 30: Notification rate for infectious syphilis (primary, secondary and early latent), less than 2 years duration, Australia, 2013, by age group and sex*

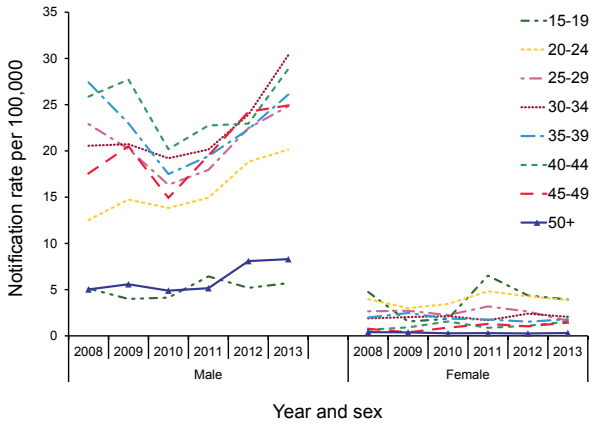


* Excludes notifications where age and/or sex were not reported and notifications where the case was aged less than 13 years and the infection was able to be determined as non-sexually acquired.

2008 to 2013. An exception was the 35–39 years age group, for which the rate declined overall from 2008 to 2013 (from 27 to 26 per 100,000) (Figure 31).

In females aged 15 years or over, notification rates between 2008 and 2013 have averaged 2.1 per 100,000 (range: 0.3–3.9). Over the 6-year period, the notification rates remained low for females across all age groups.

Figure 31: Notification rate for infectious syphilis (primary, secondary and early latent), less than 2 years duration, in persons aged 15 years or over,* Australia, 2008 to 2013, by year and sex and age group*



* Excludes notifications where age and/or sex were not reported.

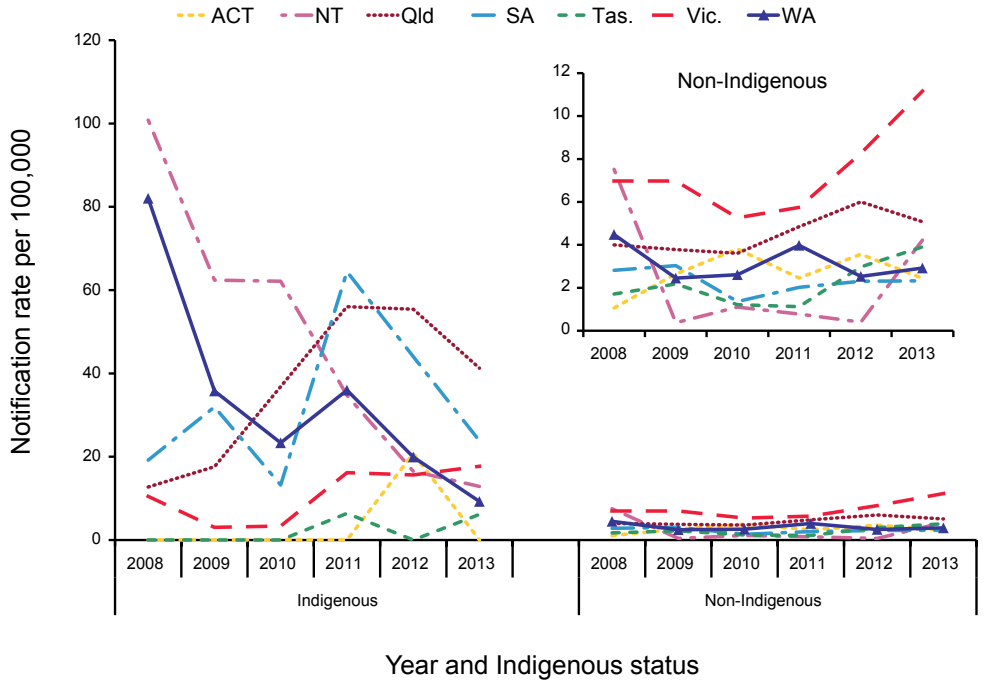
Indigenous population

The completeness of Indigenous status identification in the notification data varies by year and by jurisdiction. Nationally in 2013, data on Indigenous status were complete for 72% of notifications, an increase compared with 2012 (66% complete) and higher than the preceding 5–year average of 68% (range: 66%–73%). All states and territories except New South Wales had greater than 50% completeness of the Indigenous status field across the 2008 to 2013 period.

Among the states and territories with greater than 50% completeness for Indigenous status, the combined age standardised notification rate ratio between the Indigenous and non-Indigenous populations in 2013 was 3.7:1, which was lower than the preceding 5–year average of 7.6:1 (range: 5.7–9.3).

The age-standardised notification rate in the Indigenous population declined from 34 per 100,000 in 2012 to 25 per 100,000 in 2013. Overall, 2013 rates are 41% lower than 2008 rates (41 per 100,000). This declining trend is not seen in all jurisdictions (Figure 32) but it is likely that programs that include population screening and

Figure 32: Age-standardised notification rate for infectious syphilis (primary, secondary and early latent), less than 2 years duration, selected states and territories,* 2008 to 2013, by Indigenous status and year



* Includes the states and territories where Indigenous status was reported for more than 50% of cases between 2008 and 2013: excludes New South Wales.

case management, supported by centrally based state-wide syphilis registers, are making some progress towards elimination of infectious syphilis in Indigenous communities.[48]

The age-standardised notification rate in the non-Indigenous population has increased from 6.0 per 100,000 in 2012 to 6.6 per 100,000 in 2013. The rate in 2013 was 32% higher than it was in 2008 (5.0 per 100,000).

In terms of geographical trends, from 2012 to 2013, the age-standardised rates for syphilis infection in the Indigenous population declined in all states and territories except Victoria and Tasmania (Figure 32). Between 2008 and 2013, the Northern Territory was the only jurisdiction to report declining Indigenous age-standardised notification rates every year. The increase evident in Indigenous notification rates in Western Australia in 2008 was largely attributable to an outbreak that occurred in the Pilbara region among Aboriginal people during that year.[49]

Among the non-Indigenous population between 2012 and 2013, the age-standardised rates for syphilis infections increased in all jurisdictions except the Australian Capital Territory and Queensland (Figure 32).

Syphilis of more than 2 years or unknown duration

- 1,706 cases of syphilis of more than 2 years or unknown duration were notified in 2013.
- Overall, notification rates have increased from 6.3 per 100,000 in 2008 to 7.4 per 100,000 in 2013.
- The notification rate among males (10.8 per 100,000) was more than double that for females (3.9 per 100,000) in 2013.

Epidemiological situation in 2013

In 2013, 1,706 cases of syphilis of more than 2 years or unknown duration were reported to the NNDSS. This represents a notification rate of 7.4 per 100,000. Overall, notification rates have increased by 17% from 2008 to 2013 (6.8 to 7.4 per 100,000) (Table 6).

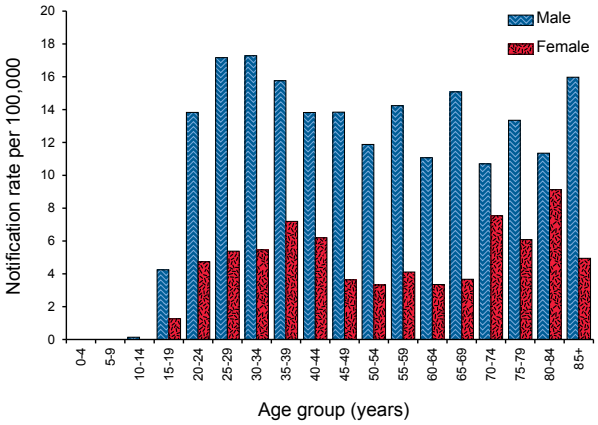
Geographical distribution

In 2013, notification rates for syphilis of more than 2 years or unknown duration were highest in the Northern Territory (39.0 per 100,000), followed by Victoria (9.8 per 100,000) (Table 5).

Age and sex distribution

Nationally in 2013, the notification rate for syphilis of more than 2 years or unknown duration was 10.8 per 100,000 for males and 3.9 per 100,000 in females, a male to female ratio of 2.8:1. In males, this was an increase of 30% when compared with the 2012 rate (8.3 per 100,000), and in females a 4% increase from the 2012 rate (3.8 per 100,000). Around 71% of all notifications occurred in males aged 20 years or over, and notification rates in males exceeded those in females in all age groups (Figure 33).

Figure 33: Notification rate for syphilis of more than 2 years or unknown duration,* Australia, 2013, by age group and sex



* Excludes notifications for whom age and or sex were not reported and notifications where the case was aged less than 13 years and the infection was able to be determined as non-sexually acquired.

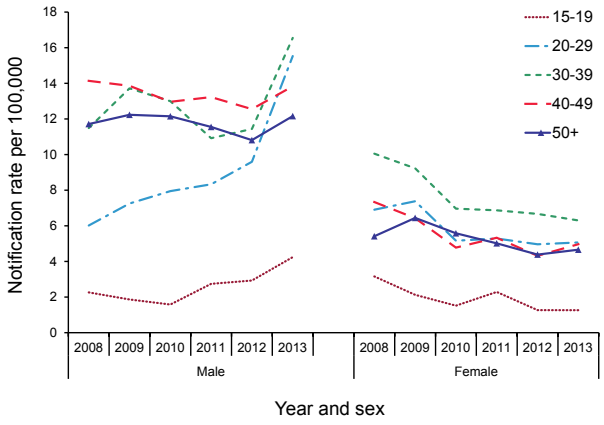
Notification rates for those aged 15 years or over from 2008 to 2013 increased overall in most age groups for males, and declined overall across all age groups for females (Figure 34).

Congenital syphilis

- Seven cases of congenital syphilis were notified in 2013.
- Congenital syphilis remains rare in Australia.

Congenital syphilis is caused by foetal infection with the bacteria *T. pallidum*. Syphilis is acquired by infants either in-utero or at birth from women with untreated early infection. Infections commonly result in abortion or stillbirth and may cause the death of a newborn infant. Congenital syphilis can be asymptomatic, especially in the first weeks of life.[21]

Figure 34: Notification rate for syphilis of more than 2 years or unknown duration, in persons aged 15 years or over,* Australia,† 2008 to 2013, by year and sex and age group



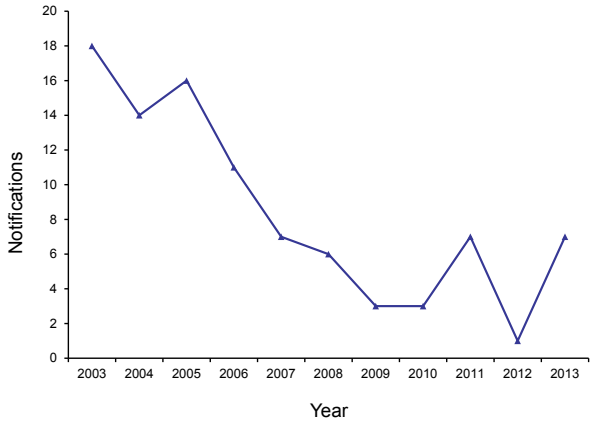
* Excludes notifications where age and/or sex were not reported.

† Data from all states and territories except South Australia in 2008–2011.

Epidemiological situation in 2013

There were 7 notifications of congenital syphilis in 2013, which remains low after a decrease observed over the 10 years prior (Figure 35). Antenatal screening for syphilis with follow-up and adequate treatment is considered to be a contributor to this decline.[48]

Figure 35: Notified cases of congenital syphilis, Australia, 2003 to 2013, by year



Vaccine preventable diseases

Overview

This section summarises the national surveillance data for notifiable diseases targeted by the National Immunisation Program (NIP) in 2013. These include diphtheria, invasive *Haemophilus influenzae* type b (Hib) infection, laboratory confirmed influenza, measles, mumps, pertussis, invasive pneumococcal disease (IPD), poliomyelitis, rubella, tetanus and varicella zoster infections (unspecified, chickenpox and shingles). Data on hepatitis B and invasive meningococcal disease, which are also targeted by the NIP, can be found in this report under 'Bloodborne diseases' and 'Other bacterial infections' respectively. Other vaccine preventable diseases (VPDs) presented in this report include hepatitis A and Q fever reported under the 'Gastrointestinal' and 'Zoonoses' sections respectively. More detailed reports on historical data, including notifications, hospitalisations and deaths are published in the CDI journal supplements 'Vaccine Preventable Diseases in Australia'[26] and additional analysis on individual diseases are published in CDI in the 'Australian Vaccine Preventable Diseases Epidemiological Review Series'.

In 2013, there were 59,630 VPD notifications reported to the NNDSS, representing 27% of all reported cases and a 31% decrease compared with 2012 (85,848 cases). Influenza was the most commonly notified VPD with 28,329 cases (48%) reported, followed by pertussis (12,341 cases, 21%). The number of notifications and notification rates for VPDs in Australia are shown in Table 3, Table 4 and Table 5.

Vaccination coverage is an important factor influencing the incidence of VPDs. Since the commencement of the Australian Childhood Immunisation Register in 1996, immunisation coverage in children has been high by international standards, although geographical pockets of lower coverage, in which there is an increased potential for VPD cases, remain. As no vaccine is 100% effective, infections with these diseases sometimes do occur in fully vaccinated people, and some are reported later in this section. However, vaccines do provide a substantially lower chance of becoming infected or will reduce the severity of disease.

Information on a case's vaccination history was previously recorded in the NNDSS using the 'vaccination status' field (fully or partially vaccinated for age or not vaccinated), plus fields capturing the number of doses, the last vaccination date and how the vaccination information was validated. In January 2008 new, more detailed fields were incorporated for recording 'vaccine type', and 'vaccination date' for each dose of vaccine given. The new fields were intended to replace the old fields, with a transition period allowing either field to be utilised. In 2013, all jurisdictions were using the new fields except for the Australian Capital Territory. In this report the vaccination status of a case is

interpreted according to the data provided by the states and territories from the 2 different formats. A case is described as fully vaccinated if they have received all doses of the relevant vaccine according to the most recent edition of *The Australian Immunisation Handbook*[50] and at least 14 days prior to disease onset. In contrast, fully vaccinated for age describes a case that has received all recommended doses of a vaccine for their age but may not yet have received the full course of vaccinations required to be considered fully vaccinated.

In 2013, the measles-mumps-rubella-varicella (MMRV) vaccine for all children at 18 months of age and the combined Hib and monovalent meningococcal C conjugate vaccine (Hib-MenCCV) for all children at 12 months of age were added to the NIP. The MMRV replaced the 2nd dose of MMR previously provided at 4 years of age and combined it with the varicella vaccine already given at 18 months of age. This change is expected to provide earlier protection and improve coverage of the second dose of MMR while being consistent with the World Health Organization (WHO) recommendation that a 2nd dose of measles containing vaccine should be given in the 2nd year of life.[51] Hib-MenCCV replaces the single dose of MenCCV and booster dose of monovalent Hib vaccine previously scheduled at 12 months of age.[50]

Diphtheria

- There were 2 cases of diphtheria notified in Australia in 2013.
- Diphtheria is now rare in Australia.

Diphtheria is an acute pharyngeal or cutaneous infection caused mainly by toxigenic strains of *Corynebacterium diphtheriae*. The exotoxin acts locally on the mucous membranes of the respiratory tract, and on damaged skin, although this is not as common. Disease is mainly due to local membranous inflammation, which for pharyngeal diphtheria can cause airway obstruction. Occasionally, systemic infections occur and cause damage to the myocardium, nervous system and kidneys. Diphtheria is spread by respiratory droplets or direct contact with nasopharyngeal secretions or skin lesions. While there are non-toxigenic strains of *C. diphtheriae,* they usually only cause mild throat or skin infection and are not nationally notifiable.[21]

Epidemiological situation in 2013

In 2013, there were 2 notifications of diphtheria reported to the NNDSS, one from Queensland and one from South Australia. Both cases were imported infections from Papua New Guinea and India respectively.

Diphtheria is rare in Australia, with most cases associated with sporadic importations from countries in which this disease remains endemic. Since the 1 case of cutaneous diphtheria reported in 2001, the only other year before 2013 in which cases were reported was 2011, when a cluster of 3 infections, including 1 death, and an unrelated case of cutaneous diphtheria were notified.

Influenza

- In 2013, notifications of laboratory confirmed influenza decreased by almost 37% from 2012 making it a mild to moderate season since the 2009 pandemic season.
- Children aged 9 years or under, and also middle aged adults, as well as those with underlying medical conditions were most affected.

Influenza is a common, highly infectious acute respiratory disease caused by infection with influenza viruses. The virus is transmitted from person to person by airborne droplets of exhaled respiratory secretions, especially by coughing or sneezing.[52] The disease caused by infection with influenza viruses ranges from asymptomatic[53] through to mild upper respiratory tract illness, to severe complications including pneumonia. The severity of disease is determined by features intrinsic to the virus including its similarity to previous circulating and vaccine strains and by host factors including the presence of chronic conditions, pregnancy and smoking in the population.[54] The goals of influenza surveillance are to determine the magnitude and distribution of illness, detect outbreaks, monitor for changes in the virus and to facilitate policy development and planning.[55]

Annual influenza vaccination is the primary means of preventing or attenuating influenza and its complications and is included in the NIP for individuals who are at increased risk of complications from influenza infection. In 2013, the NIP funded influenza vaccine for people aged 6 months or over with medical conditions placing them at risk of serious complications due to influenza, Aboriginal and Torres Strait Islander people aged 15 years or over, pregnant women and people aged 65 years or over.

Epidemiological situation in 2013

In 2013, there were 28,329 cases of laboratory confirmed influenza, which was almost two-thirds the number of notified cases reported in 2012, but similar to the number of cases notified in 2011.

Geographic distribution

Notification rates were highest in South Australia (289 per 100,000) and the Northern Territory (199 per 100,000). Notifications in the Australian Capital Territory, New South Wales, Queensland, Victoria and Western Australia were somewhat similar to the national notification rate of 123 per 100,000, while the Tasmanian rate was substantially lower than the national rate at 58 per 100,000. New South Wales reported the highest number of influenza cases of any jurisdiction, comprising 30% of all notifications, which differed from previous seasons, when Queensland reported the highest number (Figure 36).

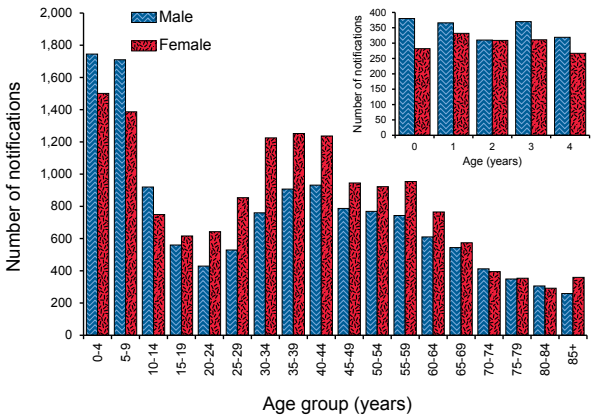
Age and sex distribution

The highest number of influenza notifications occurred in the 0–4 years and 5–9 years age groups, which together accounted for 22% of all notifications (Figure 37).

Likewise, notification rates were highest in the 0–4 years and 5–9 years age groups (214 and 213 notifications per 100,000 respectively) (Figure 38). There were also higher notification rates seen in middle aged adults (35–39 years and 40–44 years age groups).[56]

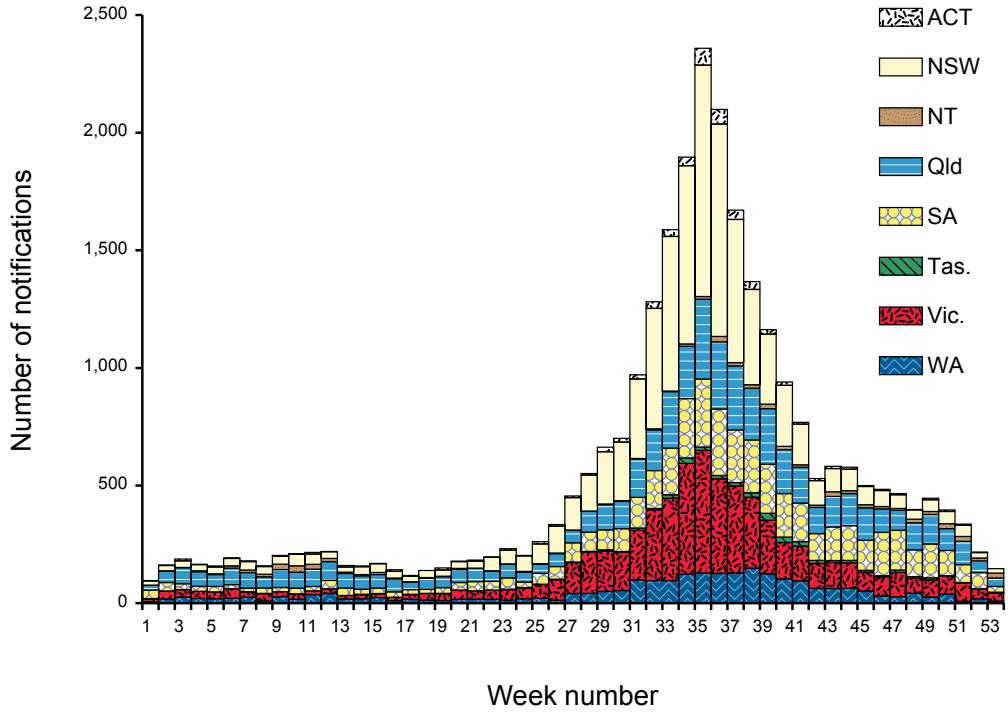
In seasons dominated by the influenza A(H1N1) pdm09 virus, such as 2009, 2010 and 2011, the age distribution of influenza notifications showed a downward trend with increasing age. For comparison, in 2012, which was dominated by influenza A(H3N2), the age distribution of influenza notifications was bimodal with peaks in those aged

Figure 37: Notified cases of laboratory confirmed influenza, Australia, 2013, by age group and sex*



* Excludes 35 notifications for which age or sex was not reported.

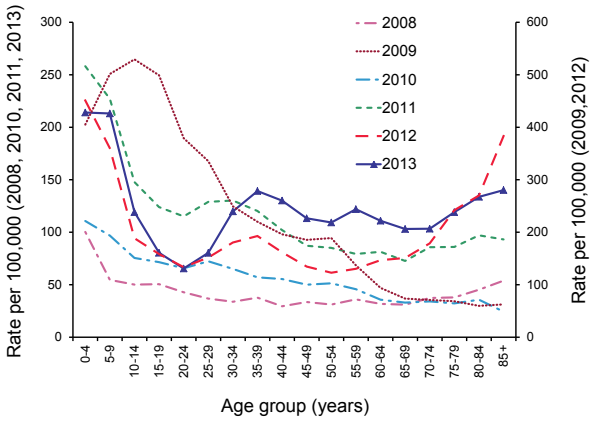
Figure 36: Notified cases of laboratory confirmed influenza, Australia, 2013, by week and state or territory



under 10 years and in those aged 70 years or over, and a small peak among those aged 30–44 years. The 2013 influenza season has been characterised by co-circulation of A(H1N1)pdm09, influenza A(H3N2) and influenza B viruses.

In 2013, females accounted for 15,033 (53%) of the influenza notifications for which sex was reported. Notification rates per 100,000 were generally higher among females in the adult age groups, whereas males dominated the younger age groups (0–14 years).

Figure 38: Notification rate for laboratory confirmed influenza, Australia, 2008 to 2013, by age group and year



Seasonality

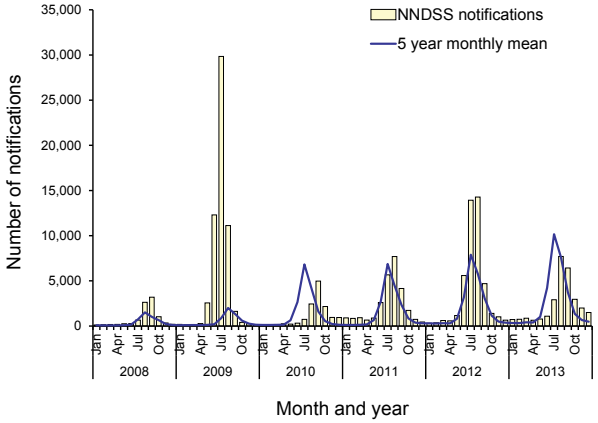
Influenza notification activity during the 2012–13 inter-seasonal period was the 2nd highest on record behind that observed during the 2010–11 inter-seasonal period. Excluding 2010, notifications of influenza in 2013 started their seasonal increase later, and rose and peaked moderately in comparison with previous years (Figure 39).

The majority of jurisdictions peaked in activity around late August, followed by a decline in influenza activity back to inter-seasonal levels. However, influenza activity remained particularly elevated in South Australia, Queensland and the Northern Territory during the latter part of 2013

Indigenous status

Of those states where Indigenous status completeness was greater than 50% (Western Australia, South Australia and the Northern Territory), the age standardised notification rate for influenza was 265 per 100,000 in the Indigenous population and 163 per 100,000 for the non-Indigenous population, representing a rate ratio of 1.6:1.

Figure 39: Notified cases of laboratory confirmed influenza, Australia,* 2008 to 2013, by month and year



* In South Australia, influenza was not made notifiable through legislation until May 2008.

Mortality

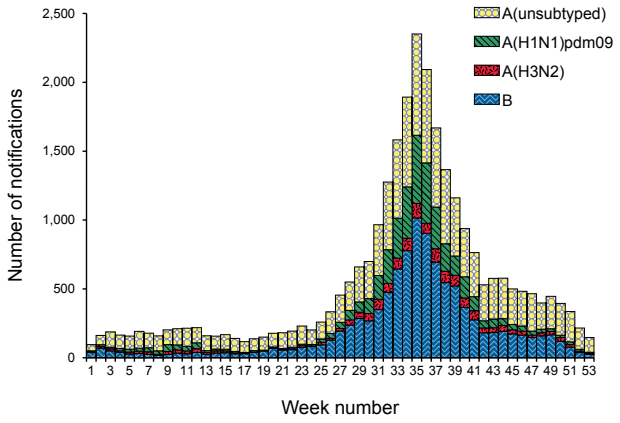
Nationally, there were 48 influenza-associated deaths notified to the NNDSS, with a median age of 67 years (range 27–97 years). The majority of deaths were associated with influenza A infections (n=39; 81%), and of these, 16 were associated with A(unsubtyped) infections, 16 were A(H3N2) and seven were A(H1N1)pdm09. Indigenous status was reported for 75% (n=36) of the influenza-associated deaths; and Indigenous peoples accounted for 8% (n=3) of these deaths. The number of influenza-associated deaths reported to the NNDSS is reliant on the follow-up of cases to determine the outcome of their infection and most likely underestimates the true mortality impact associated with this disease.

Microbiological trends

In 2013, typing data was reported for all but 11 laboratory confirmed influenza notifications. Of notifications with typing information, 63% were type A (43% A(unsubtyped), 14% (H1N1) pdm09 and 6% (H3N2) and 37% type B. Mixed influenza type A and B infections accounted for <1% of notifications. None were reported as influenza type C (Figure 40).

The overall type breakdown was similar in 2011 and 2012. Whilst the majority of influenza A reports are unsubtyped, 14% of overall notifications were reported as influenza A(H1N1)pdm09, compared with less than 1% in 2012. Further, the proportion of influenza B notifications reported in 2013 has been higher than in previous years.

Figure 40: Notified cases of laboratory confirmed influenza,* Australia, 2013, by week and subtype



* Excludes 77 mixed type A and B, and untyped influenza infections.

For 2013, the WHO Collaborating Centre for Reference and Research on Influenza (WHOCC) analysed 1,570 specimens from Australian influenza cases. This represented approximately 6% of the 28,329 laboratory confirmed cases reported to the NNDSS. Influenza A(H1N1)pdm09 comprised 45% (n=699) of influenza viruses followed by influenza B (39%; n=613) and influenza A(H3N2) (16%; n=258) (Figure 41).

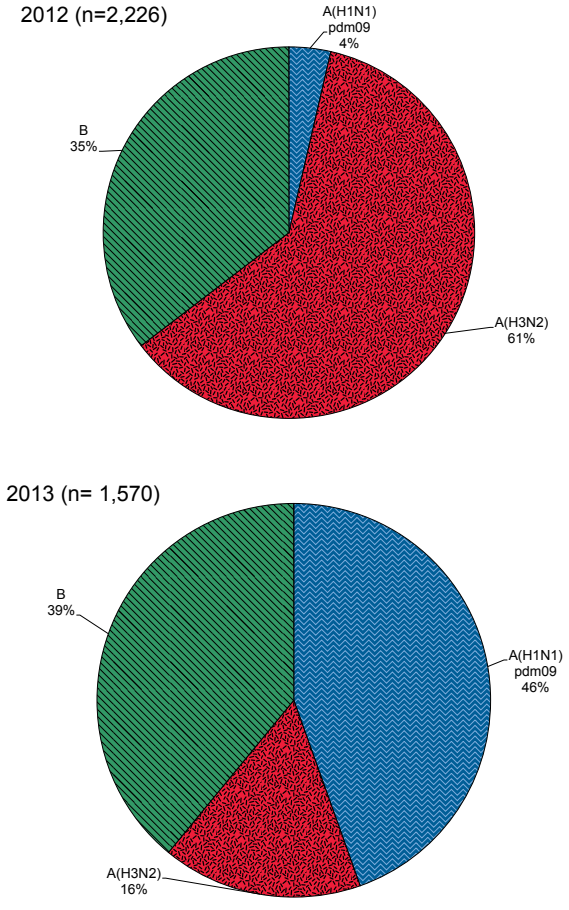
The WHOCC assessed the antigenic similarity of circulating influenza virus isolates to reference strains by haemagglutination inhibition (n=1,532 influenza virus isolates). All of the A(H1N1) pdm09 isolates (n=688) were antigenically similar to the A/California/7/2009 vaccine virus, with 5% (n=35) characterised as 'low reactors'. The haemagglutinin of these low reactor viruses mostly contained amino acid substitutions acquired *in vitro* as a result of adaptation to growth in Madin-Darby canine kidney cells as these changes were not present in the original clinical samples. All of the A(H3N2) isolates (n=234) were antigenically similar to the A/Victoria/361/2011 vaccine virus.

Of the influenza B viruses (n=610), 96% were from the B/Yamagata lineage, with the remainder from the B/Victoria lineage. Of the B/Yamagata lineage viruses (n=583), almost all were B/Massachusetts/2/2012-like viruses (n=580), including a few 'low reactors' (n=7), with the remaining B/Yamagata lineage viruses (n=3) characterised as low reactor B/Wisconsin/1/2010-like viruses. The small number of B/Victoria lineage viruses (n=27) were all antigenically similar to the former vaccine strain B/Brisbane/60/2008.

Of the viruses included in the 2013 trivalent Australian influenza vaccine, the influenza A(H1N1)pmd09 and A(H3N2) viruses that were isolated during 2013 were antigenically similar to the 2013 vaccine viruses. However, the B/Yamagata lineage virus isolates were mostly antigenically similar to the B/Massachusetts/2/2012 virus, with very few detections of the B/Wisconsin/1/2010 vaccine strain.

Viruses collected in 2013 were also tested for sensitivity to the neuraminidase inhibitor class of antiviral drugs. Neuraminidase inhibition assays were performed on 1,458 virus isolates consisting of 665 A(H1N1)pdm09, 575 B and 218 A(H3N2) viruses. Reduced inhibition by oseltamivir was detected in 4 A(H1N1)pdm09 isolates and was mediated by the well characterised H275Y mutation, which is known to confer resistance to oseltamivir.[57] Reduced inhibition to zanamivir was detected in a single B/Yamagata lineage isolate.

Figure 41: World Health Organization Collaborating Centre for Reference and Research on Influenza subtyped influenza virus samples, Australia, 2012 and 2013



Enhanced surveillance data sets

In addition to NNDSS data, a series of targeted influenza surveillance systems operated during 2013. Together, these systems collected data that were used to describe the season under the areas of epidemiology, morbidity, mortality and virology and supported the conclusions drawn from analyses of NNDSS notification data. Enhanced influenza surveillance was based on the following additional sources of data:

- the number and proportion of calls to a national health call centre network for influenza or influenza-like illness (ILI);
- rates of ILI from a community survey;
- consultation rates for ILI identified by sentinel general practitioners;
- consultation rates for ILI identified by hospital emergency departments in Western Australia, New South Wales and the Northern Territory;
- hospitalised cases of influenza from 15 sentinel hospitals across Australia;
- mortality data from the New South Wales Registry of Births, Deaths and Marriages; and
- typing and subtyping for influenza from sentinel laboratories in New South Wales, Victoria, Western Australia and Tasmania.

These data sources were used to inform the overall picture of influenza activity in Australia and comprehensive analysis of these data are provided in the fortnightly Australian Influenza Surveillance Report, which was published during the influenza season, and in the annual National Influenza Surveillance Scheme report.

Discussion

The 2013 influenza season in Australia began in early July, peaked in late August and was largely concluded by mid-December. Australia experienced sustained virus circulation until mid-October, which continued until mid-December for South Australia, Queensland and the Northern Territory in particular. Peak NNDSS notifications occurred approximately 1 month later than the median week of peak transmission for 2012.[58] The most commonly detected virus was influenza A(H1N1)pdm09, however influenza type B and A(H3N2) were also prominent. The age distribution of influenza notifications in the 2013 season was mixed, with infants and young children most affected, followed by middle-aged adults.

Taken together, data from most influenza surveillance systems showed that the overall impact of influenza in 2013 was indicative of a relatively mild to moderate season with some sustained activity experienced in the latter part of the year. The average number of influenza notifications reported per week during the season were around half that in 2012, and both ILI and influenza activity across systems were overall lower than in 2012.[59]

Invasive *Haemophilus influenzae* type b

- There were 20 cases of invasive Hib reported in 2013.
- Of the cases reported 60% were female and 55% were under the age of 5 years.
- The 2013 notification rate of Hib remains low at 0.1 per 100,000 population.

Invasive Hib is a bacterium that causes disease with symptoms dependant on which part of the body is infected. These include: septicaemia (infection of the blood stream); meningitis (infection of the membranes around the brain and spinal cord); epiglottitis (severe swelling of the epiglottis at the back of the throat); pneumonia (infection of the lungs); osteomyelitis (infection of the bones and joints) and cellulitis (infection of the tissue under the skin, usually on the face).

Since the introduction of the Hib vaccine on to the NIP in 1993, there has been a reduction of more than 95% in notified cases of Hib disease in Australia, which now has one of the lowest rates in the world.[26]

Epidemiological situation in 2013

In 2013, there were 20 notifications of Hib disease reported. This was a slight increase compared with cases reported in 2012 (n=15), and representing a ratio of 1.0 compared with the mean of the previous 5 years. The 2013 notification rate was 0.1 per 100,000 and was consistent with the very low rates seen since the introduction of the vaccine on the NIP in July 1993 (Figure 42).

Cases occurred in the 3 most populous states of New South Wales (n=9), Queensland (n=7) and Victoria (n=4). The notification rates were consistent between states ranging from 0.1 per 100,000 in Victoria to 0.2 per 100,000 in Queensland. There were no infants deaths reported in 2013. One Hib associated death was reported in a 65-year-old non-Indigenous female of unknown vaccination status.

Age and sex distribution

In 2013, the male to female ratio was 0.7:1, 8 male and 12 female cases. More than half of the cases (n=11) were in children aged less than 5 years

and 73% (n=8) of these were among infants less than 1 year. Consistent with previous years, the 0–4 years age group had the highest notification rate (0.7 per 100,000). There were no cases reported among young adults between 20 and 39 years of age while adults 40 years of age or over accounted for 35% (n=8) of cases (Figure 43).

Figure 42: Notified cases and notification rate for invasive *Haemophilus influenzae* type b infection, Australia, 1993 to 2013, by year of diagnosis

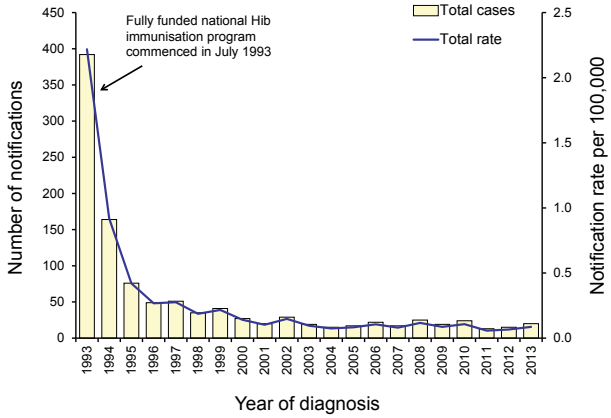
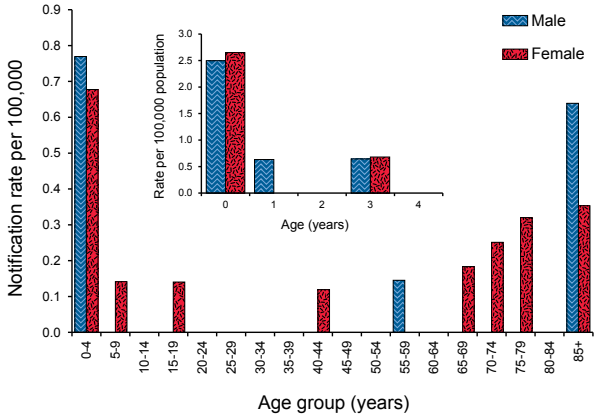


Figure 43: Notification rate for invasive *Haemophilus influenzae* type b infection, Australia, 2013, by age group and sex



Indigenous status

Indigenous status was reported for 90% (n=18) of all cases in 2013. Two cases were reported as being Indigenous, representing a notification rate of 0.3 per 100,000, which was consistent with 2012 and 2011 (0.3 per 100,000 and 0.4 per 100,000 respectively) and lower than 2010 (1.4 per 100,000).

Vaccination

The NIP schedule in 2013 recommended a primary course of 3 doses at 2, 4, and 6 months of age, with additional booster doses at 4 years and between 10 and 15 years, delivered through school-based programs.[50]

In 2013, persons aged less than 21 years of age were eligible for Hib vaccination under the NIP during their infancy. Twelve of the 20 Hib cases reported in 2013 were eligible for vaccination. Of the 5 cases who were 12 months of age or over and therefore eligible for the full vaccine course, none were fully vaccinated. Of the 7 cases who were less than 12 months of age, six were partially vaccinated and one was not vaccinated. Two partially vaccinated cases had received all 3 primary vaccine doses, two had received 2 doses and two had received 1 dose.

Invasive pneumococcal disease

- A total of 1,546 cases of invasive pneumococcal disease were notified in 2013, representing a notification rate of 6.7 per 100,000.
- This was the lowest national rate reported since the introduction of the universal 7-valent pneumococcal conjugate vaccine (7vPCV) program for young children in 2005 and follows the replacement of the 7vPCV with the 13-valent pneumococcal conjugate vaccine (13vPCV) in July 2011.

Invasive pneumococcal disease is a disease in which *Streptococcus pneumoniae* is isolated from a normally sterile site such as blood, cerebrospinal fluid or pleural fluid. Transmission of the bacterium from person to person is usually via the inhalation of infected respiratory droplets. Many of the signs and symptoms of IPD are non-specific including fever, chills, headache, stiff neck and a general feeling of being 'out-of-sorts', through to seizures and occasionally coma.

Epidemiological situation in 2013

There were 1,546 notified cases of IPD reported in 2013, representing a notification rate of 6.7 per 100,000. This was the lowest national rate reported since the introduction of the universal 7vPCV program for young children in 2005.

Geographic description

The number of cases in all states and territories, except for Victoria, decreased in 2013 with the Australian Capital Territory recording the greatest reduction in cases (48% decrease) when compared

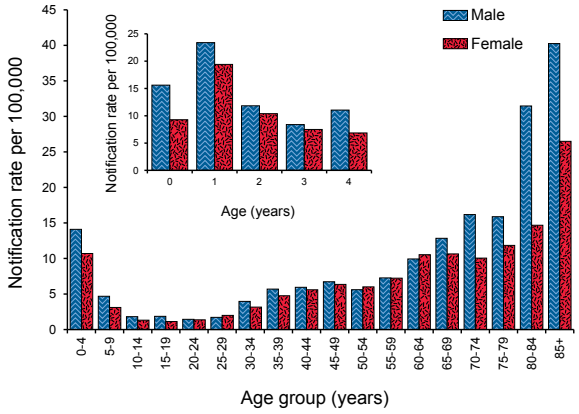
with 2012. The notification rate of IPD varied from 3.7 per 100,000 in the Australian Capital Territory to 24.0 per 100,000 in the Northern Territory. Victoria recorded only a small increase in the number of cases notified, maintaining the same rate as recorded in 2012 (6.8 per 100,000).

Age and sex distribution

In 2013, males accounted for 54% (n=829) of cases of IPD, resulting in a male to female ratio of 1.2:1. The rate for disease in males exceeded that in females in all age groups except for the 25–29, 50–54, 55–59 and 60–64 years age groups (Figure 44).

In 2013, the notification rate for IPD was highest in the elderly and in young children, with an age distribution similar to the distribution seen in 2012. In the elderly, the highest notification rate was in those aged 85 years or over (31.4 per 100,000), while the highest rate in children aged less than 5 years was in those aged 1 year (21.4 per 100,000) (Figure 44).

Figure 44: Notification rate for invasive pneumococcal disease, Australia, 2013, by age group and sex



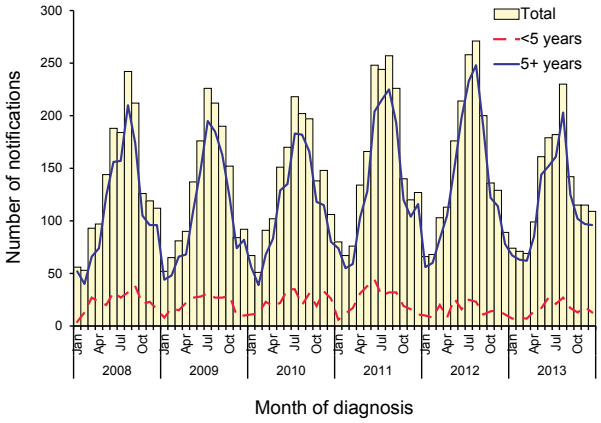
Seasonality

Many respiratory diseases including IPD are known to show a distinct seasonal trend that generally peaks during the winter months. In 2013, the seasonal trend of IPD was consistent with previous years with notifications peaking in August (n=230) (Figure 45).

Indigenous status

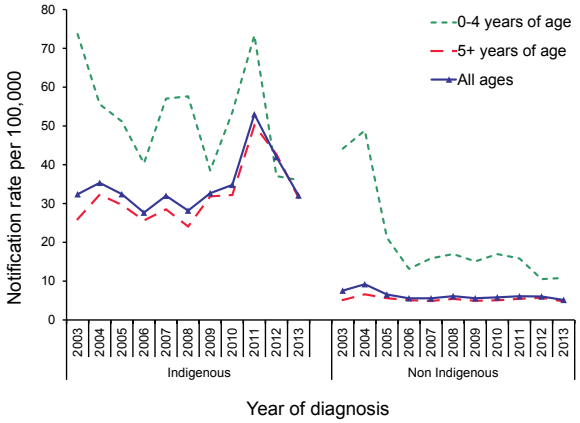
Completeness of Indigenous status reporting was reasonable in 2013, with 88% (n=1,356) of cases reported with a known Indigenous status. Of those cases with a known Indigenous status,

Figure 45: Notified cases of invasive pneumococcal disease, Australia, 2008 to 2013, by month of diagnosis



14% (n=193) of notifications were reported as Indigenous. In 2013, the notification rate for IPD in the Indigenous population (32.1 per 100,000) was approximately 6 times the rate for non-Indigenous people (5.2 per 100,000). In 2013, the notification rate for IPD in the non-Indigenous population was the lowest recorded since the introduction of the universal 7vPCV program for young children in 2005 (Figure 46).

Figure 46: Notification rate for invasive pneumococcal disease, Australia, 2003 to 2013, by Indigenous status, year of diagnosis and age group



2005 – Introduction of universal childhood 7vPCV immunisation program.

July 2011 – The 13vPCV immunisation replaced the 7vPCV component in the universal childhood immunisation program.

In 2013, the notification rate for IPD in Indigenous children aged under 5 years (36.1 per 100,000) reached its lowest since 2005. The rate in non-Indigenous children aged under 5 years (10.8 per

100,000) only slightly exceeds the lowest rate recorded in this subgroup since 2005 (10.5 per 100,000 in 2012).

Vaccination

In Australia, pneumococcal vaccination is recommended as part of routine immunisation for the medically at-risk, children under 5 years of age, Aboriginal and Torres Strait Islander peoples aged 50 years or over and other Australians aged 65 years or over.[50]

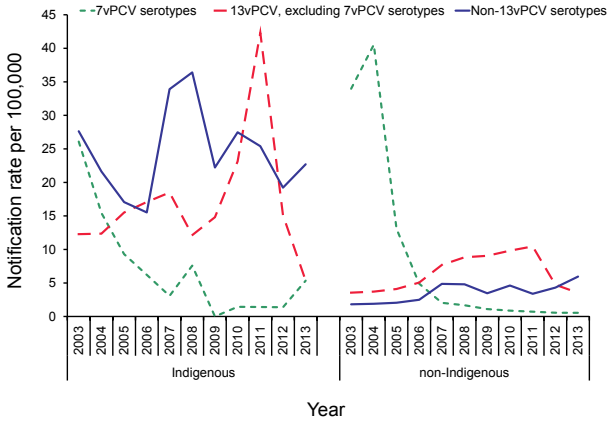
The 7vPCV was added to the NIP schedule in 2001 for Indigenous and medically at-risk children and then expanded in 2005 to include all infants nationally, together with a catch-up vaccination for all children aged less than 2 years. In 2011, the 7vPCV was replaced on the NIP by the 13vPCV and further expanded to include all children aged under 5 years. The 7vPCV targets 7 *S. pneumoniae* serotypes (4, 6B, 9V, 14, 18C, 19F and 23F) and the 13vPCV targets the same 7 serotypes plus 6 additional serotypes (1, 3, 5, 6A, 7F, 19A). In 2013, 37% of notifications in children aged under 5 years were a result of a serotype included in either the 7vPCV or 13vPCV vaccines.

Vaccination with the 23-valent pneumococcal polysaccharide vaccine (23vPPV) was added to the NIP for Indigenous Australians aged 50 years or over in 1999 and for non-Indigenous Australians aged 65 years or over from January 2005.[60] The 23vPPV targets 23 *S. pneumoniae* serotypes (1, 2, 3, 4, 5, 6B, 7F, 8, 9N, 9V, 10A, 11A, 12F, 14, 15B, 17F, 18C, 19A, 19F, 20, 22F, 23F and 33F). In 2013, 63% of notifications in Indigenous peoples aged 50 years or over, and 59% of notifications in non-Indigenous Australians aged 65 years or over, were a result of a serotype included in 23vPPV.

Microbiological trends

Although there are over 90 *S. pneumoniae* serotypes, a relatively limited number cause the majority of IPD. Data on serotypes of pneumococcal isolates is critical for understanding of vaccine effects in both immunised and non-immunised populations such as herd immunity effects and serotype replacement. IPD serotypes were reported in 96% (n=1,491) of notified cases in 2013. The dramatic reduction in IPD due to serotypes targeted by the 7vPCV, following the introduction of the vaccine, in children aged under 5 years has been maintained, although the rate for IPD in Indigenous children aged under 5 years increased from 1.4 per 100,000 (n=1) in 2012 to 5.3 per 100,000 (n=4) in 2013 (Figure 47). This is likely to be a sporadic increase in this group as all 4 cases were caused by a different serotype (14, 18C, 19F and 9V). In 2013, the 7vPCV serotypes accounted for only 7% (n=12) of IPD notifications with known serotype in children aged under 5 years.

Figure 47: Notification rate for invasive pneumococcal disease in children aged less than 5 years, Australia, 2002 to 2013, by Indigenous status, year and serotype category



2001 – Introduction of 7vPCV immunisation for Aboriginal and Torres Strait Islander and medically at-risk children and 23vPPV booster for Aboriginal and Torres Strait Island children in the Northern Territory, Western Australia, South Australia and Queensland.

2005 – Introduction of universal childhood 7vPCV immunisation program.

July 2011 – The 13vPCV immunisation replaced the 7vPCV component in the universal childhood immunisation program.

Enhanced surveillance data sets

Enhanced data are available for IPD notifications. Further analyses, including risk factors and antibiotic susceptibilities can be found in the IPD annual report series also published in CDI.

Prior to 2011, an increasing trend in IPD due to the 6 additional serotypes targeted by the 13vPCV (13vnon7v), indicative of serotype replacement, was observed in children under 5 years of age. However, since the introduction of the 13vPCV in 2011, the rate for IPD due to the 13vnon7v serotypes in both Indigenous children and non-Indigenous children has reduced to the lowest rate recorded in a decade (5.3 per 100,000 and 3.5 per 100,000 respectively). Overall in 2013, 13vnon7v serotypes accounted for 31% (n=58) of IPD notifications in children aged under 5 years compared with 44% (n=82) in 2012. For both Indigenous and non-Indigenous children, the most common 13vnon7v serotype causing disease in 2013 was still serotype 19A: 100% of cases in Indigenous children and 61% of cases in non-Indigenous children.

Measles

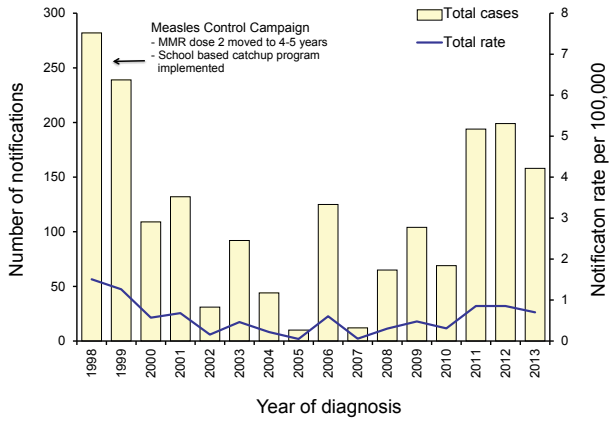
- In 2013, there were 158 notified cases of measles, the majority of which were either imported or import-related.
- There were 20 clusters of two or more epidemiologically linked cases in 2013.
- Transmission was interrupted quickly in all except 1 outbreak involving 44 cases over 17 weeks.
- Of cases eligible for vaccination, the majority were either not vaccinated (43%) or their vaccination status was not able to be established (38%).

Measles is a highly infectious, acute viral illness spread by respiratory secretions, including aerosol transmission.[61] Initial symptoms last 2 to 4 days and are characterised by fever and malaise, followed by a cough, coryza and conjunctivitis. It is usually followed by a red blotchy rash, which typically begins on the face, and then becomes generalised. Measles is often a severe disease with complications more common in the chronically ill, including otitis media, pneumonia, diarrhoea and acute encephalitis.[62] Subacute sclerosing panencephalitis is a late, rare (approximately 1 in 100,000 cases) complication of measles caused by persistent infection and is always fatal.[50] Complications are more common in children under 5 years of age and in adults over 20 years of age.[63]

Epidemiological situation in 2013

In 2013, there were 158 notifications of measles. This represents a notification rate of 0.7 per 100,000, which is 1.3 times the mean of the previous 5 years but a decrease compared with 2012 and 2011, where 199 and 194 cases were reported respectively (Figure 48).

Figure 48: Notified cases and notification rate for measles, Australia, 1998 to 2013, by year



Geographic description

In 2013, cases of measles occurred in all states and territories, except the Northern Territory and Tasmania. The majority of cases occurred in Queensland (n=52), followed by Victoria (n=41), New South Wales (n=34), South Australia (n=16), Western Australia (n=14) and the Australian Capital Territory (n=1) (Figure 49).

Age and sex distribution

The overall male to female ratio was 1.3:1 in 2013, representing a male rate for 0.8 per 100,000 compared with a female rate for 0.6 per 100,000. There was a wide variation in the male to female rate ratio across the age groups with 3 times as many males compared with females in the 25–29 years age group (n=13 and n=4 respectively) and equal numbers in the 0–4 years age group (n=10) (Figure 50)

In 2013, age at diagnosis ranged from 0 to 51 years with a median age of 21 years. Notification rates decreased or remained consistent across all age groups in 2013, compared with 2012. Consistent with recent years, infants less than 1 year of age had the highest age specific rate, 2.3 per 100,000, in 2013. Rates have remained below 2.5 per 100,000 in all age groups between 2008 and 2013, with the exception of the less than 1 year age group in 2011 and 2012 (Figure 51).

Twenty-three cases occurred in those born between 1978 and 1982 (31–35 years of age in 2013), a cohort previously identified as susceptible to measles infection.[64] One case was born before 1966, a cohort that is considered to have high levels of natural immunity.[65]

Seasonality

In Australia, a seasonal pattern is no longer evident due to the virus no longer being endemic (Figure 49). In temperate climates and where measles transmission remains endemic, the majority of cases occur in late winter to early spring.[66]

Indigenous status

Indigenous status was completed for 91% of cases in 2013 (n=143), a decrease compared with the 98% of cases in 2012. Of these cases, 2.1% (n=3) were reported as Indigenous.

Figure 49: Notified cases of measles, Australia, 2008 to 2013, by month of diagnosis and state and territory

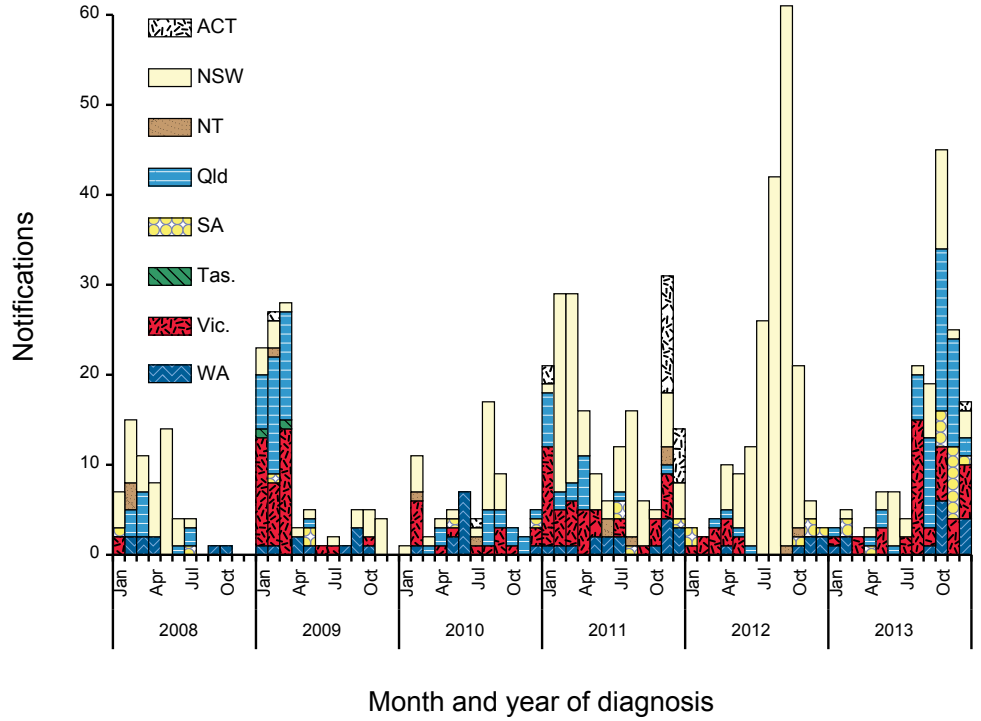


Figure 50: Notification rate for measles, Australia, 2013, by age group and sex

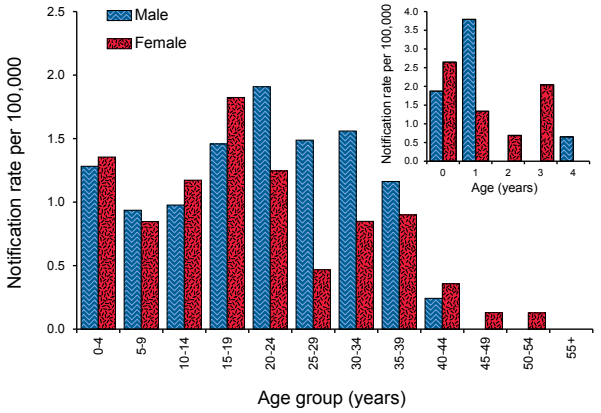
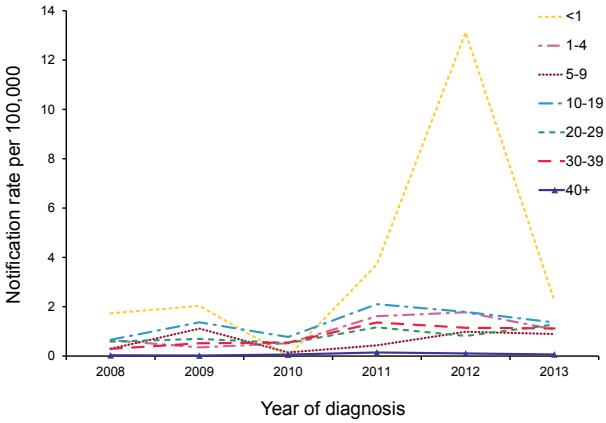


Figure 51: Notification rate for measles, Australia, 2008 to 2013, by year of diagnosis and selected age groups



Source of infection and outbreaks

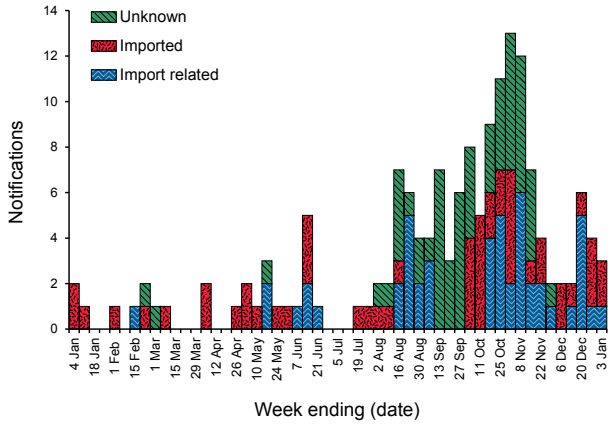
Sixty-four per cent of cases in 2013 were either imported (n=52) or import-related (n=49) with the remaining 36% (n=57) of unknown source (Figure 52).

Of the imported cases, 65% (n=34) were from the WHO defined South East Asia Region, the majority of which were from Indonesia (n=20). The WHO Western Pacific Region, of which Australia is a part, accounted for 17% (n=9) of imported cases. The remaining 18% from the WHO Eastern Mediterranean Region (n=4) and the European Region (n=5).

There were 20 clusters of two or more epidemiologically linked cases in 2013 accounting for 75% (n=119) of all cases. The remaining 25% of cases comprised sporadic imported cases (n=32) and sporadic cases acquired in Australia of unknown source (n=7). The majority of clusters were import related (n=17) comprising 58% (n=69) of cluster cases. The 3 clusters of locally-acquired cases of unknown source occurred in 3 separate states

including Western Australia: 1 cluster of 2 cases; New South Wales: 1 cluster of 4 cases; and the large outbreak involving both Victoria and Queensland (n=44 cases).

Figure 52: Notified cases of measles, Australia, 2013, by diagnosis week ending and source of infection



Transmission was interrupted quickly in all except 1 outbreak. The median duration was 18 days (range 1–118 days) between the onset of symptoms in the index and the last case and the median numbers of generations[67] was 2 (range 0–12). Nineteen of 20 clusters had less than 10 cases with a median of 3.5 (range 2–44) cases. The largest outbreak, comprising 44 cases, commenced in Victoria (n=7) with subsequent linked cases in Queensland (n=37). This outbreak lasted approximately 17 weeks from the end of July and included 12 generations of spread. While it was classified as being of unknown source, it was most likely associated with an imported case at an international gaming convention in Melbourne, which the index case had attended during the exposure period.

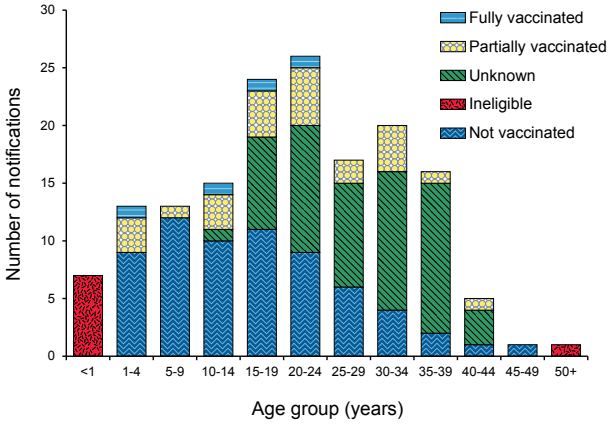
Vaccination

Two doses of the measles containing vaccine are recommended for all persons born during or after 1966. The MMR induces long term measles immunity in 95% of recipients after a single dose and 99% of recipients after the 2nd dose.[50]

Of the 158 cases notified in 2013, 95% (n=150) were born after 1965 and were 12 months of age or over and therefore eligible for at least 1 dose of a publicly funded measles-containing vaccine. Over 80% of cases eligible for vaccination were either not vaccinated (43%, n=65) or of unknown vaccination status (38%, n=57). Of the remaining 19% (n=28) who were vaccinated, four had received the full course of 2 doses of a measles-containing vaccine and 24 were partially vaccinated with 1 dose (Figure 53).

The 5–9 years age group had the highest proportion of unvaccinated cases (18%) with young children and adolescents between 5 and 19 years of age accounting for 51% of all unvaccinated cases. The proportion of cases with unknown vaccination status increases with age and where provided may be less reliable mostly being based on self-reporting. In 2013, there was 1 case less than 15 years of age reported as of unknown vaccination in contrast to 44% (n=48) of cases 15 years or over having unknown vaccination status (Figure 53).

Figure 53: Notified cases of measles, Australia, 2013, by selected age groups and vaccination status

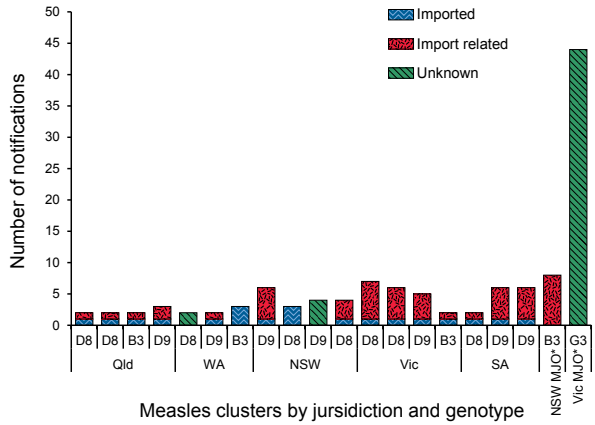


Microbiological trends

Genotyping data were available for all 20 clusters with 2 or more linked cases in 2013. Genotype D9 was associated with 7 separate clusters (n=32 cases), D8 with 8 clusters (n=28 cases), B3 with 3 clusters (n=15 cases) and G3 with the large outbreak across Victoria and Queensland (n=44 cases) (Figure 54). Of the 39 sporadic cases 72% (n=28) were genotyped.

Imported genotypes varied by WHO region. A single genotype was imported from 2 regions; 4 separate importations of B3 from the Eastern Mediterranean Region and 3 separate importations of D8 from the European Region. Multiple genotypes were imported from the South East Asia Region (B3, D8, D9 and G3) and the Western Pacific Region (B3, D8 and D9).

Figure 54: Measles clusters, Australia, 2013, by state or territory, genotype and source of infection



MJO = multi-jurisdictional outbreak

Discussion

The increasing prevalence of measles in some parts of the world and the continued circulation of the virus in countries of close geographical proximity to Australia will result in a continual source of imported virus in Australia. This was particularly the case in 2013 with 52 separate importations occurring. Despite this large number of importations in 2013, the majority were sporadic and did not lead to ongoing local transmission.

Evidence suggests that endemic measles has been eliminated from Australia, since at least 2005.[66] Based on the WHO definitions, Australia has continued to maintain this status. In 2013, none of the outbreaks persisted for more than 12 months and there was no evidence of a single genotype continuously circulating. Ongoing evidence of high population immunity was demonstrated by the small number of cases and the short duration of outbreaks. Only 1 outbreak in 2013 involved more than 4 generations of transmission, or lasted greater than 6 weeks.

However, due to the highly infectious nature of measles, local transmission and outbreaks will continue to occur, mostly among susceptible contacts of non-immune travellers from countries where measles remains prevalent.

Mumps

- The mumps notification rate has been less than 1 per 100,000 since 2009.
- In 2013 there were 217 notified cases of mumps.

Mumps is an acute viral illness with an incubation period of 12 to 25 days. Transmission is usually by respiratory secretions, including aerosol transmission, or by direct contact with saliva. Asymptomatic infections occur in one-third of cases. Symptomatic disease ranges from mild upper respiratory tract infections to systemic involvement. The characteristic bilateral, or occasionally unilateral, parotid swelling occurs in 60% to 70% of clinical cases, however a high proportion have non-specific symptoms including fever, headache, malaise, myalgia and anorexia.[68] Mumps encephalitis has been estimated to occur in 1 to 2 per 10,000 cases, with a case fatality rate of around 1%.

Epidemiological situation in 2013

In 2013, there were 217 notifications of mumps. A notification rate of 0.9 per 100,000, which represented an 8.5% increase compared with the 200 cases reported in 2012 and continues the slight upward trend noted since 2011 (Figure 55). From 2007 to 2010 the overall notification rate of mumps declined, falling from a peak of 2.8 per 100,000 in 2007 to 0.4 per 100,000 in 2010.

Geographic description

Cases were reported from all states and territories. Jurisdictional specific rates were highest in the Northern Territory (2.5 per 100,000) followed by Western Australia (1.8 per 100,000).

Age and sex distribution

In 2013, the overall male to female ratio was 1.4:1, with some variation in this ratio between age groups. The highest rates for males occurred in the 15–19 years age group at 2.5 per 100,000, while for females rates were highest in the 40–44 years age group at 1.4 per 100,000 (Figure 56).

There were cases of mumps notified across all age groups with the median age at diagnosis being 32 years (range 0–90 years). Consistent with recent years, young adults in the 30–39 and 20–29 years age groups had the highest rates of infection with 1.6 per 100,000 and 1.4 per 100,000 respectively. The most notable increase in age group rates occurred among children less than 1 year of age (Figure 57).

Indigenous status

A known Indigenous status was reported for 79% (n=171) of mumps cases in 2013. This was higher than the level of completeness over the previous 5-year period (mean 63%, range 51% to 77%). Of the cases with a known Indigenous status reported,

Figure 55: Notified cases of mumps, Australia, 2008 to 2013, by month of diagnosis and state or territory

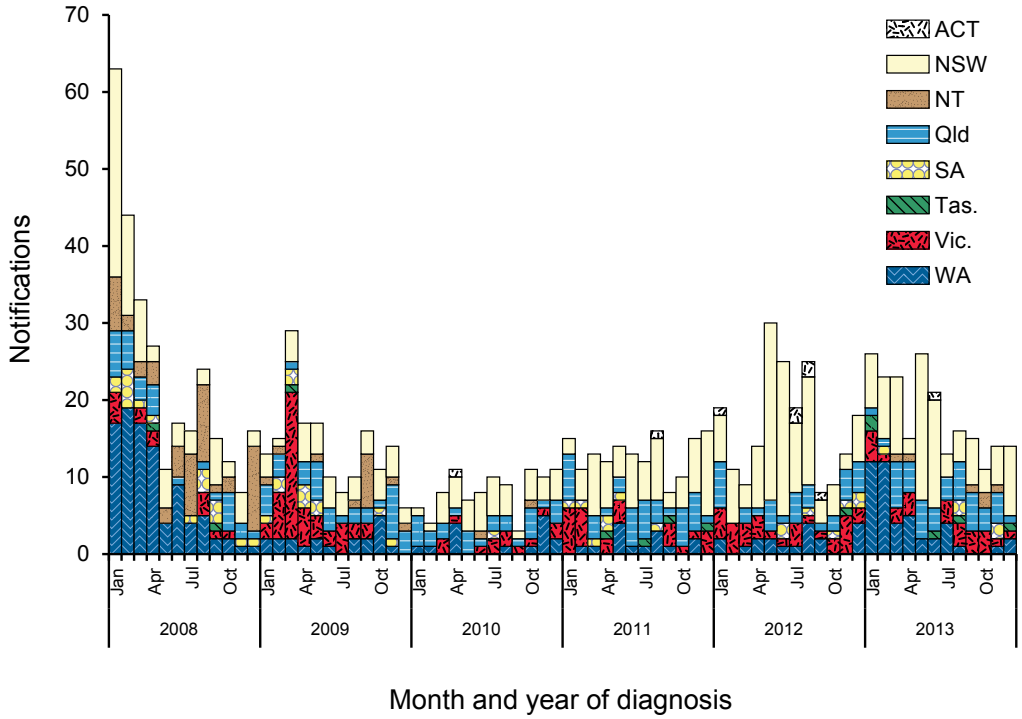


Figure 56: Notification rate for mumps, Australia, 2013, by age group and sex

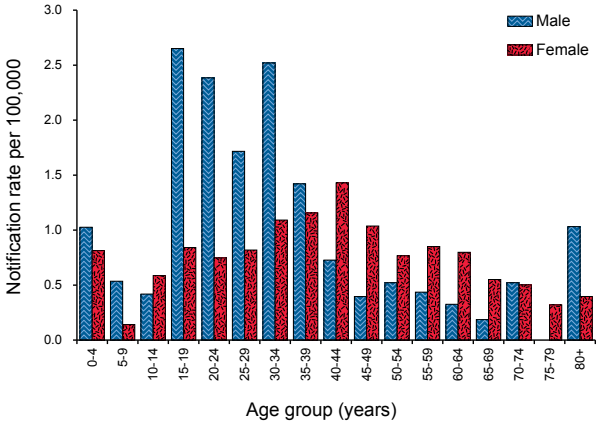
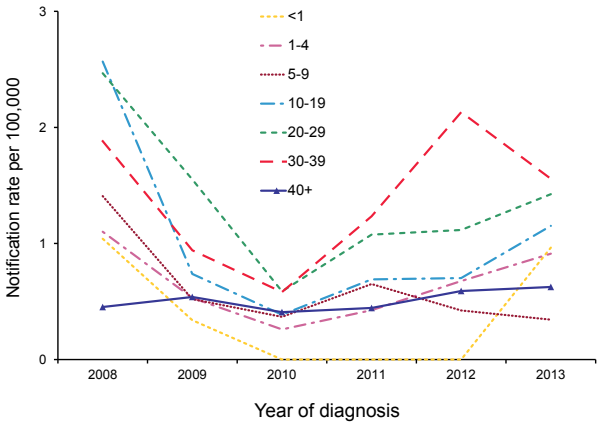


Figure 57: Notification rate for mumps, Australia, 2008 to 2013, by year of diagnosis and selected age groups



5 cases (3%) were reported as Indigenous. The proportion of mumps notifications reported as Indigenous has been less than 5% since 2010.

Outbreaks

Place of acquisition was complete for 74% (n=160) of cases in 2013 of which 18% were imported from overseas: 21 from Asia, five from Europe, two from the Americas and one from Africa. Eighty-two per cent were reported as locally acquired in Australia.

The outbreak reference field was completed for 6% (n=13) of cases in 2013. There were 6 outbreaks of two or more epidemiologically linked cases, all of which occurred in Western Australia. Two of these outbreaks were linked to imported cases.

Vaccination

The mumps vaccine was first funded on the NIP schedule in 1982 for infants at 12 months of age, with those born after 1980 eligible for at least 1 dose of a mumps-containing vaccine.

The mumps component of the MMR vaccine is considered to be the least effective of the 3 com-

ponents with 1 dose vaccine effectiveness varying between 60% and 90%.[69–71] While protection is greater in 2-dose vaccine recipients, recent outbreaks have been reported among these, particularly young adults who received their vaccines more than 10 years previously.[72,73] Reduced effectiveness of the mumps vaccine over time may also partially account for the proportion of vaccinated cases and contribute to mumps outbreaks in older vaccinated populations.[74]

Of the 217 cases in 2013, 50% (n=109) were eligible for at least 1 dose of a publicly funded mumps-containing vaccine. Of these, 12% (n=13) were unvaccinated and 50% (n=54) were of unknown vaccination status. Of the remaining 38% of cases (n=42), 18 were fully vaccinated, having received 2 doses of a mumps-containing vaccine, 22 were partially vaccinated with 1 dose of a mumps-containing vaccine and 2 cases had no dose number information provided.

Pertussis

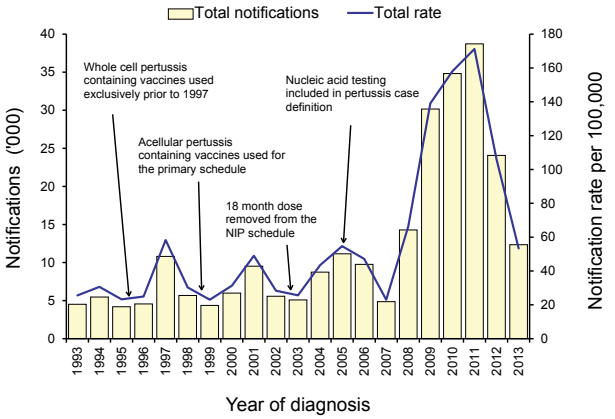
- Pertussis is the least well controlled of all VPDs and remains highly prevalent in Australia.
- In 2013 there were 12,341 cases of pertussis reported, representing a notification rate of 106 per 100,000 population and continuing the downward trend in annual notifications since 2011.
- In 2013, children under 15 years of age had a notification rate 2.7 times higher compared with those 15 years of age or over.

Pertussis, commonly known as whooping cough, is a highly infectious respiratory disease caused by *Bordetella pertussis* and is spread by respiratory droplets. The characteristic paroxysmal cough with inspiratory whoop seen among unvaccinated children is less common in individuals who have some acquired immunity from vaccination or infection.[75] Most deaths occur in unvaccinated infants under 6 months of age. Complications include pneumonia, atelectasis, seizures, encephalopathy, and hernias, with pneumonia as the most common cause of death.[21]

Epidemiological situation in 2013

In 2013, there were 12,341 notifications of pertussis. A 49% decrease in notified cases compared with 2012 (n=24,074) and 14% less than in 2008 (n=14,286) the year in which the most recent Australia-wide epidemic,[76] which peaked in 2011, began (Figure 58 and Figure 59). There were no pertussis related deaths reported in 2013.

Figure 58: Notified cases and notification rate for pertussis, Australia, 1993 to 2013, by year of diagnosis



Geographic description

In 2013, all jurisdictional specific rates decreased compared with 2012. Despite the timing of peak pertussis activity in the most recent epidemic period varying across jurisdictions, in 2013 most jurisdictional specific rates had returned to or were approaching, pre-epidemic levels. However, activity remained high in Tasmania (100 per 100,000) and Western Australia (65 per 100,000) compared with pre-epidemic rates in those states (Figure 60).

Age and sex distribution

Females accounted for 57% (n=6,986) of cases in 2013. Females had higher rates across all age groups, except among adults aged 80 years or over (Figure 61). The highest notification rate in both males and females occurred in the 5–9 years age group (117 and 130 per 100,000 respectively). Notification rates in females were on average 1.6 times that of males in the 25–64 years age groups.

In 2013, the trend prominent in this recent epidemic period of higher notification rates in children less than 15 years of age compared with those 15 years of age or over continued. Children less than 15 years of age represented 39% (n=4,807) of notifications and had a notification rate (110 per 100,000) 2.7 times higher compared with those 15 years of age or over (40 per 100,000). However, rates in children less than 15 years of age have declined steeply since reaching a peak in 2011. The highest age specific rates in 2013 occurred in the 5–9 years age group (123 per 100,000) consistent with the trend since 2010, while all age group rates continued the downward trend commenced in 2012 (Figure 62).

Figure 59: Notified cases of pertussis, Australia, 2008 to 2013, by month and year of diagnosis and state or territory

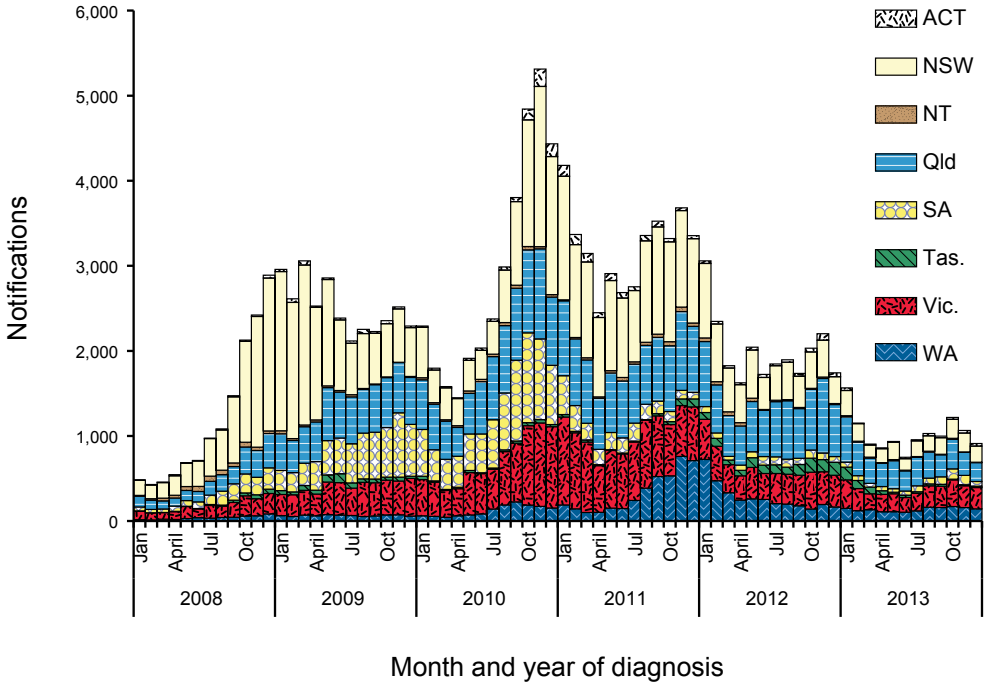


Figure 60: Notification rate for pertussis, 2008 to 2013, by year of diagnosis and state and territory

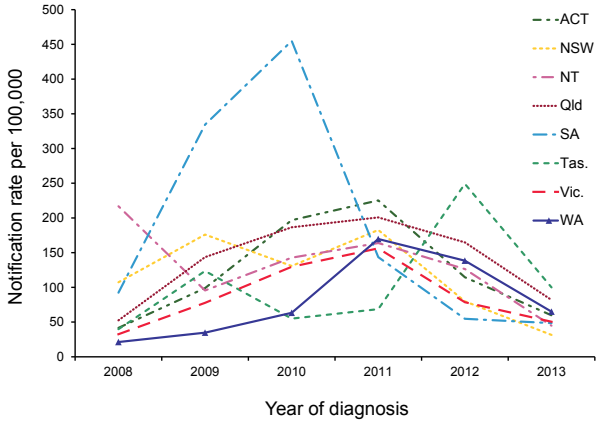


Figure 61: Notification rate for pertussis, Australia, 2013, by age group and sex

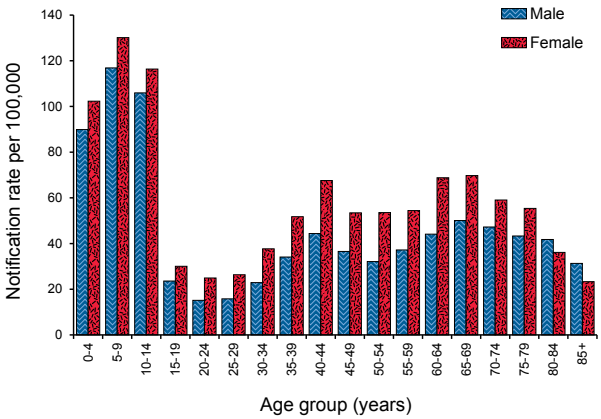
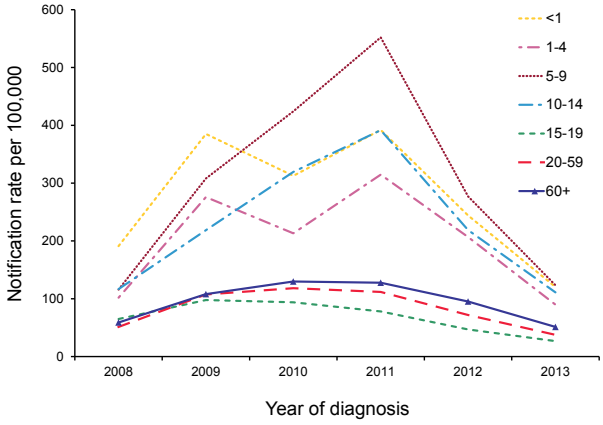


Figure 62: Notification rate for pertussis, Australia, 2008 to 2013, by year of diagnosis and selected age groups



Vaccination

The NIP schedule in 2013 included a primary course of 3 doses of vaccine at 2, 4, and 6 months of age, with additional booster doses provided at 4 years of age and between 10 and 15 years of age.[50]

Pertussis vaccine effectiveness among Australian children has been estimated to range from 82%–89% with the lower figure representing the cohort of children who would not have been eligible for the 18-month booster dose, which was removed from the NIP in 2003.[77] Immunity to disease decreases over time post-vaccination with estimates of protection remaining for 4–12 years.[78–80]

In order to determine the vaccination status of cases, public health follow-up is required. During large epidemic periods the follow-up of all cases is not feasible and as per the pertussis national guidelines for public health units,[81] jurisdictions prioritise case follow-up to those less than 5 years of age. During 2013, those aged less than 5 years accounted for 12% (n=1,458) of all notified cases and information about vaccination status was available for 93% (n=1,350) of these cases.

For children eligible to receive the full primary course of 3 vaccinations, 66% (n=664) had done so and 24% (n=49) of those eligible had received the full scheduled course of 4 doses (Table 15).

While pertussis can affect people of any age, infants are at highest risk of more severe disease as adequate immunity is not achieved through infant vaccination until receiving at least the 2nd vaccine dose at 4 months of age.[82] Seventy-one per cent (n=1,029) of cases less than 5 years of age had received at least 2 doses of a pertussis-containing vaccine.

Discussion

Epidemics of pertussis have historically occurred at regular intervals of approximately 4 years on a background of endemic circulation in Australia. The most recent epidemic appears to be over, with most jurisdictions reporting pertussis activity consistent with pre-epidemic levels. Notification rates have decreased in all states and territories and across all age groups in 2013 compared with their epidemic peaks. Most jurisdictions correspondingly ceased their respective cocooning programs in 2012, which included various combinations of providing free booster vaccinations to pregnant women, parents and carers of infants with only the Northern Territory and New South Wales continuing this strategy into 2013.

Poliomyelitis

- Australia was certified by the WHO in 2000 as having eradicated Indigenous poliovirus.
- There were no cases of poliomyelitis identified in Australia in 2013.

Poliomyelitis is a highly infectious disease caused by gastrointestinal infection by poliovirus. Transmission occurs primarily person-to-person via the faecal-oral route. In most cases, poliovirus infection is not symptomatic; however in less than 1% of cases the virus may invade the nervous system and cause acute flaccid paralysis (AFP).[21]

Vaccines formulated with inactivated poliovirus, are available in combination with diphtheria toxin, tetanus and other antigens. The NIP schedule in 2013 recommended a primary course of 3 doses at 2, 4, and 6 months of age, with additional booster doses at 4 years and between 10 and 15 years, delivered through school based programs.[50]

In 2013 there were no notifications of poliomyelitis. Australia, along with the Western Pacific Region, remains poliomyelitis free.

Poliomyelitis is a notifiable disease in Australia with clinical and laboratory investigation conducted for cases involving patients of any age with a clinical suspicion of poliomyelitis. Australia follows the WHO protocol for poliomyelitis surveillance and focuses on investigating cases of AFP in children under 15 years of age. The WHO target for AFP surveillance in a polio free country is 1 case of AFP per 100,000 children less than 15 years of age. Australia has achieved this surveillance target in all years since 2008. However, the virological surveillance indicator of adequate stool specimen collection in 80% of AFP cases has never been met. More details can be found in the annual report

Table 15: Notified cases of pertussis in children aged 0 to 5 years, Australia, 2013, by age group and number of doses of vaccine

Age group	Number of vaccine doses					Unknown	Total
	0	1	2	3	4		
Less than 6 weeks of age (not eligible for vaccination)	49	5				20	74
6 weeks to <4 months (eligible for 1 dose of vaccine)	22	72	6			7	107
4 to < 6 months (eligible for two doses of vaccine)	7	17	33			1	58
6 months to < 4 years (eligible for 3 doses of vaccine)	85	33	174	662	2	56	1,012
4 to 5 years (eligible for 4 doses of vaccine)	27	4	28	75	49	24	207
Total	190	131	241	737	51	108	1,458

series published in the CDI by the Australian Enterovirus Reference Laboratory who coordinate poliovirus surveillance activities in Australia.

Rubella and congenital rubella syndrome

- Rubella is a rare disease in Australia.
- Since 2003, rubella notifications have been less than 0.3 per 100,000.
- In 2013 there were 25 cases of rubella and 2 cases of congenital rubella syndrome reported.

Rubella is generally a mild and self-limiting viral infectious disease. It is spread from person to person through contact with respiratory secretions, including aerosol transmission. Clinically, rubella can be difficult to distinguish from other diseases that also cause febrile rash, such as measles, and is asymptomatic in up to 50% of cases.[21]

Epidemiological situation in 2013

In 2013 there were 25 cases of rubella reported, which was a rate of 0.1 per 100,000. While this is consistent with the low rates of this disease experienced since 2003, it is a marked decline from the peak rate of more than 30 per 100,000 in 1995 (Figure 63). Indigenous status was recorded for all cases, none of which were reported as Indigenous. There were 2 cases of congenital rubella syndrome (CRS) reported in 2013. Both cases were imported, one each from Nepal and Thailand. The first was a newly arrived refugee infant born overseas and diagnosed later on arrival in Australia. The 2nd infant was born in Australia to a non-immune mother who acquired her infection whilst overseas.

Geographic distribution

Cases were reported from New South Wales (n=12), Queensland (n=6), Victoria (n=3), South Australia (n=2) and one each from the Australian Capital Territory and Western Australia (Figure 64).

Figure 63: Notification rate for rubella, Australia, 1991 to 2013, by year of diagnosis

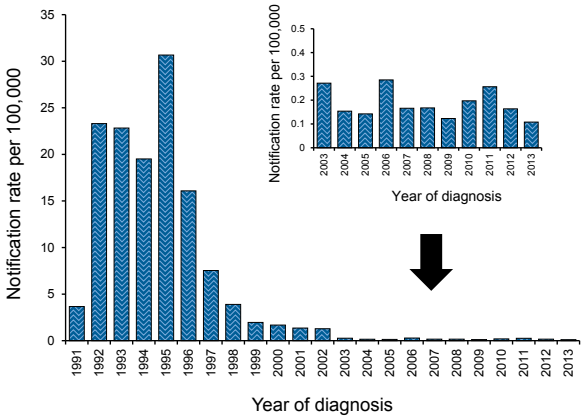
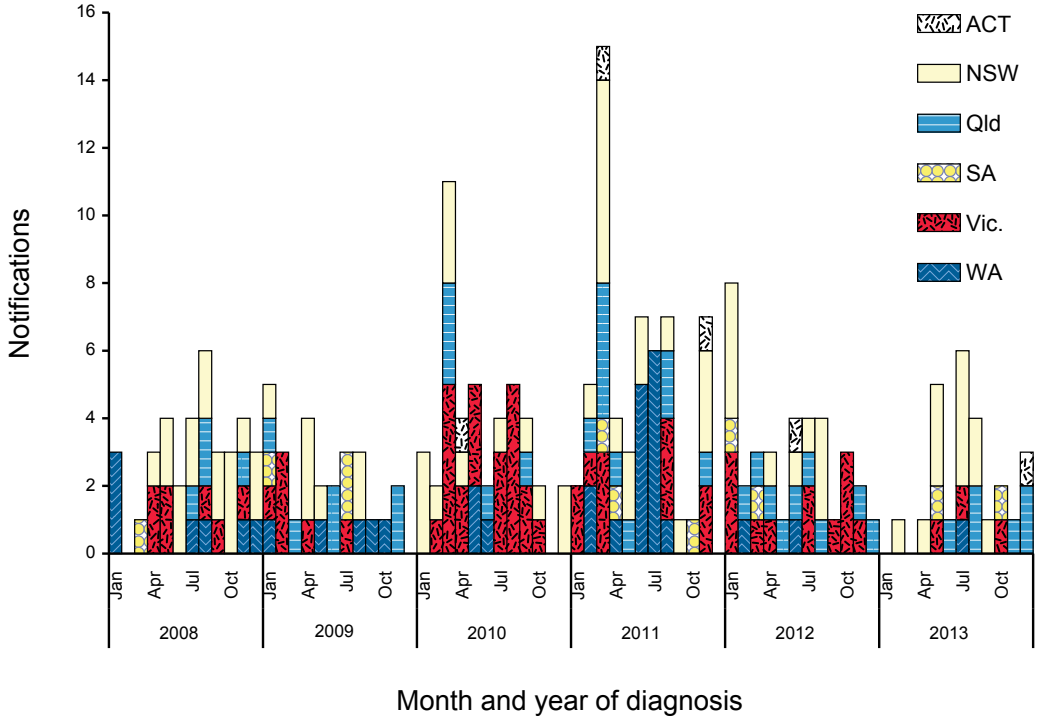


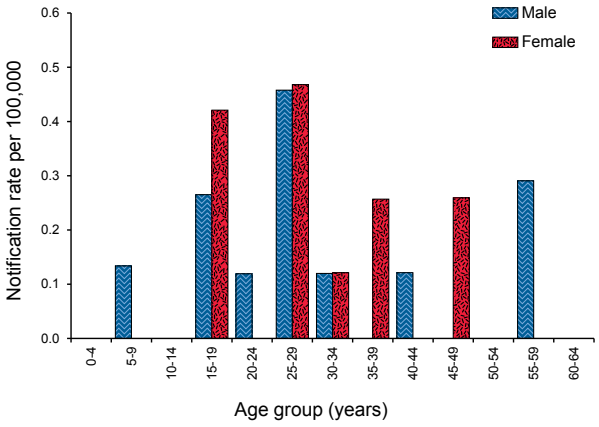
Figure 64: Notified cases of rubella, Australia, 2008 to 2013, by month and year of diagnosis and state or territory



Age and sex distribution

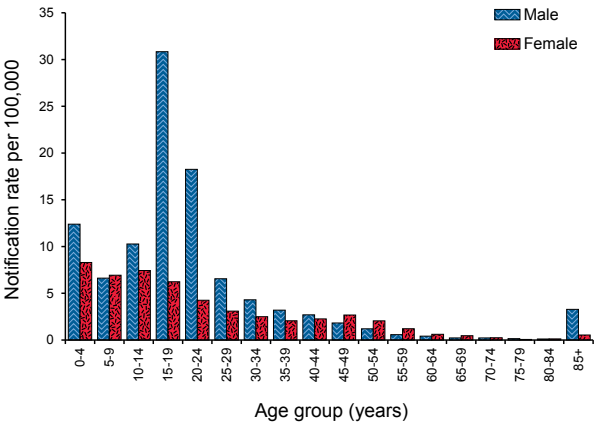
There were approximately equal numbers of males (n=13) and females (n=12) in 2013. The median age was 29 years (range 8–85 years) (Figure 65). Consistent with previous years, the majority of cases (52%) occurred in adults aged 20–39 years of age. Of all female cases, 83% (n=10) were notified in women of child bearing age (15–44 years) (Figure 65).

Figure 65: Notification rate for rubella, Australia, 2013, by age group and sex



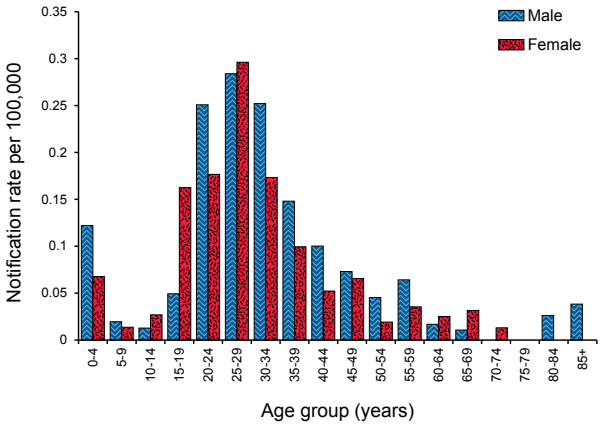
When reviewing the age and sex trend since 1991, a change in the epidemiology is evident. Between 1991 and 2002, males represented 67% of all notifications. The highest age group rates during this period occurred in the male 15–19 years age group at 31 notifications per 100,000 (Figure 66). In contrast, between 2003 and 2013 the male to female rate ratio was roughly equal with males representing 54% of all notifications. During this later period the highest rates occurred at an older age occurring in the 25–29 years age group

Figure 66: Notification rate for rubella, Australia, 1991 to 2002, by age group and sex



(0.3 per 100,000) (Figure 67). The median age also increased from 18 years of age between 1991 and 2002 to 29 years of age between 2003 and 2013.

Figure 67: Notification rate for rubella, Australia, 2003 to 2013, by age group and sex



Vaccination

Rubella vaccine is provided in the combined MMR or MMRV vaccine and in 2013 was provided under the NIP schedule at 12 months and 4 years of age. From 1 July 2013, the 2nd dose is recommended at 18 months of age.[50]

The primary aim of immunisation against rubella is to prevent cases of CRS.[83] Two doses of a rubella containing vaccine are recommended for all non-immune persons born during or since 1966 and who are greater than 18 months of age.

Of the 25 cases notified in 2013, 72% (n=18) were of unknown vaccination status and a further 24% (n=6) were reported as unvaccinated. One case was vaccinated; an 8-year-old child who had received both doses of a rubella-containing vaccine. The high level of incompleteness in this field for rubella makes any additional analysis difficult.

Discussion

The WHO Western Pacific Region, of which Australia is a member, has also endorsed accelerated rubella and CRS goals, and more recently proposed a regional goal of elimination with a target year yet to be determined.[84]

Evidence suggests that endemic rubella is well controlled in Australia. A marked decline in rubella notifications since 2003 has seen rates consistently well below the 1 per 100,000 WHO goal indicative of rubella control.[85] The increasing trend in

age of notifications likely reflects the declining rates of rubella among children since routine MMR immunisation was implemented and the subsequent achievement of high 2 dose coverage. Males, historically more susceptible because universal vaccination was not introduced until 1989, no longer appear to be at greater risk of infection compared with females.

Congenital rubella syndrome is rare in Australia and in recent years mainly occurs among infants of overseas-born women, a cohort previously identified as being at risk of non-immunity to rubella.

Improvements in surveillance data would include more routine genotyping of cases and increased completeness of vaccination status, particularly among high risk groups.

Tetanus

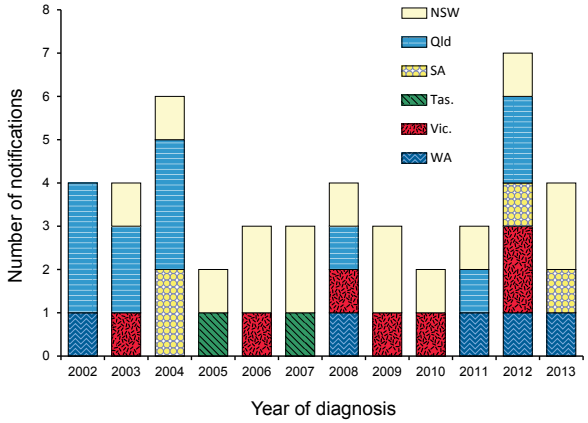
- Cases of tetanus are uncommon in Australia.
- Cases generally occur in older unvaccinated people or in those who have not received a booster dose in the last 10 years.
- In 2013, there were 4 cases of tetanus reported, with no notified deaths

Tetanus is an acute, often fatal, disease caused by the toxin produced by the bacterium *Clostridium tetani*. Tetanus spores usually enter the body through contamination of a wound with manured soil.[21] The neurotoxin acts on the central nervous system to cause muscle rigidity with painful spasms. Generalised tetanus, the most common form of the disease, is characterised by increased muscle tone and generalised spasms. The disease usually occurs after an incubation period of 3 to 21 days (ranging from 1 day to several months), with a median time of onset at 10 days post injury. In Australia, tetanus is rare, occurring primarily in older adults who have never been vaccinated or were vaccinated in the remote past. A high level of diagnostic awareness of tetanus is important in the elderly, as most deaths occur in people over 70 years of age, especially women, and may be associated with an apparent minor injury.[50]

Epidemiological situation in 2013

In 2013, there were 4 notifications of tetanus, which was consistent with the low numbers of this disease notified in recent years. The place of acquisition for 3 cases was reported as Australia (Figure 68). There were no reported deaths due to tetanus.

Figure 68: Notified cases of tetanus, Australia, 2002 to 2013, by year of diagnosis and state or territory



Age and sex distribution

The 4 cases comprised 3 males and 1 female. One case was in the 30–34 years age group and the remaining 3 cases were over 60 years of age.

Indigenous status

Indigenous status was complete for 3 of the 4 cases, none of which were reported as Indigenous.

Vaccination

The NIP schedule in 2012 recommends a primary course of tetanus vaccination including 3 doses provided at 2, 4, and 6 months of age. Two booster doses are provided at 4 years and between 10 and 15 years delivered through school based programs. Booster doses are additionally recommended for all adults at the age of 50 years who have not received one in the previous 10 years. Complete immunisation induces protection lasting throughout childhood but by middle age 50% of vaccinees have low or undetectable levels of antibodies. However, tetanus is uncommon in people who have received 4 or more doses of a tetanus-containing vaccine and in those who received their last dose within 10 years.[50]

Of the 4 cases in 2013, one had received a single dose of a tetanus-containing vaccine and the remaining 3 cases, all of whom were over 60 years of age, were either not vaccinated or reported with an unknown vaccination status.

Varicella zoster virus

- In 2013, a total of 16,986 cases of varicella zoster virus infection were reported, which was an increase of 14% from 2012.
- 58% of cases were reported as unspecified varicella zoster infection, 30% of cases were reported as shingles and 12% of cases were reported as chickenpox.

The varicella zoster virus (VZV) is a highly contagious member of the herpesvirus family and causes 2 distinct illnesses; chickenpox as the primary infection; and following initial infection, shingles (herpes zoster), which occurs following reactivation, often many years later, of latent virus in approximately 20% to 30% of cases of chickenpox overall. Shingles occurs more frequently among older adults (most commonly after 50 years of age) and in immunocompromised people.[21]

In 2006, CDNA agreed to make the 3 categories of VZV infection nationally notifiable; 'chickenpox', 'shingles' and 'varicella zoster virus unspecified'. By 2009 all jurisdictions were notifying VZV infections to the NNDSS with the exception of New South Wales, where VZV is not notifiable.

The ability to categorise a VZV infection as chickenpox or shingles depends largely on clinical evidence. Due to the absence of information on clinical presentation for many cases, the majority of VZV infections nationally are reported as unspecified.

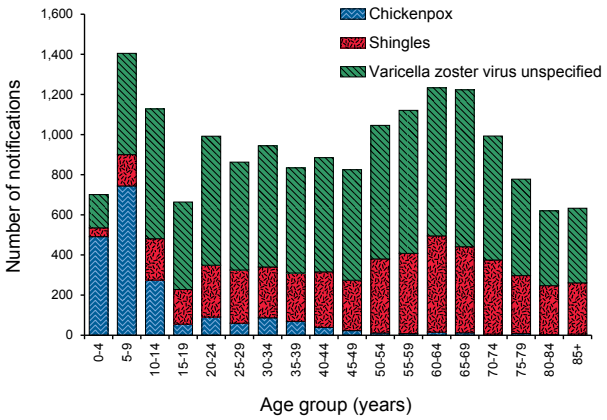
Epidemiological situation in 2013

In 2013, there were 16,986 VZV notifications from the 7 reporting jurisdictions. This was a 14% increase on cases notified in 2012 (n=14,898). Of the total VZV notifications in 2013, 58% (n=9,927) of cases were reported as unspecified varicella infection, 30% (n=5,071) as shingles and 12% (n=2,042) as chickenpox (Figure 69 and Figure 70).

Varicella zoster virus (unspecified)

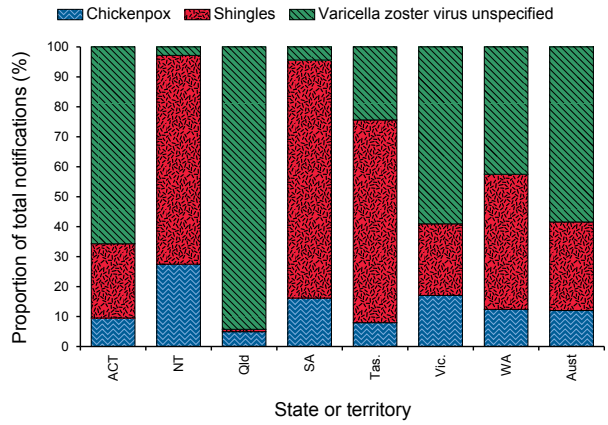
- Notifications of varicella zoster virus infection (unspecified) are laboratory confirmed cases that are positive for varicella zoster virus, that do not have the clinical diagnosis available to distinguish as chickenpox or shingles.
- In 2013 there were 9,927 cases of varicella zoster virus (unspecified) reported, which was an increase of 18% from 2012.

Figure 69: Notified cases of varicella zoster virus infection, 2013, by age group*



* Excluding New South Wales.

Figure 70: Proportion of notified cases of varicella zoster virus unspecified, chickenpox and shingles, 2013, by state or territory*



* Excluding New South Wales.

Epidemiological situation in 2013

In 2013, there were 9,927 cases of unspecified VZV infections reported. This represented a notification rate of 63 per 100,000 and an 18% increase in notifications compared with 2012 (n=8,437).

Geographic description

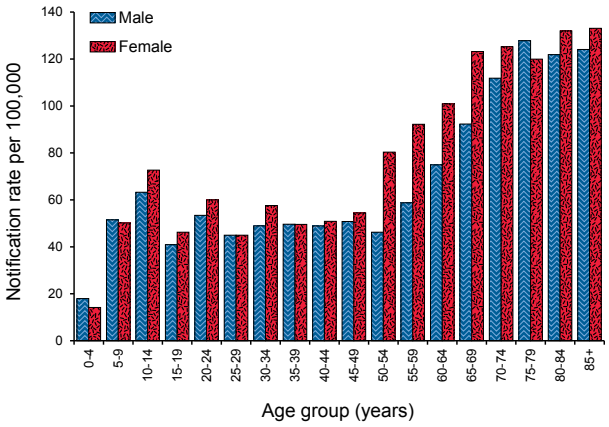
The highest notification rate for unspecified VZV was reported from Queensland at 115 per 100,000 (n=5,337) followed by Victoria at 53 per 100,000 (n=3,018) and Western Australia at 49 per 100,000 (n=1,230). VZV unspecified rates should be interpreted with caution as they are dependent on the individual jurisdictional practice of following up laboratory notifications to establish clinical presentation. For example, Queensland routinely conducts follow-up for cases of VZV in those

under 8 years of age, leading to a high proportion of VZV infections in older age groups classified as unspecified.

Age and sex distribution

The male to female ratio in the unspecified VZV notifications was 0.8:1. Females have an overall higher notification rate (69 cases per 100,000) compared with males (57 per 100,000), which predominates across the majority of age groups. The highest age group specific notification rates occurred in the 85 years or over age group for females, (133 per 100,000) and in the 75–79 years age group for males, (124 per 100,000). The lowest age group specific notification rates were in the 0–4 years age group for both males and females. These age distribution trends are likely reflect the practice of increased follow-up among younger age groups, especially in children aged less than 15 years, to determine clinical presentation (Figure 71).

Figure 71: Notification rate for varicella zoster virus unspecified, Australia,* 2013, by age group and sex



* Excluding New South Wales.

Chickenpox

- Chickenpox is normally a mild disease that occurs in childhood. However, in about 1% of cases complications can arise.
- The primary purpose of the vaccine is to prevent deaths, reduce the severity of disease and in the longer term reduce rates of VZV reactivation as shingles.
- In 2013, there were 2,042 cases of chickenpox reported, a 3% increase from 2012 (n=1,977).

Chickenpox is a highly contagious infection spread by respiratory secretions, including aerosol transmission, or from the vesicle fluid of skin lesions from a patient with chickenpox or shingles infection. Chickenpox is usually a mild disease of childhood; however, complications occur in approximately 1% of cases. It is more severe in adults and in persons of any age who are immunocompromised, in whom complications, disseminated disease, and fatal illness are more likely to occur.[50]

Epidemiological situation in 2013

In 2013, there were 2,042 cases of chickenpox reported. Representing a notification rate of 13 per 100,000 and a 3% increase in the number of notifications compared with 2012 (n=1,977). Rates of chickenpox have remained stable between 12 and 14 per 100,000 in all years since 2009.

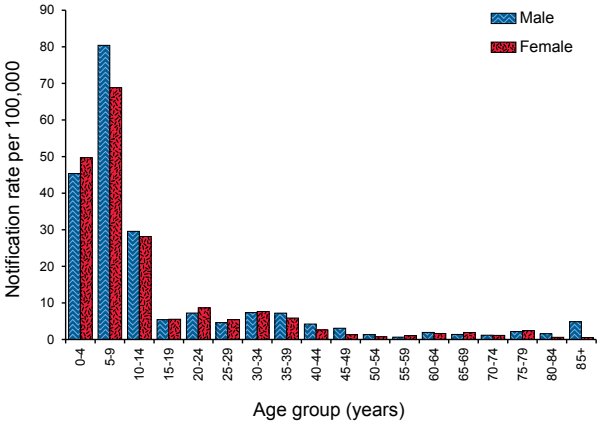
Geographic description

The highest notification rate, 40 per 100,000, was reported from the Northern Territory (n=97), followed by South Australia, 23 per 100,000 (n=386), reflecting the increased case ascertainment in these jurisdictions compared with others.

Age and sex distribution

The male to female ratio in 2013 was 1.1:1 with 1,065 notifications for males and 968 for females. Seventy-four per cent of notified chickenpox cases (n=1,501) occurred in children less than 10 years of age. The 5–9 years age group had the highest notification rate for both sexes, (80 per 100,000 for males and 69 per 100,000 for females) (Figure 72). Although higher rates among children compared with adults is expected for chickenpox, the distri-

Figure 72: Notification rate for chickenpox, Australia,* 2013, by age group and sex



* Excluding New South Wales.

bution of cases by age group also reflects general jurisdictional practice of limiting follow-up for cases in children less than 15 years of age.

Vaccination

Routine use of a varicella-containing vaccine in children was first recommended in Australia in 2003. In November 2005, the vaccine was funded under the NIP for all children at 18 months of age, with a school-based catch-up program for children 10–13 years of age with no history of disease or previous vaccination.

In 2013, the oldest cohort of children eligible for varicella vaccination at 18 months of age under the NIP would be 9 years of age. The analysis of vaccination status is restricted to this cohort. Vaccination status information was available for 50% (n=504) of cases that occurred in children less than 10 years of age who were eligible for vaccination with 76% (n=381) vaccinated and 24% not vaccinated (n=123). Post-marketing studies in the United States of America have estimated the effectiveness of 1 dose of monovalent varicella vaccine in children to be 80%–85% against any disease and 95%–98% against severe varicella.[86]

Shingles

- Herpes zoster or shingles is a sporadic disease caused by reactivation of latent varicella zoster virus following primary infection of chickenpox.
- In 2013, there were 5,017 cases of shingles reported, which was a 12% increase from 2012.

Shingles occurs most commonly with increasing age, impaired immunity, and a history of chickenpox in the 1st year of life.[50] Reactivation of VZV to cause shingles is thought to be due to a decline in cellular immunity to the virus. Shingles typically presents as a unilateral vesicular rash localised in a dermatomal distribution. Associated symptoms may include headache, photophobia, malaise, and itching, tingling, or severe pain in the affected dermatome. In the majority of patients, shingles is an acute and self-limiting disease however, complications develop in approximately 30% of cases, the most common of which is chronic severe neuropathic pain or post herpetic neuralgia.[21]

A single dose of zoster vaccine is recommended, but not presently funded through the NIP, for adults aged 60 years or over who have not previously received a dose of zoster vaccine.[50]

Epidemiological situation in 2013

In 2013, there were 5,017 cases of shingles reported to the NNDSS. This was a notification rate of 32 per 100,000 and an 11% increase compared with 2012 (n=4,507).

Geographic description

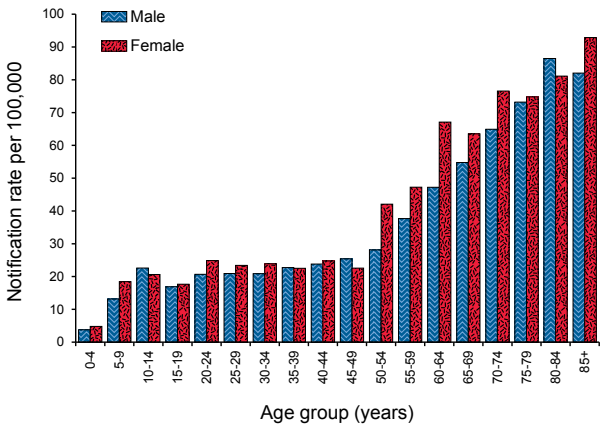
The highest rates of shingles occurred in South Australia with 114 per 100,000 (n=1,899), followed by the Northern Territory, 102 per 100,000 (n=246). The high rates in these jurisdictions most likely reflect their higher levels of case ascertainment compared with other jurisdictions.

Age and sex distribution

The notification rate was lower in males at 29 per 100,000 compared with females at 35 per 100,000, representing a ratio of 0.8:1.

As expected, rates increased with age with the highest in the 80–84 years age group for males, 87 per 100,000 and in the 85 years or over age group for females, 93 per 100,000 (Figure 73).

Figure 73: Notification rate for shingles, Australia,* 2013, by age group and sex



* Excluding New South Wales.

Discussion

Rates of chickenpox have remained relatively stable in all years since 2009. Noting that 2009 was the 1st year in which all jurisdictions, with the exception of New South Wales, reported cases to the NNDSS. An unpublished analysis of these data show an increase in shingles notifications in South Australia since 2006, which is consistent with an upward trend noted in the national data since 2009. This increase is likely to be due to multiple factors

including changes in health-care seeking behaviour, clinical practice, and awareness of reporting requirements as well as an ageing population.

Vectorborne diseases

Overview

Vectorborne diseases are infections transmitted by arthropods such as mosquitoes and ticks. A vectorborne disease may involve a simple transfer via the arthropod, or, may involve replication of the disease-causing organism in the vector.[21] Vectorborne diseases of public health importance in Australia listed in this chapter are: arbovirus not elsewhere classified (NEC); Barmah Forest virus (BFV) infection; dengue virus (DENV) infection; Japanese encephalitis virus (JEV) infection; Kunjin virus (KUNV) infection, malaria, Murray Valley encephalitis virus (MVEV) infection and Ross River virus (RRV) infection. Some vectorborne diseases, including yellow fever infection, plague and certain viral haemorrhagic fevers, are listed under quarantinable diseases. The National Arbovirus and Malaria Advisory Committee provide expert technical advice on vectorborne diseases to the Australian Health Protection Principal Committee through CDNA.

Alphaviruses

Viruses in the genus *Alphavirus* that are notifiable in Australia are BFV and RRV. These viruses are unique to the Australasian region.[87] Infection can cause a clinical illness, which is characterised by fever, rash and polyarthritis. The viruses are transmitted by numerous species of mosquito that breed in diverse environments.[88] The alphavirus chikungunya was not nationally notifiable in 2013, and thus not included in this annual report. However, it is notifiable in all states and territories except the Australian Capital Territory, and states and territories send information about cases to the Commonwealth for national collation and analysis.[89,90] Chikungunya virus infection was made nationally notifiable in January 2015.

The national case definitions for RRV and BFV require only a single IgM positive test to 1 virus, in the absence of IgM to the other.[18] False positive IgM diagnoses for BFV in particular are a known issue, thus it is unclear what proportion of notifications represent true cases.

Barmah Forest virus infection

- There was a dramatic increase in case numbers and rates thought to be due to an increase in false positive notifications.
- Females were disproportionately affected in 2013, and the most affected age groups were younger than in previous years.

Epidemiological situation in 2013

In 2013, there were 4,239 notifications of BFV infection, for a rate of 18.3 per 100,000 population. This compares with a 5-year mean of 1,723 notifications and a 5-year mean rate of 7.8 per 100,000. The number of notifications of Barmah Forest virus increased sharply from October 2012 (Figure 74). This increase continued into late 2013 and beyond for some jurisdictions. The increase was considered likely to have been due to a high rate of false positive IgM test results from the use of a commercial test kit in private laboratories, and resulted in a recall of the affected kits in September 2013.[91]

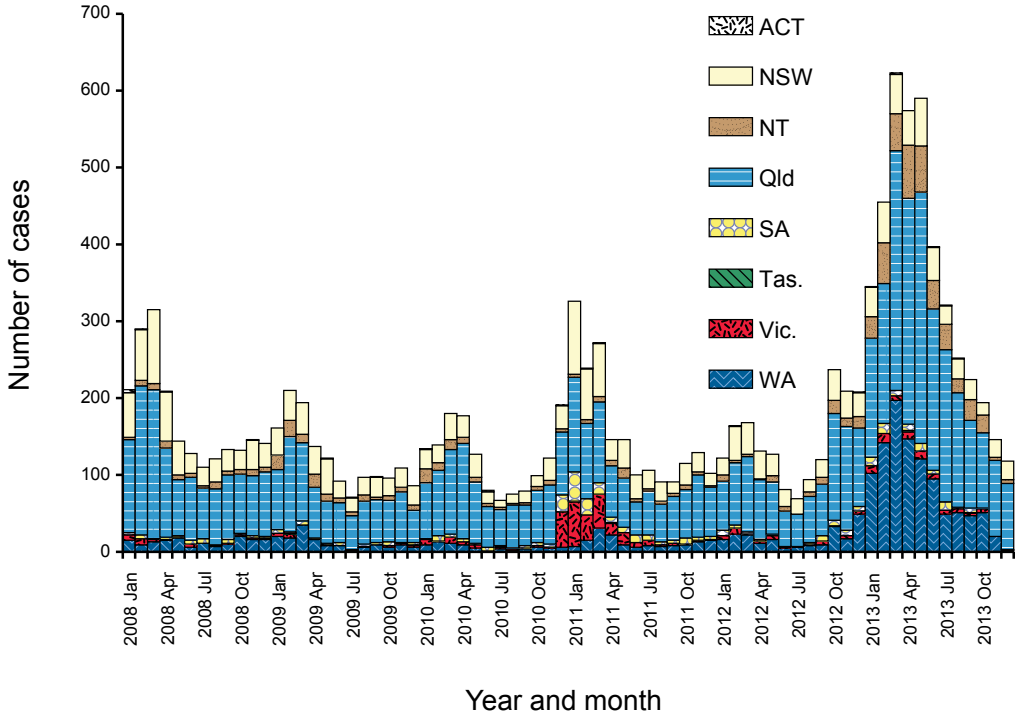
Geographic description

More than half of all BFV notifications in 2013 were from Queensland (52%, 2,224/4,239) and population rates were highest in the Northern Territory (167.9 per 100,000), Queensland (47.8 per 100,000) and Western Australia (40.6 per 100,000). All of these rates were more than double the 5-year mean, with rate ratios of 4.5, 2.2 and 6.4 respectively for 2013 compared with the 5-year mean rate. In New South Wales, South Australia and Victoria, rates were similar to the 5-year mean.

Age and sex distribution

In 2013, BFV infection was most frequently reported in people aged between 10 and 54 years (median 46 years, range 0–92 years), in contrast to previous years where the age groups most affected were middle aged and older adults. In 2013, age and sex specific rates were highest among females in the 35–54 years age group, and the next highest rate was among females aged 15–34 years (Figure 75). In 2013, rates were much higher in females overall than in males (21.9 and 14.7 per 100,000 respectively) with a rate ratio of 1.5:1. By contrast, between 2008 and 2012, rates in females were marginally lower than in males (7.6 and 8.0 per 100,000 respectively) with a rate ratio of 0.9:1.

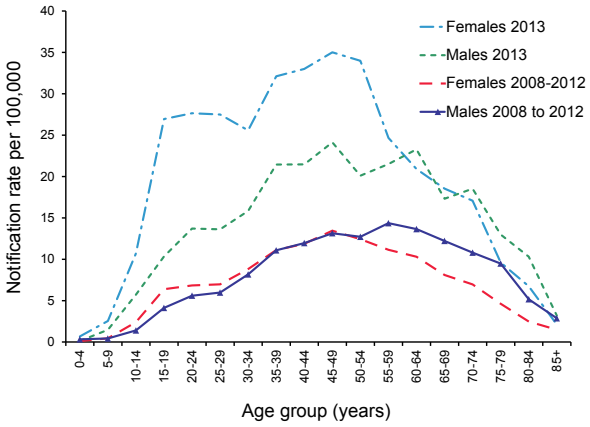
Figure 74: Notified cases of Barmah Forest virus infection, Australia, 2008 to 2013, by year and month and state or territory



Seasonality

Peak incidence of BFV could be expected to occur during the warmer months (or during wetter months in northern areas of Australia) when mosquito numbers are high. However, seasonality of notifications is less marked than expected (Figure 75), and a high proportion of inter-seasonal notifications are thought to be due to false positive diagnoses. Peak notification of BFV in 2013 was between January and April, with 47% (1,997/4,239) of notifications being during this period, similar to between 2008 and 2012 (46% ,3,925/8,616). The increase from October 2012 that was thought to be due to false positive notifications was earlier than the expected seasonal increase.

Figure 75: Notification rate for Barmah Forest virus, Australia, 2013 and 2008 to 2012, by age group and sex (n=12,852)



Discussion

The dramatic increase in counts and rates in 2013 disproportionately affected females, with much higher rates in females and in younger age groups than observed in previous years. The CDNA surveillance case definition for BFV[92] in 2013 allowed for confirmation based on a single positive IgM, in the absence of IgM to other alphaviruses. Not all jurisdictions reported increases, and this may in part be due to differences in laboratory and notification practices. South Australia requires seroconversion to BFV, and in Victoria, metropolitan cases without any travel to non-metropolitan areas require evidence of seroconversion.

Given the dramatic increase in notifications in late 2012 and 2013, the possibility of false positive diagnoses based on a single positive IgM, and also the difference in surveillance and notification practices, CDNA has referred the BFV surveillance case definition to the CDWG for review.

Ross River virus infection

- Notifications were similar to the 5-year mean.

Epidemiological situation in 2013

In 2013, there were 4,308 notifications of RRV, which was a rate of 18.6 per 100,000. This compares with a 5-year mean of 5,061 cases and a 5-year mean rate of 23.0 per 100,000.

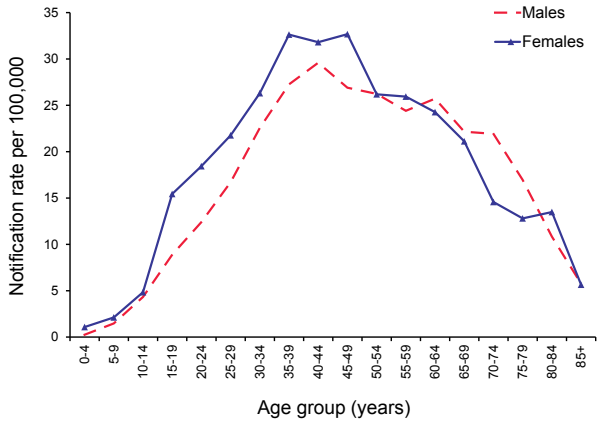
Geographic description

In 2013, nearly half of all RRV infections were from Queensland (41% of all cases, 1,787/4,308, for a rate of 38.4 cases per 100,000), but population rates were highest in the Northern Territory (124.4 per 100,000) and Western Australia (54.3 per 100,000).

Age and sex distribution

RRV was most frequently reported in adults aged in their 30s or 40s (median 44 years, range 0–95 years), similar to previous years. Rates were similar in females and males (rates of 19.7 and 17.5 per 100,000 respectively) with a ratio of 1.1:1, similar to previous years. In 2013, age specific rates were highest among the 35–49 year age range for females, and the 35–44 year age range for males (Figure 76).

Figure 76: Notification rates for Ross River virus, 2013, by age group and sex (n=4,308)



Seasonality

Peak notification for RRV in 2013 was between January and April, and 44% of cases were diagnosed during these months (Figure 77). Between 2008 and 2012, 58% of notifications were between January and April, indicating that in 2013, the proportion of inter-seasonal notifications was higher than in previous years.

Flaviviruses

In Australia, flavivirus infections of particular public health importance are DENV, KUNV, MVEV and JEV. Yellow fever is reported under Quarantinable diseases. These infections are nationally notifiable. No specific treatment is available for these diseases and care is largely supportive. A vaccine is available to prevent JEV infection[50] but there are no vaccines currently for DENV, MVEV or KUNV infection.

Infection with MVEV, KUNV or JEV is usually asymptomatic or produces a non-specific illness, but a small percentage of cases progress to encephalomyelitis of variable severity. *Culex annulirostris* is the major vector of MVEV, JEV and KUNV. DENV has 4 serotypes, each containing numerous genotypes. The serotypes isolated from returning travellers (and thus involved in local outbreaks) vary by year and geographical region. Infection with 1 serotype probably confers lifelong immunity to that serotype,[21] but subsequent infection with a different serotype is 1 factor thought to increase the risk of severe outcomes, along with the infecting serotype and genotype, and host factors.[21,93–95] The clinical illness is characterised by mild to severe febrile illness with fever, headache, muscle and joint pain and sometimes a rash. A minority of cases progress to severe dengue with haemorrhage and shock. *Aedes aegypti* is the major vector of DENV in Australia.

Arbovirus NEC

- 21 cases of arbovirus (NEC) were notified in 2013.

Unspecified flavivirus infections are reported under arbovirus NEC. From 2015, arbovirus NEC has been renamed flavivirus NEC.

Epidemiological situation in 2013

In 2013, there were 21 notifications of arbovirus (NEC) compared with an average of 11.8 during the previous 5 years. All but one of these notifications was from Queensland. These notifications comprised Alfuy (1 case), Kokobera (5 cases) and Zika (1 case), and the infecting flavivirus was unknown or not supplied for a further 14 cases (Table 16).

Figure 77: Notified cases of Ross River virus, Australia, 2008 to 2013, by year and month and state or territory

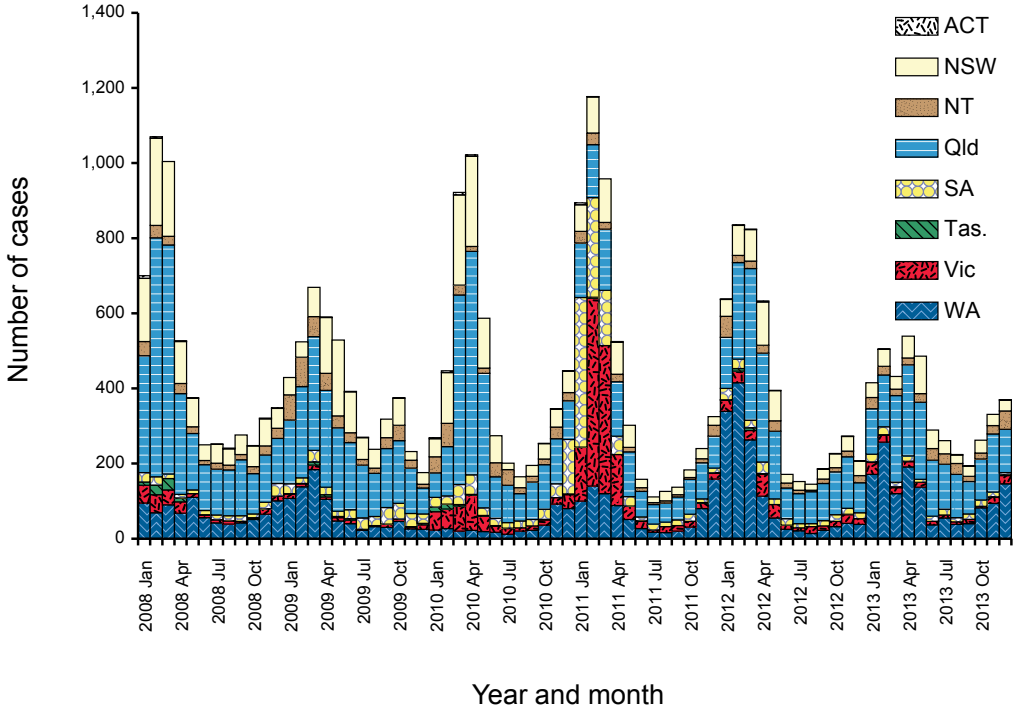


Table 16: Notified cases of arbovirus NEC, Australia, 2013

State or territory	Country of acquisition	Organism	Age group	Sex
Qld	Unknown	Kokobera	20–24	Female
Qld	Unknown	Kokobera	35–39	Female
Qld	Unknown	Kokobera	55–59	Male
Qld	Unknown	Kokobera	75–79	Male
Qld	Unknown	Untyped	25–29	Male
Qld	Unknown	Untyped	50–54	Female
Qld	Unknown	Untyped	55–59	Female
Qld	Unknown	Untyped	60–64	Male
Qld	Australia	Kokobera	15–19	Female
Qld	Papua New Guinea	Untyped	20–24	Female
Qld	Papua New Guinea	Untyped	35–39	Male
Qld	Vanuatu	Alfuy	35–39	Female
Qld	Democratic Republic of Korea (North Korea)	Untyped	45–49	Male
Qld	Democratic Republic of Korea (North Korea)	Untyped	50–54	Male
Qld	Republic of Korea (South Korea)	Untyped	30–34	Male
Qld	Indonesia	Untyped	20–24	Male
Qld	Indonesia	Untyped	45–49	Male
Qld	Indonesia	Untyped	50–54	Male
Qld	Philippines	Untyped	40–44	Female
Qld	India	Untyped	45–49	Female
NT	Indonesia	Zika	25–29	Male

Information about the country of acquisition was available for 62% of cases (13/21), and 12 of these were acquired overseas.

The median age of cases was 43 years (range 19–76 years). Nine cases were female and 12 cases were male.

Dengue virus infection

- There was a continuing increase in the number of overseas acquired cases.
- 235 cases were acquired in Australia in 2013, including acquired in Western Australia.

Local transmission of dengue in Australia is normally restricted to areas of northern Queensland where the key mosquito vector, *Ae. aegypti* is present.[96] Dengue is not endemic in North Queensland, but local transmission can occur upon introduction of the virus to the mosquito vector by a viraemic tourist or a resident returning from a dengue-affected area overseas.[97]

The CDNA case definition for dengue was changed in 2013 to accept dengue non-structural protein 1 (NS1) antigen in blood as laboratory definitive evidence for infection and a number of states and territories had been sending notifications based on a positive NS1 antigen prior to this change.

Epidemiological situation in 2013

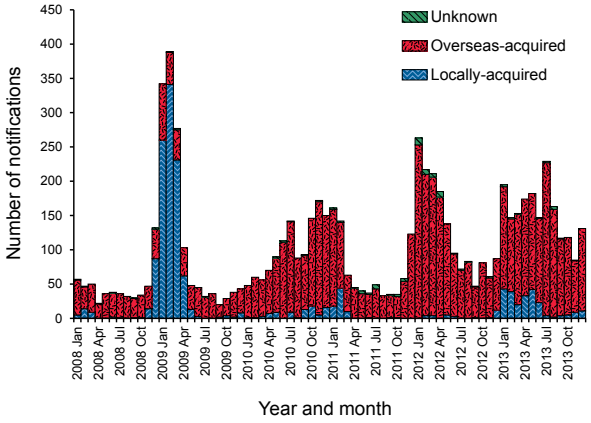
There were 1,841 notifications of dengue in 2013, which was 1.7 times the 5-year mean of 1,110.4 notifications. Most infections were acquired overseas (n=1,591) (Figure 78). There were 235 infections acquired in Australia. For 15 cases, no information was supplied on the place of acquisition.

Geographic description

More than 99% (1,826/1,841) of notifications in 2013 contained complete information on the place of acquisition. Overseas acquired infections comprised 86% of notifications (1,591/1,841) (Table 17). The number of overseas-acquired infections was the largest number ever reported, up from 1,473 in 2012, which was previously the largest number ever reported.[76] Between 2007 and 2010, the number of DENV cases known to have been acquired overseas increased each year, from 254 in 2007 to 1,137 in 2010 (Figure 78).

Cases acquired in Indonesia continue to account for the largest number and proportion of all notifications, accounting for 50% (800/1,591) of

Figure 78: Notified cases of dengue virus infection, Australia, 2008 to 2013, by year and month and place of acquisition



all overseas-acquired cases in 2013 (Table 18), up from an average of 30% per year in 2008 and 2009, but down from an average of 60% between 2010 and 2012. DENV acquired in Indonesia was frequently serotype 1, comprising 50% of cases with a known serotype (72/143 cases), although data completeness for serotype was very low. Other frequently reported source countries in 2013 included Thailand, the Philippines, India and Malaysia.

All but 13 of the 235 locally-acquired cases in 2013 were reported in NNDSS to have been associated with one of the 10 outbreaks of locally-acquired infection in Queensland in 2013.[98] The largest of these outbreaks was in Cairns and began in late 2012, with 141 associated notifications in 2013, the last case of them with onset in July 2013. One case in the Pilbara Region of Western Australia was locally acquired from an unknown source,[99] and another case in Western Australia, while notified as locally acquired in the data on which this report is based, should have been listed as overseas acquired.

Age and sex distribution

DENV infections acquired overseas in 2013 were most commonly reported among younger and middle aged adults (median 39 years, range 1–86 years), with a slight peak of notifications among females aged 25–29 years and males aged 50–54 years, but with similar numbers notified in all age groups between 20 and 54 years (Figure 79). Females comprised 50% (793/1,541) of overseas acquired cases.

Locally-acquired cases peaked in several adult age groups, but was less common among people aged less than 20 years or more than 74 years

Table 17: Notified cases of dengue virus infection, 2013, by serotype and place of acquisition

Country of acquisition	Serotype				Untyped	Total
	DENV 1	DENV 2	DENV 3	DENV 4		
Locally-acquired						
Australia	175	4	11	0	45	235
Unknown						
Not stated	1	0	1	0	13	15
Overseas-acquired						
Indonesia	72	36	28	7	657	800
Thailand	35	12	14	1	206	268
Philippines	9	5	1	4	44	63
India	3	5	2	0	48	58
Malaysia	3	4	0	3	43	53
East Timor	10	0	9	0	29	48
Papua New Guinea	5	6	2	0	22	35
Cambodia	4	0	4	1	22	31
South-East Asia, nfd	2	1	1	0	24	28
Sri Lanka	6	0	0	0	22	28
Vietnam	1	1	1	3	20	26
Singapore	3	3	1	1	10	18
Bangladesh	0	1	0	0	15	16
Solomon Islands	0	0	6	0	9	15
Fiji	3	2	1	0	8	14
Burma (Myanmar)	3	0	0	0	8	11
Other countries	12	3	2	4	55	76
Overseas – country unknown	1	0	0	0	2	3
Total for overseas acquired	172	79	72	24	1,244	1,591
Total	348	83	84	24	1,302	1,841

nfd Not further defined.

Table 18: Notifications of dengue virus infection acquired overseas between 2008 and 2013, by selected countries of acquisition

Country of acquisition	2008	2009	2010	2011	2012	2013	Total
Indonesia	101	169	715	458	803	800	3,046
Thailand	55	24	124	85	278	268	834
India	8	15	43	29	60	58	213
The Philippines	7	9	42	23	54	63	198
East Timor	11	24	37	12	52	48	184
Malaysia	9	15	17	20	20	53	134
Vietnam	8	18	34	14	21	26	121
Papua New Guinea	13	11	21	15	16	35	111
Cambodia		5	11	5	30	31	82
Fiji	13	8	1	6	32	14	74
Sri Lanka	3		4	12	26	28	73
Total	420	472	1,137	721	1,473	1,591	5,814

(Figure 80). The median age of locally-acquired cases was 41 years (range 1 to 86 years). Females comprised 49% (116/235) of locally-acquired cases.

Figure 79: Notified cases of overseas-acquired dengue virus infection, Australia, 2013, by age group and sex (n=1,591)

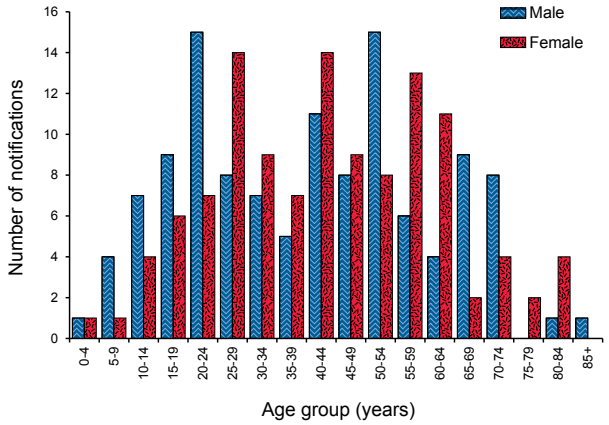
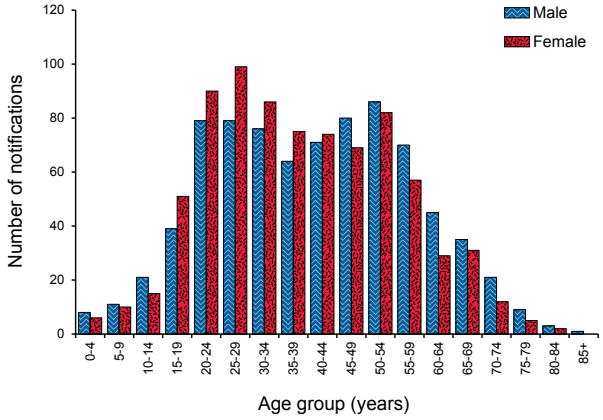


Figure 80: Notified cases of dengue virus infection acquired in Australia, 2013, by age group and sex (n=235)



Seasonality

No particular pattern of seasonality was evident for overseas acquired cases of dengue, although the largest numbers were reported in July. For locally-acquired cases, only 15 cases were reported between July and October demonstrating that outbreaks are not continuing through the cooler months.

Microbiological trends

In 2013, serotype information was available for 29% of notifications (539/1,841), which was a decrease compared with the 5-year mean of 43% (Table 19). In 2013, 65% (348/539) of cases with a known serotype were due to DENV 1 in contrast to 2012, when DENV2 was more frequently reported, noting the low completeness of reporting serotype information (Table 19).

Discussion

The number of overseas-acquired cases reported in Australia continues to increase each year. In recent years, improved diagnostic techniques, in particular the availability of the rapid NS1 antigen detection kit, have improved detection and would have contributed to the observed increase in reported numbers of overseas-acquired dengue in Australia,[100] along with the dramatic re-emergence and geographical expansion of dengue overseas over the past 50 years, combined with explosive outbreaks.[95]

While local outbreaks of dengue occur each year in North Queensland, each outbreak is relatively small, and prompt and effective responses by public health authorities in Queensland have ensured that the disease does not become endemic there.

The number of dengue infections that are serotyped continues to decline. The decreased reporting of a serotype may reflect the increasing use of NS1 antigen detection and/or other diagnostic methods that do not provide a serotype.

Japanese encephalitis virus infection

- Four cases of JEV were notified in 2013.

Epidemiological situation in 2013

There were 4 notifications of JEV infection in 2013. One of these notifications (a notification from Western Australia) was subsequently found not to meet the case definition. The 3 remaining cases were acquired in Thailand, Taiwan and the Philippines. There was 1 notification in 2012, and 1 notification in 2008, both acquired overseas. The last locally-acquired case was in 1998.[101]

Kunjin virus infection

- Three cases of KUNV were notified in 2013.

Epidemiological situation in 2013

There were 3 notifications of KUNV infection in 2013, one each acquired in East Timor, Indonesia and Papua New Guinea. There were no notifications of KUNV infection in 2012.

Table 19: Serotype of dengue virus infection, Australia, 2008 to 2013

Serotype	2008	2009	2010	2011	2012	2013
DENV1	40	82	190	139	81	348
DENV 1 and DENV 4	0	0	0	0	1	0
DENV 2	32	54	255	153	137	83
DENV 3	143	771	106	78	57	84
DENV 4	37	43	47	43	8	24
Untyped/unknown	309	452	630	408	1,256	1,302
Total	561	1,402	1,228	821	1,540	1,841
% with a serotype supplied	45	68	49	50	18	29

Murray Valley encephalitis infection

- One case of MVEV was notified in 2013.
- MVEV is a rare disease in Australia, but also acquired in the region.

Epidemiological situation in 2013

There was 1 notification of MVEV infection in 2013, acquired in Indonesia.

There was 1 case in 2012, 16 cases in 2011, 2 cases in 2008 and 4 cases in 2009. The cases notified in 2011, including an outbreak in south east Australia, have been described elsewhere.[89,102–104]

Malaria

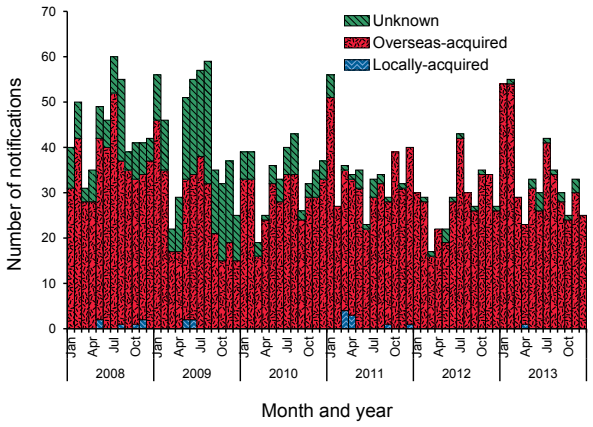
- Notifications continued the gradual decline observed since 2005.
- One case was known to have been acquired in Australia in 2013.

Malaria is caused by a protozoan parasite in the genus *Plasmodium*, and 5 species are known to infect humans; *Plasmodium vivax, Plasmodium falciparum, Plasmodium malariae, Plasmodium ovale* and *Plasmodium knowlesi*.[21,105] Malaria is a serious acute febrile illness that is transmitted from person to person via the bite of an infected mosquito of the genus *Anopheles*. Australia was declared free of malaria in 1981,[106] but suitable vectors are present in northern Australia, and the area remains malaria-receptive. Malaria is the most frequently reported cause of fever in returned travellers worldwide.[107] A case series in the Northern Territory showed that malaria cases were reported in travellers returning from endemic areas, but also reflected current events such as military operations and increased refugee arrivals from malaria endemic areas.[108] Malaria cases in Australia can be found either through testing of symptomatic persons with a compatible travel history, or through screening of refugees who may be asymptomatic.

Epidemiological situation in 2013

There were 414 cases of malaria notified in Australia in 2013, a 6% decrease compared with a 5-year mean of 440 cases, and continuing the trend of gradually decreasing notifications since 2005 (Figure 81). The largest number of cases was reported by Queensland (108 cases).

Figure 81: Notified cases of malaria, Australia, 2008 to 2013, by month and year and place of acquisition



Geographic description

Malaria in Australia is a disease associated with overseas travel or residence in areas with endemic transmission. The last cases acquired on mainland Australia were during an outbreak in North Queensland in 2002.[109] Limited transmission occurs occasionally in the Torres Strait.

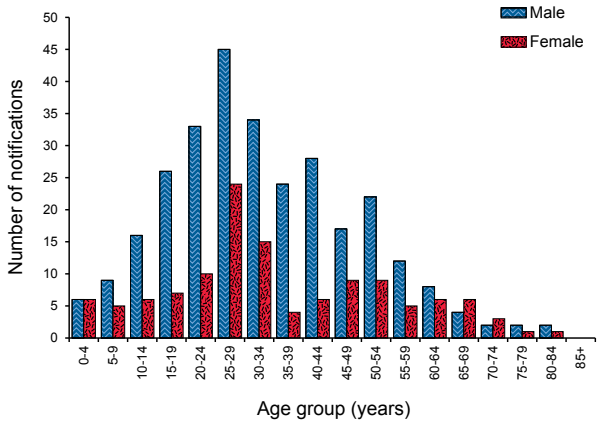
The place of acquisition for malaria notifications in 2013 was listed as overseas for 398 cases. For 16 cases, no place of acquisition information was supplied to NNDSS. One case in 2013 was listed as locally-acquired on Saibai Island in the Torres Strait. Prior to this case, the last known locally-acquired infections were during the 2011 outbreak in the Torres Strait.[110]

Complete information on the country or region of acquisition was supplied for all but eight of the cases known to have been acquired overseas, and these remaining cases were notified as being overseas acquired, country unknown or not stated. The most frequent countries of acquisition were Papua New Guinea (16% of cases with complete information) and India (16%) (Table 20). Most cases acquired in Papua New Guinea were reported by Queensland (31 cases).

Age and sex distribution

In 2013, sex was stated for all cases while age was supplied for all but 1 case. Malaria was most commonly reported in males (70%, 290/414 cases) with a peak of notifications in males aged 25 to 29 years (Figure 82). The median age of cases was 30 years (range 0–83 years).

Figure 82: Notified cases of malaria, Australia, 2013, by age group and sex (n=413)*



* Age was not reported for 1 case.

Seasonality

Increases in notifications or an observable pattern of seasonality in a predominantly overseas-acquired infection can relate to the seasonality of travel patterns, or to local disease epidemiology in the source countries. In 2013, there was apparent increase in notifications in January and February compared with other months (54 and 55 notifications respectively, compared with an average of 30.5 notifications for the other months).

Microbiological trends

The infecting species was supplied for 98% (404/414) of cases in 2012 (Table 20). The most frequent infecting species was *P. falciparum* (reported in 55% of cases with complete information). *P. vivax* was associated with Asia and the Pacific, whilst most cases acquired in African countries were *P. falciparum*. In cases acquired in Indonesia and Papua New Guinea however, *P. falciparum* and *P. vivax* infections were reported in similar numbers.

Zoonoses

Overview

Zoonoses are those diseases and infections that are naturally transmitted between vertebrate animals and humans.[111] Approximately 60%–70% of emerging human infectious diseases are zoonoses[112–114] and more than 70% of emerging zoonoses originate from wildlife.[113] An emerging zoonosis is defined by WHO as "a zoonosis that is newly recognised or newly evolved, or that has occurred previously but shows an increase in incidence or expansion in geographical, host or vector range".[115]

The zoonoses notifiable to the NNDSS included in this chapter are: anthrax, Australian bat lyssavirus (ABLV) or lyssavirus (unspecified) infection, brucellosis, leptospirosis, ornithosis, Q fever, and tularaemia.

Several zoonoses notifiable to the NNDSS are included under other headings in this report. For example, *Salmonella* and *Campylobacter* infections are typically acquired from contaminated food and are listed under the gastrointestinal diseases section. Rabies is listed under Quarantinable diseases.

Anthrax

- No cases of anthrax were notified in 2013.

Anthrax is caused by the bacterium *Bacillus anthracis* and most frequently causes cutaneous infection. However, it can also cause gastrointestinal and respiratory infections. Anthrax is primarily a disease of herbivores; humans and carnivores are incidental hosts. It can be an occupational hazard for veterinarians, and agriculture, wildlife and livestock workers who handle infected animals or by-products. [21]

Table 20: Notified cases of malaria, Australia 2013, by infecting species and region and country of acquisition

Region and country	*P. falciparum*	*P. malariae*	*P. ovale*	*P. vivax*	Mixed species infection	*Plasmodium* species	Total
Oceania							
Australia	0	0	0	1	0	0	1
Papua New Guinea	23	1	0	35	0	1	60
Solomon Islands	0	0	0	7	0	0	7
Vanuatu	0	0	0	3	0	0	3
Fiji	1	0	0	0	0	0	1
South East Asia							
South-East Asia, NFD	0	0	0	1	0	0	1
Mainland South-East Asia, NFD	0	0	0	1	0	0	1
Burma (Myanmar)	0	0	0	2	0	0	2
Cambodia	1	0	0	3	0	0	4
Thailand	2	0	0	0	0	0	2
Vietnam	1	0	0	1	0	0	2
Brunei Darussalam	0	1	0	0	0	0	1
Indonesia	5	1	2	9	1	0	18
Malaysia	0	0	1	0	0	1	2
East Timor	0	0	0	0	1	0	1
North East Asia							
Korea, Republic of (South)	0	0	0	1	0	0	1
Southern and Central Asia							
India	2	0	0	60	1	2	65
Nepal	0	0	0	1	0	0	1
Pakistan	0	0	0	6	0	0	6
Africa							
Africa NFD	1	0	0	0	0	0	1
North Africa and the Middle East							
North Africa, NFD	2	0	0	0	0	0	2
Sudan	47	1	4	1	1	1	55
Western Sahara	1	0	0	1	0	0	2
Sub-saharan Africa							
Sub-Saharan Africa, NFD	12	1	0	0	0	0	13
Burkina Faso	1	0	0	0	0	1	2
Cameroon	1	0	0	0	0	0	1
Congo, Democratic Republic of	2	1	0	0	0	0	3
Cote d'Ivoire	3	0	0	0	0	0	3
Equatorial Guinea	1	0	2	0	0	0	3
Gabon	1	0	0	0	0	1	2
Ghana	14	0	0	0	0	0	14
Guinea	1	0	0	0	0	0	1
Liberia	5	0	1	0	0	0	6
Mali	4	0	1	1	0	0	6
Nigeria	9	0	3	0	0	1	13
Sierra Leone	10	0	0	0	0	1	11
Togo	1	0	0	0	0	0	1

Table 20 (cont'd): Notified cases of malaria, Australia 2013, by infecting species and region and country of acquisition

Region and country	*P. falciparum*	*P. malariae*	*P. ovale*	*P. vivax*	Mixed species infection	*Plasmodium* species	Total
Southern and East Africa							
Southern and East Africa, NFD	1	0	1	0	0	0	2
Burundi	3	0	0	0	0	0	3
Ethiopia	1	0	0	2	0	0	3
Kenya	15	1	1	0	1	0	18
Malawi	0	0	0	0	1	0	1
Mozambique	2	0	1	0	0	0	3
South Africa	2	0	0	0	0	0	2
Tanzania	10	1	1	0	0	0	12
Uganda	20	0	0	1	2	0	23
Zambia	3	0	0	0	0	0	3
Zimbabwe	1	0	0	0	0	0	1
Europe							
South Eastern Europe NFD	0	1	0	0	0	0	1
Americas							
Peru	0	0	0	1	0	0	1
Overseas acquired – country and region not stated/unknown							
Unknown country	5	0	1	0	0	1	7
Overseas-acquired total	214	9	19	138	8	10	398
Place of acquisition unknown	7	1	0	6	2	0	16
Total	221	10	19	144	10	10	414

NFD Not further defined.

In Australia, the areas of anthrax risk are well defined and include the northern and north-eastern districts of Victoria and central New South Wales.[116] Anthrax occurs only sporadically in livestock in the at-risk areas. Rare or isolated incidents or cases have historically occurred in Queensland, South Australia, Tasmania and Western Australia.[116]

Epidemiological situation in 2013

In 2013 there were no notified cases of anthrax in Australia. Over the previous 10 years, only 3 human cases of anthrax were reported in Australia; in 2006, 2007 and 2010.[117–119] All had domestic farm or animal related exposures and all were cutaneous anthrax. Australia has never recorded a human case of inhalational or gastrointestinal anthrax.

There were 2 anthrax incidents reported in livestock in Australia in 2013. All properties were located within the known New South Wales anthrax endemic area.[116]

Australian bat lyssavirus and lyssavirus (unspecified)

- 1 case of ABLV was notified in 2013.

ABLV belongs to the genus lyssavirus, which also includes the rabies virus. Both invariably result in progressive, fatal encephalomyelitis in humans.[120] ABLV was first identified in Australia in 1996.[121,122] and is present in some Australian bats and flying foxes. Australia is free of terrestrial rabies.

The best way to prevent ABLV infection is to avoid contact with bats. For people whose occupation (including volunteer work) or recreational activities place them at increased risk of being exposed to ABLV, rabies virus vaccine is effective in preventing infection. Pre-exposure vaccination with rabies virus vaccine is recommended for bat handlers, veterinarians and laboratory personnel working with live lyssaviruses.[123] Post-exposure prophylaxis for ABLV consists of wound care and administration of a combination of rabies virus

vaccine and human rabies virus immunoglobulin, depending on exposure category and prior vaccination or antibody status.[50,123]

Epidemiological situation in 2013

In 2013, there was 1 notified case of ABLV. The case was an 8-year-old boy in Queensland.[124] Also in 2013, the Queensland Department of Agriculture, Fisheries and Forestry confirmed the first known equine cases of ABLV infection in 2 horses on a Queensland property.[125,126] There were no cases of lyssavirus (unspecified) infection in Australia. There were no cases of rabies in 2013.

There have been 3 human cases of ABLV infection in humans in Australia, in 1996, 1998 and 2013. All cases occurred after close contact with an infected bat and all were fatal.[124,127,128]

The bat health focus group in Wildlife Health Australia (formerly the Australian Wildlife Health Network) gathers and collates information from a range of organisations on opportunistic testing of bats for ABLV. In 2013, there were 14 ABLV detections in bats compared with 5 detections during 2012.[129]

Brucellosis

- 14 cases of brucellosis were notified in 2013.

Brucellosis is characterised by a fever of variable duration with symptoms including headache, weakness, profuse sweating, chills, arthralgia, depression, weight loss and generalised aching.[21] *Brucella* species that can cause illness in humans include *Brucella melitensis* acquired from sheep and goats, *Brucella suis* from pigs and *Brucella abortus* from cattle. *B. abortus* was eradicated from Australian cattle herds in 1989 and *B. melitensis* has never been reported in Australian sheep or goats.[130] Therefore, all cases of *B. melitensis* or *B. abortus* in Australia are related to overseas travel. *B. suis* is confined to some areas of Queensland, where it occurs in feral pigs. Eales et al (2010)[131] found that feral pig hunting was the most common risk factor for brucellosis in Townsville during 1996 to 2009.

Internationally, brucellosis is mainly an occupational disease of farm workers, veterinarians, and abattoir workers who work with infected animals or their tissues.[21]

Epidemiological situation in 2013

In 2013, there were 14 notified cases of brucellosis in Australia (a rate of 0.1 cases per 100,000), compared with the 5-year mean of 33.4 cases (2008 to 2012). Seventy-nine per cent of cases (n=11) were from Queensland (Figure 83), with a rate of 0.2 cases per 100,000. Since 1991, 83% of cases have been from Queensland.

The species of the infecting organism was available for 57% of cases (n=8). Of these, 5 cases were *B. suis*; four from Queensland and one from New South Wales. All were males aged between 22 and 46 years. There were 3 cases of *B. melitensis*, with the countries of acquisition listed as Iraq, Iran and Afghanistan. All other cases of brucellosis acquired the organism in Australia.

Age and sex distribution

The median age of cases of brucellosis was 30 years (range 20–67 years) and 79% of cases (n=11) were male.

Leptospirosis

- 95 cases of leptospirosis were notified in 2013.

Leptospirosis can cause a variety of illnesses varying in severity from a mild influenza-like illness to Weil's syndrome, meningitis or pulmonary haemorrhage with respiratory failure possibly leading to death.[21] Leptospirosis is caused by spirochaetes of the genus *Leptospira,* which is found in the genital tract and renal tubules of domestic and wild animals. In affected areas where there is exposure to infected urine of domestic and wild animals, this disease can be an occupational and recreational hazard (such as in certain agricultural sectors and swimming or wading in contaminated water.[132,133] The last reported death in Australia attributed to leptospirosis was in 2002.[134]

Epidemiological situation in 2013

In 2013 there were 95 notified cases of leptospirosis in Australia (a rate of 0.4 cases per 100,000), compared with the 5-year mean of 143 cases (2008 to 2012). In 2013, Queensland accounted for 71% (n=67) of cases (Figure 84).

Age and sex distribution

The median age of leptospirosis cases was 42 years (range 15–80 years) and 76% of cases (n=72) were male. The highest notification rate was observed in males aged 35–39 years (1.3 cases per 100,000 male population).

Microbiological trends

The WHO Food and Agriculture Organization World Organisation for Animal Health

Figure 83: Notified cases of brucellosis, Australia, 2008 to 2013, by month and year of diagnosis and state or territory

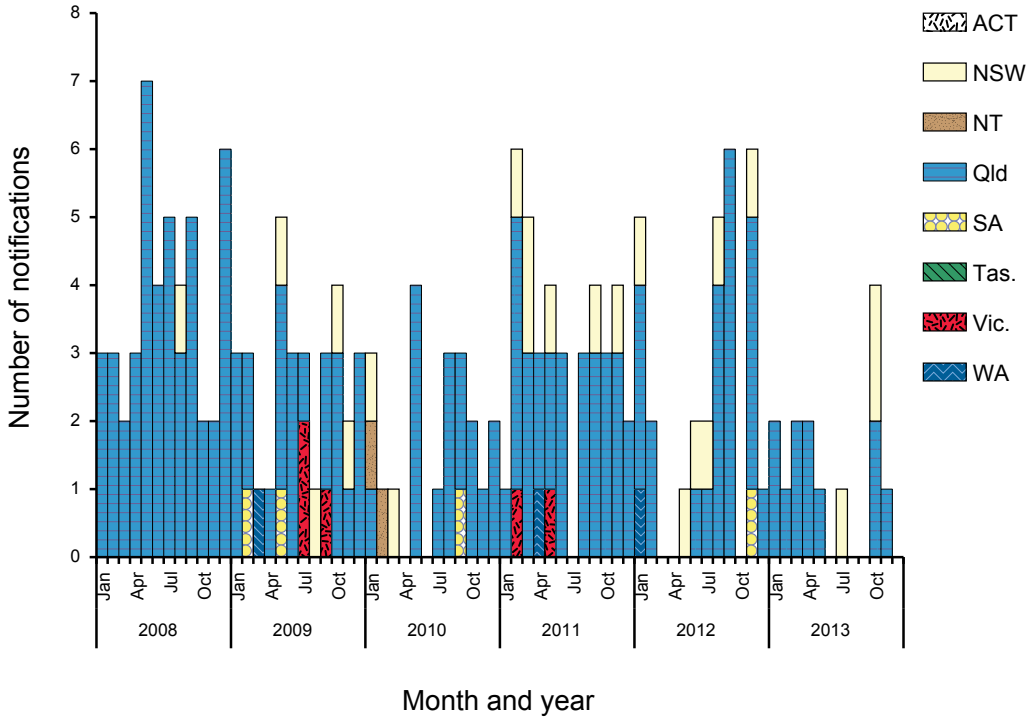
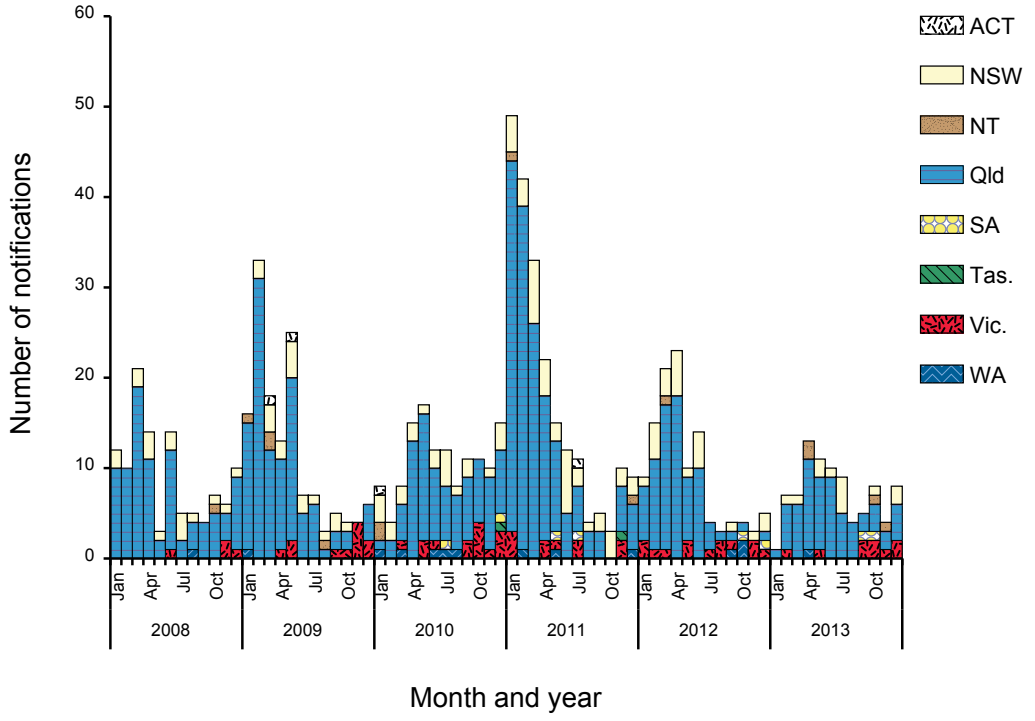


Figure 84: Notified cases of leptospirosis, Australia, 2008 to 2013, by month and year of diagnosis and state or territory



Collaborating Centre for Reference and Research on Leptospirosis routinely conducts PCR-based serotyping for leptospirosis cases from Queensland (from whence the majority of cases are reported), and collates national data that may be submitted to the laboratory from other states or territories. At the time of compiling this report, data for 2013 were not publicly available.

In Australia, serotyping is only conducted on pathogenic *Leptospira* species of which typing information was available for 89% (76/85). The most frequently reported serovars were *L. interrogans* serovar Australis (20%, n=17), *L. borgpetersenii* serovar Arborea (15%, n=13), *L. interrogans* serovar Hardjo (15%, n=13) and *L. interrogans* serovar Zanoni (15%, n=13). In 2012, *L. borgpetersenii* serovar Arborea was the most frequently reported serovar (22/103).

Ornithosis

- 47 cases of ornithosis were notified in 2013.
- The majority of notified cases in 2013 were from Victoria.

Ornithosis (or psittacosis) is a pneumonia-like illness caused by infection with the bacterium *Chlamydophila psittaci*.[21] It is transmitted to humans primarily from infected parrots of many species, but also poultry and a range of other birds.[135] Transmission to humans can occur via the inhalation of contaminated dried faeces, nasal or eye secretions and dust from the feathers. Individuals at risk of contracting ornithosis include bird owners and those with occupational exposure to birds.[136]

Epidemiological situation in 2013

In 2013 there were 47 notified cases of ornithosis in Australia (a rate of 0.2 cases per 100,000), compared with the 5-year mean of 79 cases (2008 to 2012, Figure 85). Similar to previous years, the majority of cases in 2013 were from Victoria (72%, n=34).

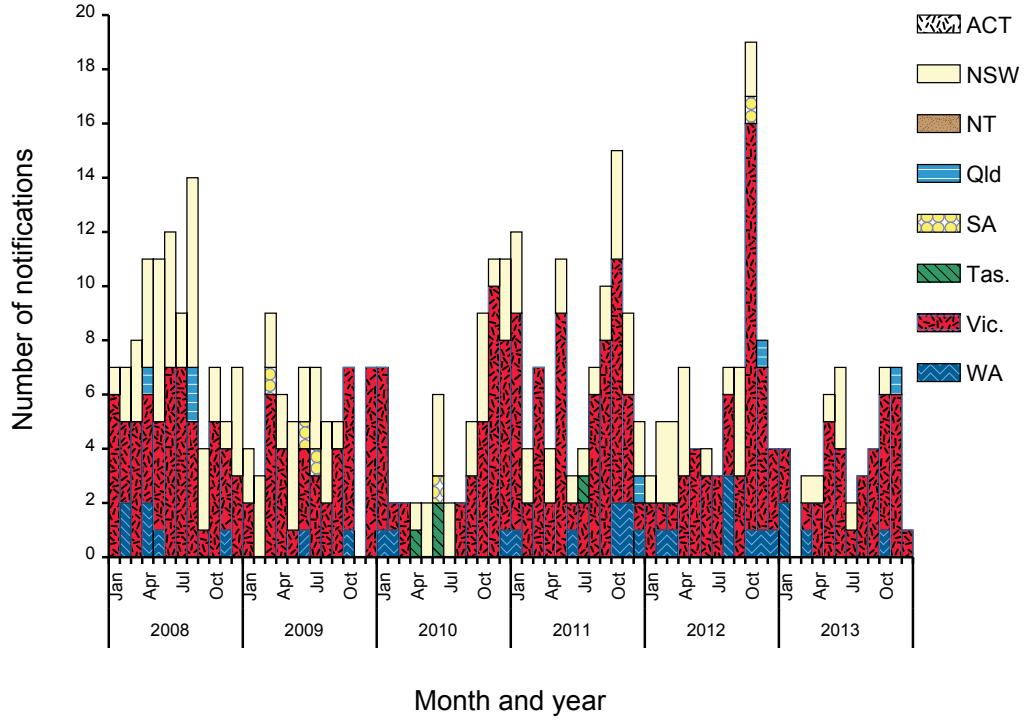
Age and sex distribution

The median age of ornithosis cases was 53 years (range 10–91 years) and 53% (n=25) of cases were female.

Q fever

- 477 cases of Q fever were notified in 2013.
- 78% of cases were male and the highest notification rate was observed in males aged 55–64 years (6.2 cases per 100,000).

Figure 85: Notified cases of ornithosis, Australia, 2008 to 2013, by month and year of diagnosis and state or territory



Q fever is caused by infection with the bacterium, *Coxiella burnetii*. The primary reservoirs of these bacteria are cattle, sheep and goats. *Coxiella burnetii* is resistant to environmental conditions and many common disinfectants.[137] Q fever is most commonly transmitted via the airborne route, where the organism is carried in dust contaminated with tissue, birth fluids or excreta from infected animals.[138] Prior to the commencement of vaccination programs in Australia, approximately half of all cases in New South Wales, Queensland and Victoria were among abattoir workers.[139,140]

The Australian Government funded the National Q Fever Management Program (NQFMP) between 2001 and 2006 for states and territories to provide free vaccine to at-risk groups (such as abattoir workers).[141]

Adults at risk of Q fever infection, including abattoir workers, farmers, veterinarians, stockyard workers, shearers and animal transporters, should be considered for vaccination. The administration of the Q fever vaccine requires a pre-vaccination screening test to exclude those recipients with a previous (possibly unrecognised) exposure to the organism, including previous vaccination. A Q fever vaccine may cause an adverse reaction in a person who has already been exposed to the bacterium. Vaccination is not recommended for children under 15 years of age.[50]

Epidemiological situation in 2013

In 2013 there were 477 notified cases of Q fever in Australia (a rate of 2.1 cases per 100,000), which was a 37% increase compared with the 5-year mean of 348 cases (2008 to 2012), and higher than any year since 2003.[141] This can be attributed to an increase in the number of cases notified in New South Wales during December. Whereas nationally, 10% (50/477) of cases were notified in December, 16% (26/167) of cases in New South Wales were notified in the same time period.

Between 1991 and 2001, and prior to the introduction of the NQFMP, Q fever notification rates ranged between 2.5 and 4.9 cases per 100,000.[141] In 2013, the highest notification rate was in Queensland (5.2 cases per 100,000, n=243). Cases were reported in all jurisdictions except the Australian Capital Territory and Tasmania (Figure 86).

Age and sex distribution

The median age of Q fever cases was 47 years (range 1–88 years) and 78% (n=370) were male (Figure 87). The highest notification rate was observed in males aged 55–64 years (6.2 cases per 100,000 male population). This was consistent with a report that found higher rates of Q fever in men aged 50–59 years, and that agriculture-related occupations (including farming) are the most commonly reported.[138]

Figure 86: Notified cases of Q fever, Australia, 2008 to 2013, by month and year of diagnosis and state or territory

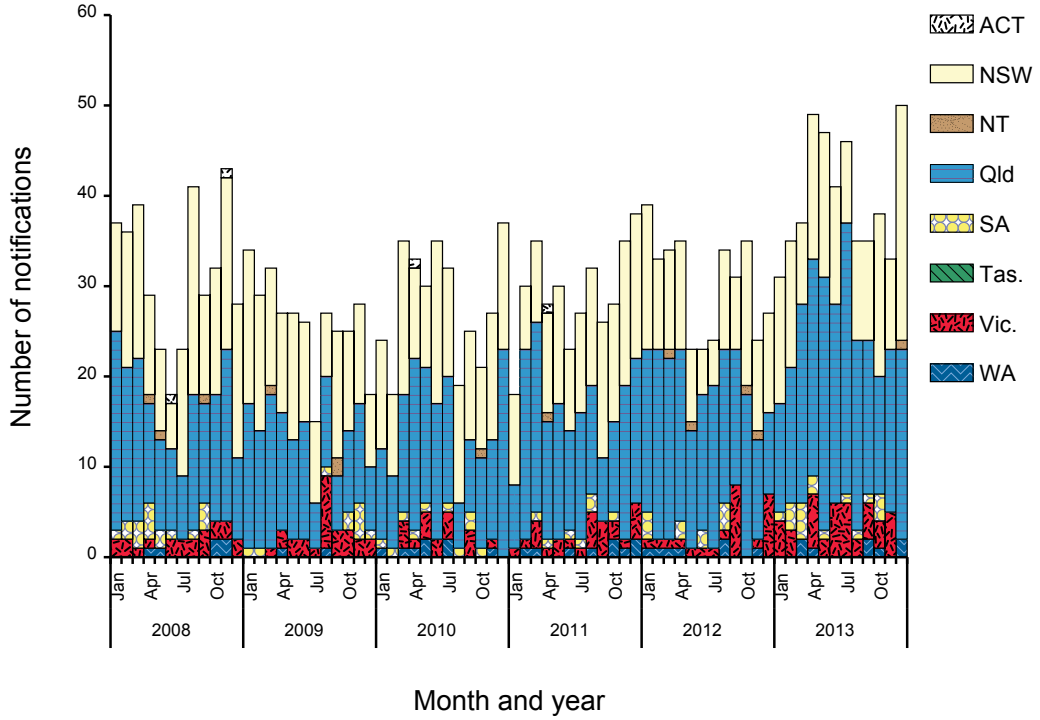
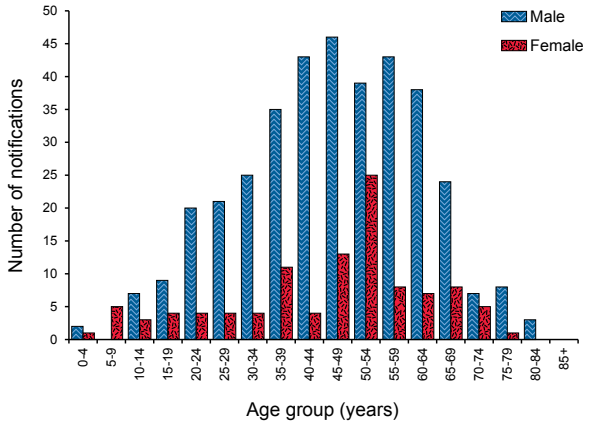


Figure 87: Notified cases of Q fever, Australia, 2013, by age group and sex



Tularaemia

- No cases of tularaemia were notified in 2013.

Tularaemia is a non-specific disease with diverse manifestations, often with an influenza-like onset, caused by infection with the bacterium *Francisella tularensis*.[21] The most common modes of transmission are through arthropod bites, handling infected animals, inhalation of infectious aerosols or exposure to contaminated food or water. Small mammals such as rodents, rabbits and hares are often the reservoir.[142]

Tularaemia was last notified in 2011, with 2 cases from Tasmania. This was the first time that *F. tularensis* type B had been detected in the Southern Hemisphere.[38,143,144]

Other bacterial infections

Legionellosis

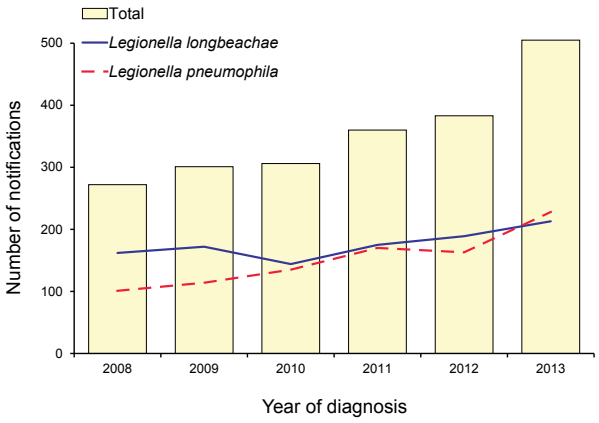
- A total of 505 cases of legionellosis were notified in 2013.
- Compared with 2012, notifications of legionellosis increased by 32% in 2013.
- *Legionella pneumophila*, commonly associated with man-made water systems, was the most frequently reported causative species in 2013.
- Five clusters and 3 outbreaks of legionellosis were reported in 2013.

Legionellosis is an environmentally acquired pneumonia caused by the bacteria *Legionella*. It can take the form of either Legionnaires' disease, a severe form of infection of the lungs, or Pontiac fever, a milder influenza-like illness.[21] The species most commonly associated with human disease in Australia are *Legionella pneumophila* and *Legionella longbeachae*. *Legionella* bacteria are found naturally in low levels in the environment. In the absence of effective environmental treatments *Legionella* organisms can breed in air conditioning cooling towers, hot water systems, showerheads, spa pools, fountains, commercial potting mix and other decomposing material such as bark and sawdust.[145–148] *Legionella* is generally transmitted to humans through contaminated water or dust aerosols.

Epidemiological situation in 2013

There were 505 notifications of legionellosis in 2013, representing a rate of 2.2 notifications per 100,000. Compared with the previous reporting period notifications of legionellosis increased in 2013 by 32% and were the highest since 2008 (Figure 88). It is likely that at least half of the increase in 2013 can be attributed to the outbreak at the Wesley Hospital in Queensland and the subsequent increase in serological testing during that period.[149] This outbreak received significant media coverage and resulted in Queensland issuing public health alerts to the community.

Figure 88: Notified cases of legionellosis, Australia, 2008 to 2013, by year of diagnosis and species

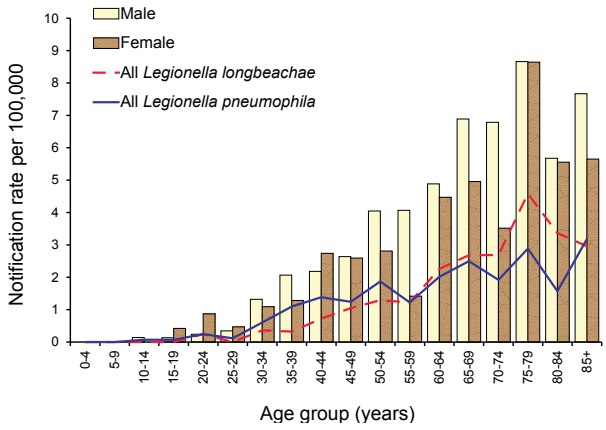


In 2013, data on the causative species were available for 88% (n=444) of notifications reported. Proportionally, there were slightly more notifications of *Le. pneumophila* (51%) than *Le. longbeachae* (48%). A single notification of *Le. anisa* and 2 notifications of *Le. micdadei* were also reported (Table 21). Serogroup information was only reported for 70% of *Le. pneumophila* notifications and 17% of *Le. longbeachae* notifications. Of these, 91% of *Le. pneumophila* notifications were typed

to *Le. pneumophila* serogroup 1 and all *Le. longbeachae* notifications were typed to *Le. longbeachae* serogroup 1.

Over the period 2008 to 2013, the notified cases of *Le. pneumophila* ranged from 101 to 228, whilst notified cases of *Le. longbeachae* ranged from 144 to 213 (Figure 89). When compared with 2012, notifications of *Le. pneumophila* increased by 40% and *Le. longbeachae* by 13%.

Figure 89: Notification rate for legionellosis, Australia, 2013, by age group and sex and species



In 2013, mortality data was available for 71% (n=358) of notifications. Of these, 4% (n=15) were reported to have died due to legionellosis. This proportion was equivalent to the proportion of notifications reported to have died in 2012 (3%, n=11). The majority of deaths were attributed to infection with *Le. pneumophila* (80%, n=12) (Table 21). Over the last 5 years (2008 to 2013) the mortality data of legionellosis notification has improved with the proportion of cases reported with death information increasing from 49% in 2008 to 71% in 2013.

Geographic description

In 2013, jurisdictional-specific rates of legionellosis varied from 0.3 per 100,000 in the Australian Capital Territory to 3.8 per 100,000 in South Australia (Table 21).

In 2013, *Le. pneumophila* was the most notified infecting species in the Australian Capital Territory, New South Wales, Queensland, South Australia and Victoria, while *Le. longbeachae* was more common in the Northern Territory and Western Australia. Tasmania reported and equal number of notifications of both species. The geographic distribution in 2013 differed from 2012 in that *Le. pneumophila* was the most commonly notified species in only New South Wales, Tasmania and Victoria, with *Le. longbeachae* being more commonly notified in all other remaining states and territories.

Age and sex distribution

In 2013, legionellosis was predominantly seen in older males. Males accounted for the majority (54%, n=271) of the notifications resulting in a male to female ratio of 1.2:1. There were no notifications in people under the age of 10 years. The highest age and sex specific rates were observed in men and women aged 75–79 years or over at 8.7 per 100,000 and 8.6 per 100,000, respectively (Figure 89). The ages of the 15 cases reported to have died due to legionellosis in 2013 ranged between 38 and 96 years (median 72 years); 11 deaths were male

Table 21: Notified cases, rates and deaths for legionellosis, Australia, 2013, by species and state or territory

Species	ACT	NSW	NT	Qld	SA	Tas.	Vic	WA	Aust.	Deaths due to legionellosis
Le. longbeachae	0	38	4	45	31‡	3	13‡	79	213	2
Le. pneumophila	1	54*	1	73*	32†	3	50†	14†	228	12
Le. anisa	0	0	0	1	0	0	0	0	1	0
Le. micdadei	0	0	0	0	0	0	2	0	2	0
Unknown species	0	13	1	46‡	0	0	1	0	61	1
Total	1	105	6	165	63	6	66	93	505	15
Rate (per 100,000)	0.3	1.4	2.5	3.5	3.8	1.2	1.2	3.7	2.2	

* 3 deaths.
† 2 deaths.
‡ 1 death.

and 4 were female. In 2013, the demographic profile of legionellosis remained consistent with the recognised epidemiology of the disease.[21,150,151]

Analysis by infecting species and age group identified that 93% of *Le. longbeachae* notifications were reported in persons aged 40 years or over and was the predominant species reported in the 75–79 years age groups (4.6 per 100,000). Similarly, 85% of notified *Le. pneumophila* infections were in persons aged 40 years or over and was the predominant species in the 85 years or over age group (3.2 per 100,000).

Seasonality

In 2013, diagnoses of legionellosis were highest in September, with 60 notified cases (Figure 90). In 2013, the seasonal pattern of *Le. pneumophila* and *Le. longbeachae* differed from the seasonal patterns seen in the previous 5 years. From 2008 to 2012, the diagnosis of *Le. pneumophila* commonly occurred in the autumn and summer months, whilst a diagnosis of *Le. longbeachae* was more common in the spring months. In 2013, the diagnosis of both species peaked in winter, with 70 *Le. pneumophila* cases and 71 *Le. longbeachae* cases notified in June, July and August (Figure 90). It is unclear why this change in seasonality occurred, but it may be the result of the increase in legionellosis testing in Queensland between June and September 2013 following the Wesley Hospital outbreak.

Place of acquisition

In 2013, a place of acquisition was reported in 80% (n=402) of legionellosis notifications. Of these, 94% (n=379) were reported to be acquired within Australia and 6% (n=23) were reported to be acquired overseas. Of the overseas acquired notifications, Thailand (17%, n=4) and Indonesia (13%, n=3) were the most commonly reported places of acquisition.

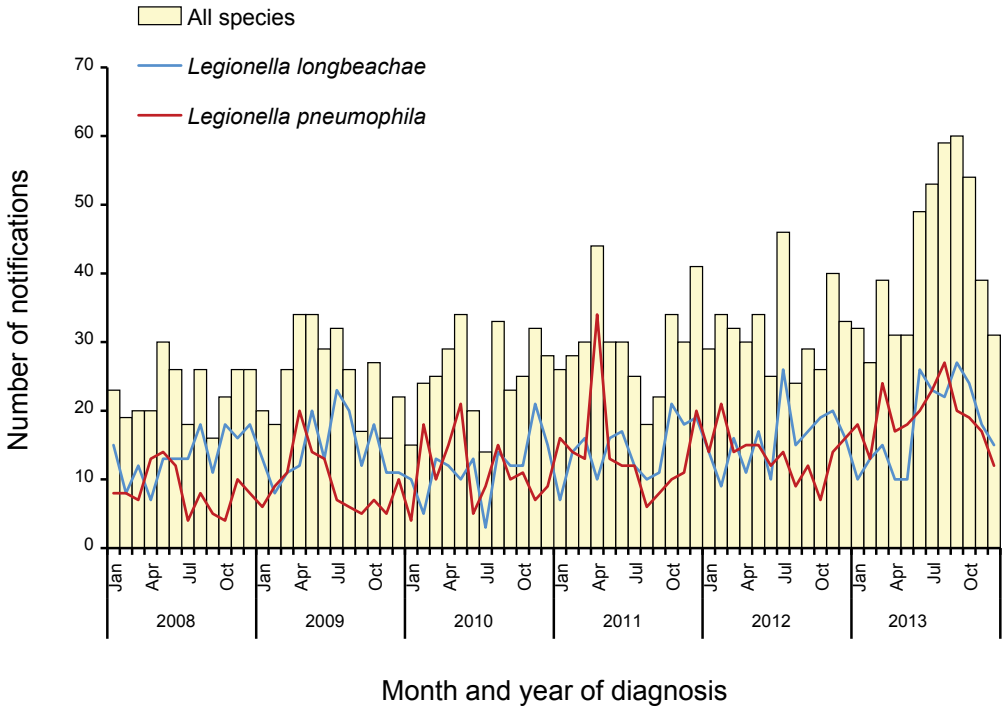
Outbreaks

In 2013, there were 5 clusters and 3 outbreaks of legionellosis notified to the NNDSS. All were attributed to *Le. pneumophila* serogroup 1 and occurred in 3 jurisdictions; Queensland, South Australia and Victoria.

There was 1 outbreak reported in Queensland. On 5 June 2013, the Wesley Hospital notified Queensland Health of 2 legionellosis cases, one resulting in death. Environmental investigations identified the most probable source of infection for this outbreak of *Le. pneumophila* was contamination of the hospitals heated water systems.[149]

In 2013, Victoria reported 4 clusters and 2 outbreaks, involving a total of 26 cases, and South Australia reported 1 cluster involving 12 cases. The sources of infection of these clusters and outbreaks were not determined.

Figure 90: Notified cases of legionellosis, Australia, 2008 to 2013, by month and year of diagnosis and species



Leprosy

- A total of 13 cases of leprosy were notified in 2013, maintaining a notification rate of less than 0.1 per 100,000.

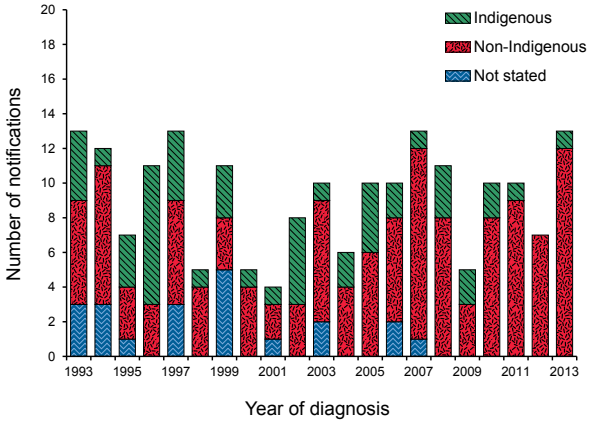
Leprosy is a chronic infection of the skin and peripheral nerves with the bacterium *Mycobacterium leprae*. Leprosy is an uncommon disease in Australia with the majority of cases occurring among migrants from leprosy endemic countries. The incidence of leprosy worldwide is declining due to various factors including economic development, bacillus Calmette-Guérin (BCG) immunisation and high coverage with multi-drug therapy.[21] Leprosy is not a highly infectious disease and is typically slow to progress to a symptomatic stage. The incubation period for leprosy is about 5 years; however, it can take as long as 20 years for symptoms to appear.[152] People at risk are generally in close and frequent contact with leprosy patients or living in countries where the disease is more common. New treatments mean the disease is now curable and once a person with leprosy begins appropriate treatment, they quickly become non-infectious.

In January 2014, the NSC redefined the diagnosis date methodology used to count leprosy cases due to the considerable amount of time that can elapse between the initial infection, the onset of symptoms and the subsequent diagnosis; and that in many of Australia's leprosy cases the infection was acquired prior to the case migrating to Australia. The diagnosis date is now derived from the 'notification received date' field rather than the earliest date recorded in either the 'true onset date', 'specimen date', 'notification date' or 'notification received date' fields. This definition also aligns with the methodology to count tuberculosis cases in Australia. Note that the new methodology has been applied retrospectively to the historical notification data described in this report. Therefore, data presented in this report may not correspond with NNDSS leprosy data published prior to January 2014.

Epidemiological situation in 2013

In 2013, a total of 13 cases of leprosy were notified (8 male, 5 female), representing a rate of 0.1 per 100,000. Cases were spread across all jurisdictions except Tasmania (Table 4). Cases ranged in age from 28 to 56 years, with a median age of 36 years. Only 1 case was recorded as Indigenous. Since 1993, annual notifications of leprosy have ranged from 4 to 13 cases per year (Figure 91).

Figure 91: Notified cases of leprosy, Australia, 1993 to 2013, by year of diagnosis and Indigenous status



Meningococcal disease (invasive)

Meningococcal disease is caused by the bacterium *Neisseria meningitidis,* and invasive disease occurs when bacteria enter a normally sterile site, usually the blood (septicaemia), cerebrospinal fluid (meningitis) or both. Asymptomatic respiratory tract carriage of meningococci is present in 5%–10% of the population and prevalence may be higher when groups of people occupy small areas of any space.[21, 50] The disease is transmitted via respiratory droplets and has an incubation period of between 1 and 10 days, more commonly 3 to 4 days.[50,153] It occasionally causes a rapidly progressive serious illness, most commonly in previously healthy children and young adults. Globally, serogroups A, B, C, W135 and Y most commonly cause disease.[21] Historically, *N. meningitidis* serogroups B and C have been the major cause of invasive meningococcal disease (IMD) in Australia.

Epidemiological situation in 2013

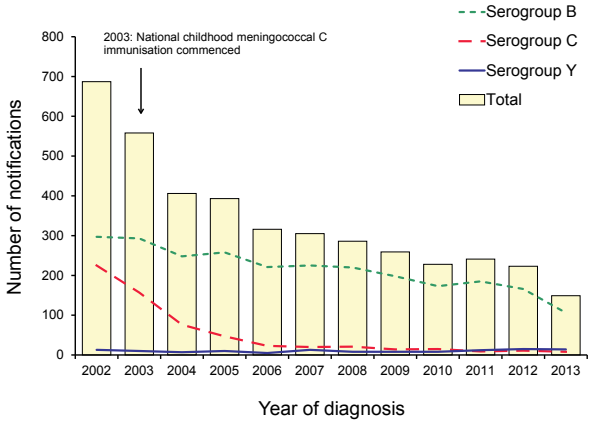
In 2013, there were 149 cases of IMD representing a rate of 0.6 per 100,000. This was a decrease of 33% compared with 2012 (n=222) and the lowest number of cases notified over the previous 10 years (Figure 92).

Most cases (n=145) notified in 2013 met the case definition as a confirmed case, that is, diagnosed based on laboratory definitive evidence, or laboratory suggestive evidence and clinical evidence.[154] A small number of cases (n=4) met the case definition as a probable case, that is, diagnosed based on clinical evidence only.

Data on serogroup were available for 94% (n = 140) of cases in 2013; 76% of which were caused by serogroup B organisms, 10% by serogroup Y, 6% by serogroup C and 9% by serogroup W135

(Table 22). The number of cases of IMD caused by serogroup B notified in 2013 was lower than in any of the preceding 10 years. Notifications of IMD caused by serogroup C organisms decreased by 27.3% from the previous year (n= 11). The number of serogroup Y cases notified in 2013 (n=14) was consistent with 2012 (n=15) but higher than the average of the previous 10 years (2003–2012) of 9.6 cases. Serogroup Y infections account for a small but increasing proportion of total IMD notifications, increasing from 3% of cases notified in 2009 to 4% in 2010, 5% in 2011 and 7% in 2012. Since the introduction of the meningococcal C vaccine on the NIP in 2003, notifications caused by serogroup C organisms have decreased by 94% with fewer than 10 cases reported annually for the past 3 years.

Figure 92: Notified cases of invasive meningococcal disease, Australia, 2002 to 2013, by year of diagnosis and serogroup



Mortality data were available for 60% (n=89) of cases reported to the NNDSS in 2013. There were 5 cases reported as having died from IMD, including two due to serogroup B, one due to serogroup C, one due to serogroup Y and 1 death due to an unknown serogroup (Table 22).

The serogroup C related death occurred in an unvaccinated person in the 50–54 years age group. Of the deaths due to serogroup B organisms, 1 child was less than 5 years of age, and the other was in the 15–19 years age group. The unknown serogroup death occurred in the 45–49 years age group and the serogroup Y related death occurred in the 85 years or over age group.

Geographic description

All jurisdictions aligned with the national case definition for IMD, except the Australian Capital Territory and New South Wales where conjunctival cases were also reportable under the local case definition and reported nationally. Conjunctival cases cannot be distinguished from invasive cases in the national dataset.

In 2013, cases of IMD were reported from all states and territories, ranging from two cases in the Northern Territory to 48 cases from New South Wales (Table 22). Jurisdictional specific rates ranged from 0.6 per 100,000 in Tasmania to 1.8 per 100,000 in South Australia.

Age and sex distribution

More males than females were reported with IMD in 2013, with a male to female ratio of 1.3:1. Proportionally, 40% of all cases (n=102) reported were less than 25 years of age, of which those less than 5 years of age made up almost half (n=47). Specifically, the 0–4 years age group had the highest rate of 6.1 per 100,000 followed by the 15–19 years age group (3.7 per 100,000) and the 20–24 years age group (1.7 per 100,000) (Figure 93).

Serogroup B accounted for the majority of cases across all age groups including those aged less

Table 22: Notified cases of invasive meningococcal disease and deaths due to invasive meningococcal disease, Australia, 2013, by serogroup and state or territory

	ACT	NSW	NT	Qld	SA	Tas.	Vic.	WA	Aust.	Deaths due to IMD
B	2	27	2	25	18	2	19	11	106	2
C	0	3	0	2	0	0	1	2	8	1
W135	0	6	0	3	1	0	1	1	12	0
Y	1	8	0	2	1	0	1	1	14	1
Unknown	0	4	0	1	0	1	3	0	9	1
Total	3	48	2	33	20	3	25	15	149	5
Rate (cases per 100,000)	0.8	0.6	0.8	0.7	1.2	0.6	0.4	0.6	0.6	–

than 25 years. While the age-specific rates of serogroup B infection in 2013 remain high compared with other serogroups they continue to trend downward across all age groups (Figure 94).

Figure 93: Notification rate for invasive meningococcal disease, Australia, 2013, by age group and sex

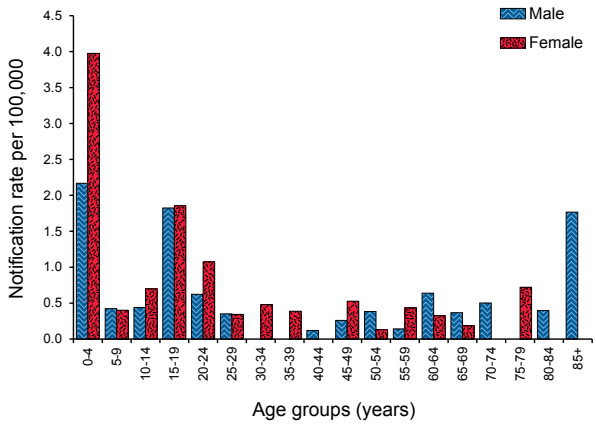
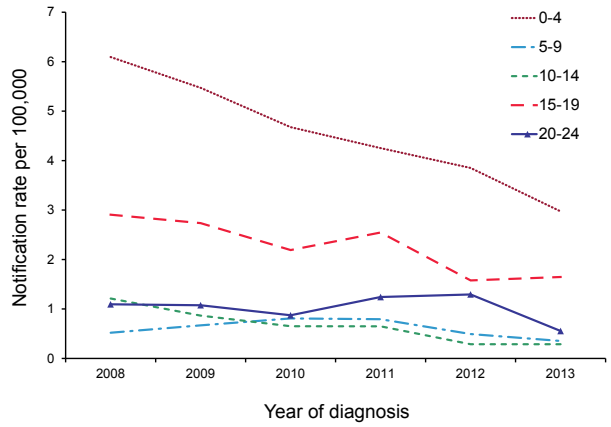


Figure 94: Notification rate for serogroup B invasive meningococcal disease, Australia, 2008 to 2013, by year of diagnosis and select age groups

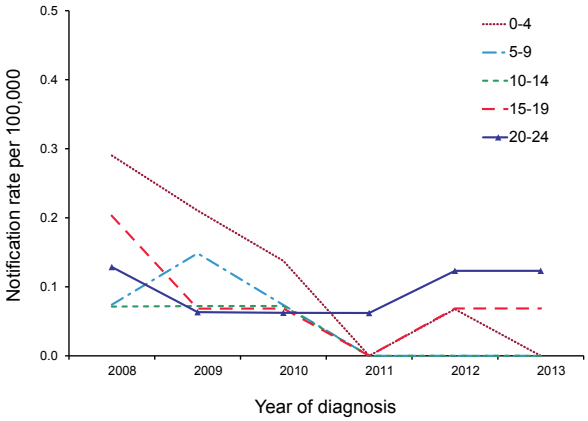


Of the 8 cases of IMD due to serogroup C notified in 2013 only 3 were among children and young adults aged less than 25 years of age, and therefore eligible for vaccination. None of the cases were in the 0–4 years of age group. Age-specific rates have been maintained at very low levels in 2013, with no age group exceeding 0.1 cases per 100,000 in 2013 (Figure 95).

Seasonality

An average of 12 cases of IMD was reported monthly in 2013, with a monthly range of 5 to 21 cases. A clear seasonal pattern was apparent, with the highest number of notifications reported in the winter months. This was consistent with the normal seasonal pattern of this disease (Figure 96). The seasonal trend was more marked in cases aged 5 years or over.

Figure 95: Notification rate for serogroup C invasive meningococcal disease, Australia, 2008 to 2013, by select age groups



Susceptibility

The Australian Meningococcal Surveillance Program (AMSP) was established in 1994 for the purpose of monitoring and analysing isolates of *N. meningitidis* from cases of IMD in Australia. The program is undertaken by a network of reference laboratories in each state and territory, using standardised methodology to determine the phenotype (serogroup, serotype and serosubtype) and the susceptibility of *N. meningitidis* to a core group of antibiotics.

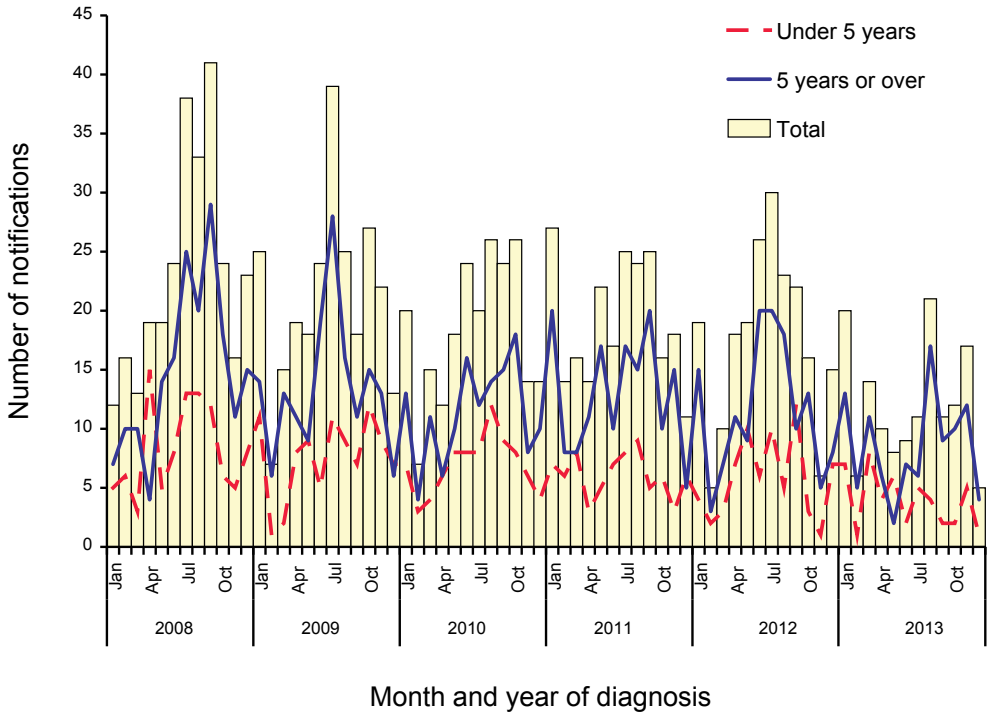
Annual reports of the AMSP are published in CDI with the most recent report published for 2013.[76] The latest data from AMSP show that 79% of isolates tested demonstrated decreased susceptibility to the penicillin group of antibiotics, and no isolates exhibited resistance to penicillin. All IMD isolates tested were susceptible to ceftriaxone; ciprofloxacin and rifampicin.

Vaccination

From 2003, meningococcal C vaccine has been available for infants aged 12 months as a part of the childhood immunisation schedule funded under the NIP. Additionally, a catch-up program provided access to the meningococcal C vaccine for children and adolescents born between 1984 and 2001.

Of the 8 cases of IMD caused by serogroup C organisms reported in 2013, three were eligible for

Figure 96: Notified cases of invasive meningococcal disease, Australia, 2008 to 2013, by age group and month and year of diagnosis



vaccination of which 1 case was reported as vaccinated and the remaining two were of unknown vaccination status.

Discussion

In Australia, IMD has reached its lowest levels since the national notification commenced in 1991. The reduction has been seen most considerably in disease caused by serogroup C; however, declines in disease caused by serogroup B are also evident. In 2013, serogroup Y continued to account for an increasing proportion of notified cases. This small but increasing trend in serogroup Y infections will continue to be monitored.

Tuberculosis

- 1,265 cases of tuberculosis were notified in 2013.

Tuberculosis (TB) is an infection caused by the bacterium *Mycobacterium tuberculosis*. TB is transmitted by airborne droplets produced by people with pulmonary or respiratory tract TB during coughing or sneezing. While Australia has one of the lowest rates of TB in the world, the disease remains a public health issue, particularly in Australia's overseas-born and Indigenous communities.[155]

Epidemiological situation in 2013

In 2013, a total of 1,265 cases of TB were notified to the NNDSS representing a rate of 5.5 per 100,000. Australia has achieved good TB control and has maintained low rates of TB since the mid-1980s; however, in the decade leading up to 2011 a steady increase in incidence was observed. Contrary to this increasing trend the 2012 and 2013 rates have both decreased and may be an indication that incidence is beginning to plateau or even decrease (Figure 97).

In 2013, 2.4% of TB notifications were recorded as being Indigenous. This represents a rate of 5.0 per 100,000 in Aboriginal and Torres Strait Islander peoples.

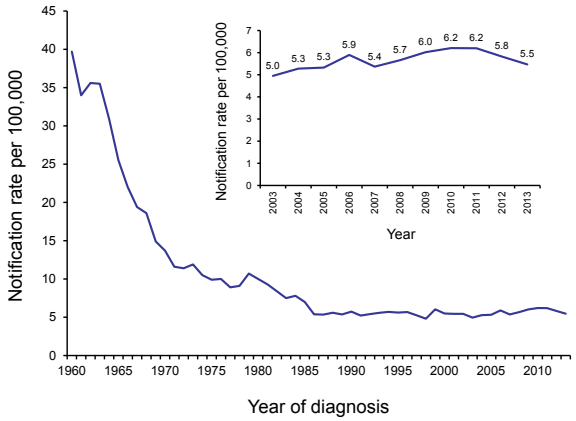
Geographic description

New South Wales (n=440), Victoria (n=383), Queensland (n=156) and Western Australia (n=150) accounted for 89% of all cases of TB diagnosed in Australia. The Northern Territory (17.0 per 100,000), Victoria (6.7 per 100,000), Western Australia (6.0 per 100,000) and New South Wales (5.9 per 100,000) all recorded a rate higher than the national notification rate.

In 2013, the Northern Territory, Tasmania and Victoria all recorded higher notification rates than the previous year. All the other states and ter-

ritories reported a decrease on the previous year, with the greatest decrease being reported by South Australia (18% decrease).

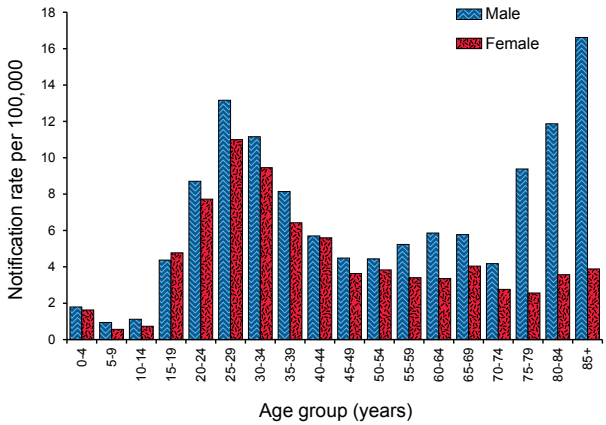
Figure 97: Notification rate for tuberculosis, Australia, 1960 to 2013, by year of diagnosis



Age and sex distribution

In 2013, 50% of all TB notifications were seen in people aged 20–39 years. Overall, the age group with the highest notification rate was the 25–29 years age group (12.1 per 100,000) and the highest age and sex specific rates were observed in men aged 85 years or over (16.6 per 100,000) and women aged 25–29 years (11.0 per 100,000)0 (Figure 98).

Figure 98: Notification rate for tuberculosis, Australia, 2013, by age group and sex



Males accounted for more than half (57%) of the TB notifications, resulting in a male to female ratio of 1.3:1.

Vaccination

The BCG vaccine was first introduced for protection against tuberculosis in the 1920s and despite variable evidence on the efficacy of the vaccine it remains the only vaccine in use for TB today.[156,157]

According to national guidelines developed by Australia's National Tuberculosis Advisory Committee, BCG vaccination is recommended for: Aboriginal and Torres Strait Islander neonates in communities with a high incidence of TB; neonates and children under 5 years of age who will be travelling to or living in countries or areas with a high prevalence of TB for extended periods; and neonates born to parents with leprosy or a family history of leprosy.

BCG vaccination is not recommended for general use in the Australian population or for most health care workers and is contraindicated in HIV infected persons.[158] Note that BCG immunisation practices may vary between states and territories due to differences in jurisdiction-specific TB vaccination policies and population demographics.

Enhanced surveillance data sets

Enhanced data are collected on all cases of TB. Further analyses, including identification of risk groups and reporting on treatment outcomes, can be found in the TB annual report series also published in CDI.

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Abbreviations

7vPCV	7 valent pneumococcal conjugate vaccine
13vPCV	13 valent pneumococcal conjugate vaccine
23vPPV	23 valent pneumococcal polysaccharide vaccine
ABLV	Australian bat lyssavirus
AFP	acute flaccid paralysis
AGSP	Australian Gonococcal Surveillance Programme
AIDS	acquired immune deficiency syndrome
AMSP	Australian Meningococcal Surveillance Programme
ANCJDR	Australian National Creutzfeldt-Jakob Disease Registry
BCG	bacillus Calmette–Guérin
BFV	Barmah Forest virus
CDI	Communicable Diseases Intelligence
CDNA	Communicable Diseases Network Australia
CDWG	Case Definitions Working Group
CJD	Creutzfeldt-Jakob disease
CRS	congenital rubella syndrome
DENV	dengue virus
Hib	*Haemophilus influenzae* type b
HIV	human immunodeficiency virus
HPAIH	highly pathogenic avian influenza in humans
HUS	haemolytic uraemic syndrome
ILI	influenza like illness
IMD	invasive meningococcal disease
IPD	invasive pneumococcal disease
JEV	Japanese encephalitis virus
KUNV	Kunjin virus
MenCCV	meningococcal serogroup C vaccine
MMR	measles-mumps-rubella
MMRV	measles-mumps-rubella-varicella
MVEV	Murray Valley encephalitis virus
NDP	no data provided
NEC	not elsewhere classified
NIP	National Immunisation Program
NN	not notifiable
NNDSS	National Notifiable Diseases Surveillance System
NQFMP	National Q Fever Management Program
NSC	National Surveillance Committee
NS1	non-structural protein 1
PCR	polymerase chain reaction
RRV	Ross River virus
SARS	severe acute respiratory syndrome
STEC	Shiga toxin-producing *Escherichia coli*
STI(s)	sexually transmissible infections(s)
TB	tuberculosis
VPD(s)	vaccine preventable disease(s)
VTEC	verotoxigenic *Escherichia coli*
VZV	varicella zoster virus
WHO	World Health Organization
WHOCC	World Health Organization Collaborating Centre for Reference and Research on Influenza

Appendices

Appendix 1: December estimate of Australian population, 2013, by state or territory

	ACT	NSW	NT	Qld	SA	Tas.	Vic.	WA	Aus.
Males	189,768	3,678,352	127,257	2,321,199	828,037	255,702	2,839,733	1,275,196	11,517,323
Females	191,701	3,731,240	113,939	2,333,812	842,652	257,422	2,899,143	1,245,377	11,616,389
Total	381,469	7,409,592	241,196	4,655,011	1,670,689	513,124	5,738,876	2,520,573	23,133,712

Source : Australian Bureau of Statistics 3101.0 Table 4, Estimated Resident Population, State and Territories. Australian Demographic Statistics, Dec 2013

Appendix 2: December estimate of Australian population, 2013, by state or territory and age

Age group	State or territory								
	ACT	NSW	NT	Qld	SA	Tas.	Vic.	WA	Aus.
00–04	25,991	487,499	19,047	315,970	100,154	31,398	368,889	168,540	1,517,647
05–09	23,173	464,532	17,794	309,807	97,897	31,619	349,581	160,960	1,455,520
10–14	21,309	446,747	17,035	298,286	97,330	32,076	332,759	153,006	1,398,739
15–19	23,139	464,824	16,441	308,284	104,427	33,778	355,920	160,096	1,467,083
20–24	33,378	506,478	19,415	333,470	115,159	31,511	415,446	186,518	1,641,597
25–29	34,120	531,807	23,298	339,744	115,037	29,710	442,778	211,548	1,728,480
30–34	31,683	527,248	21,394	323,018	107,734	29,179	424,394	193,052	1,658,077
35–39	27,927	495,899	18,392	310,893	102,797	29,506	392,703	174,020	1,552,362
40–44	28,069	523,433	18,474	339,039	116,213	34,967	417,107	186,341	1,663,899
45–49	24,901	482,285	15,938	308,603	112,752	34,093	380,574	170,171	1,529,523
50–54	24,511	499,586	15,538	310,798	116,210	37,694	377,742	166,499	1,548,794
55–59	21,274	452,953	13,249	275,044	107,068	35,756	340,422	147,621	1,393,560
60–64	18,641	402,227	10,202	248,185	98,343	33,267	301,625	127,929	1,240,619
65–69	15,272	356,695	6,898	216,879	86,919	29,431	264,253	105,312	1,081,738
70–74	10,129	260,379	3,958	153,226	63,235	21,288	194,038	74,910	781,221
75–79	7,344	200,443	2,117	108,522	49,494	15,629	150,539	55,352	589,468
80–84	5,261	153,463	1,223	79,364	38,926	11,477	115,655	40,432	445,819
85+	5,347	153,094	783	75,879	40,994	10,745	114,451	38,266	439,566
Total	381,469	7,409,592	241,196	4,655,011	1,670,689	513,124	5,738,876	2,520,573	23,133,712

Source : Australian Bureau of Statistics 3101.0 Australian Demographic Statistics Tables, Dec 2013.

Appendix 3: Indigenous status, National Notifiable Diseases Surveillance System, Australia, 2013, by notifiable disease*

Disease name	Aboriginal but not TSI origin	TSI but not Aboriginal origin	Aboriginal and TSI origin	Not Indigenous	Not stated	Blank/ missing	Total	% complete	Number complete	Number incomplete
Arbovirus (NEC)	2	–	–	8	11	–	21	48	10	11
Australian bat lyssavirus	–	–	–	1	–	–	1	100	1	–
Barmah Forest virus infection	91	7	5	1,811	1,798	527	4,239	45	1,914	2,325
Botulism	–	–	–	2	1	1	4	50	2	2
Brucellosis	–	–	–	11	3	–	14	79	11	3
Campylobacteriosis	226	12	11	7,473	6,607	369	14,698	53	7,722	6,976
Chlamydial infection	5,792	655	344	24,835	30,504	20,396	82,526	38	31,626	50,900
Cholera	–	–	–	3	–	–	3	100	3	–
Cryptosporidiosis	168	2	5	2,085	1,318	268	3,846	59	2,260	1,586
Dengue virus infection	9	5	–	1,515	269	43	1,841	83	1,529	312
Diphtheria	–	–	–	1	1	–	2	50	1	1
Gonococcal infection	3,856	217	108	6,565	2,866	1,330	14,942	72	10,746	4,196
Haemolytic uraemic syndrome	–	–	–	14	1	–	15	93	14	1
Haemophilus influenzae type b	1	–	1	16	2	–	20	90	18	2
Hepatitis A	2	–	1	177	9	–	189	95	180	9
Hepatitis B (newly acquired)	11	1	1	130	24	5	172	83	143	29
Hepatitis B (unspecified)	171	17	5	2,563	1,891	2,332	6,979	40	2,756	4,223
Hepatitis C (newly acquired)	71	–	1	273	55	7	407	85	345	62
Hepatitis C (unspecified)	687	12	25	3,323	3,372	2,889	10,308	39	4,047	6,261
Hepatitis D	–	–	–	44	8	1	53	83	44	9
Hepatitis E	–	–	–	29	2	–	31	94	29	2
Influenza (laboratory confirmed)	662	47	27	10,630	8,312	8,651	28,329	40	11,366	16,963
Japanese encephalitis virus infection	–	–	–	3	1	–	4	75	3	1
Kunjin virus infection	–	–	–	1	2	–	3	33	1	2
Legionellosis	14	1	1	390	97	2	505	80	406	99
Leprosy	1	–	–	12	–	–	13	100	13	–
Leptospirosis	2	–	–	74	19	–	95	80	76	19
Listeriosis	2	–	–	68	6	–	76	92	70	6
Malaria	1	1	–	331	80	1	414	80	333	81
Measles	3	–	–	140	15	–	158	91	143	15

Appendix 3 (cont'd): Indigenous status, National Notifiable Diseases Surveillance System, Australia, 2013, by notifiable disease*

Disease name	Aboriginal but not TSI origin	TSI but not Aboriginal origin	Aboriginal and TSI origin	Not Indigenous	Not stated	Blank/ missing	Total	% complete	Number complete	Number incomplete
Meningococcal disease (invasive)	13	1	–	130	5	–	149	97	144	5
Mumps	5	–	–	166	38	8	217	79	171	46
Murray Valley encephalitis virus infection	–	–	–	–	1	–	1	–	–	1
Ornithosis	–	–	–	38	8	1	47	81	38	9
Pertussis	265	6	17	6,019	4,780	1,254	12,341	51	6,307	6,034
Pneumococcal disease (invasive)	182	6	5	1,163	130	60	1,546	88	1,356	190
Q fever	16	1	3	375	73	9	477	83	395	82
Ross River virus infection	89	4	6	2,070	1,548	591	4,308	50	2,169	2,139
Rubella	–	–	–	20	4	1	25	80	20	5
Rubella – congenital	–	–	–	2	–	–	2	100	2	–
Salmonellosis	374	9	17	6,348	3,356	2,687	12,791	53	6,748	6,043
Shigellosis	133	1	1	369	38	14	556	91	504	52
STEC	2	–	–	134	40	4	180	75	136	44
Syphilis – congenital	3	–	–	4	–	–	7	100	7	–
Syphilis < 2 years	126	9	7	1,457	155	14	1,768	91	1,599	169
Syphilis > 2 years or unspecified duration	188	21	8	1,096	384	9	1,706	77	1,313	393
Tetanus	–	–	–	3	–	1	4	75	3	1
Tuberculosis	27	5	–	1,232	1	–	1,265	100	1,264	1
Typhoid fever	–	2	–	140	6	2	150	95	142	8
Varicella zoster (chickenpox)	97	1	5	1,735	165	39	2,042	90	1,838	204
Varicella zoster (shingles)	136	2	5	4,358	413	103	5,017	90	4,501	516
Varicella zoster (unspecified)	126	12	5	2,272	7,185	327	9,927	24	2,415	7,512
Total	13,554	1,057	614	91,659	75,604	41,946	224,434	48	106,884	117,550

* Indigenous status is usually obtained from medical notification and completeness varies by disease and by state and territory. This reflects differences in notification requirements (i.e. depending on the jurisdiction, some diseases are primarily or completely notified by pathology laboratories rather than clinicians) and the fact that it is not possible to follow-up all cases for diseases with a large volume of notifications and/or not requiring specific case-based public health action.

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Quarterly report

OzFoodNet quarterly report, 1 October to 31 December 2013

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. 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 October and 31 December 2013.

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 4th quarter of 2013, OzFoodNet sites reported 674 outbreaks of enteric illness, including those transmitted by contaminated food. Outbreaks of gastroenteritis are often not reported to health agencies or the reports may be delayed, meaning that these figures under-represent the true burden of enteric disease outbreaks. In total, these outbreaks affected 12,038 people, of whom 321 were hospitalised. There were 37 deaths reported during these outbreaks. The majority of outbreaks (543) were due to person-to-person transmission (Table 1), with 55% (298/543) of these occurring in residential aged care facilities and 25% (137/543) occurring in child care centres.

Table 1: Outbreaks and clusters of gastrointestinal illness reported by OzFoodNet, 1 October to 31 December 2013 by mode of transmission

Transmission mode	Number of outbreaks and clusters	Per cent of total*
Foodborne and suspected foodborne	37	5
Person-to-person	543	81
Unknown (*Salmonella* cluster)	10	1
Unknown (other pathogen cluster)	3	<1
Unknown	81	12
Total	674	100

* Percentages do not add to 100 due to rounding.

Foodborne and suspected foodborne disease outbreaks

There were 37 outbreaks during this quarter where consumption of contaminated food was suspected or confirmed as being the primary mode of transmission (Appendix). These outbreaks affected 1,028 people and resulted in 50 hospitalisations. There were 3 deaths reported during these outbreaks. This is the same number of outbreaks as were reported in the 4th quarter of 2012 and the same as the 5-year mean for the 4th quarter between 2008 and 2012. A limitation of the outbreak data provided by OzFoodNet sites for this report was the potential for variation in the categorisation of the features of outbreaks depending on circumstances and investigator interpretation. Changes in the number of foodborne outbreaks should be interpreted with caution due to the small number each quarter.

Salmonella Typhimurium was identified as or suspected to have been the aetiological agent in 10 (27%) foodborne or suspected foodborne outbreaks during this quarter, equal to the number from the same quarter in 2012. The aetiological agents for the remaining outbreaks included: norovirus in 8 outbreaks (22%), ciguatoxin in 7 outbreaks (19%), *Campylobacter* in 4 outbreaks (11%), and *Shigella flexneri*, fish wax ester and histamine fish poisoning for 1 outbreak (3%) each. For 5 outbreaks (14%), the aetiological agent was unknown. The 9 outbreaks associated with fish toxins affected 27 people and all but one occurred in Queensland. There were only 2 fish toxin outbreaks recorded in the 4th quarter of 2012 (both histamine fish poisoning in Queensland) affecting 6 people.

Fourteen outbreaks (38% of all the foodborne or suspected foodborne outbreaks) reported in this quarter were associated with food prepared in restaurants (Table 2), compared with 67% (18/27) in the previous quarter and 57% (21/37) in the 4th quarter of 2012.

Table 2: Outbreaks of foodborne or suspected foodborne disease reported by OzFoodNet, 1 October to 31 December 2013 by food preparation setting

Food preparation setting	Outbreaks
Restaurant	14
Primary produce	9
Commercial caterer	4
Private residence	2
Camp	2
Hospital	1
Bakery	1
Aged care	1
Function centre	1
Grocery store/delicatessen	1
Institution	1
Total	37

To investigate these outbreaks, sites conducted 7 cohort studies, 4 case control studies and collected descriptive case series data for 23 investigations, while for 3 outbreaks no individual patient data were collected. The evidence used to implicate food vehicles included analytical and microbiological evidence in 3 outbreaks, analytical evidence in 3 outbreaks, microbiological evidence in 2 outbreaks, and descriptive evidence in 29 outbreak investigations.

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

Australian Capital Territory

There was 1 outbreak of foodborne illness reported in the Australian Capital Territory during this quarter. The aetiological agent was identified as *Campylobacter jejuni*.

Description of key outbreak

An outbreak of campylobacteriosis was investigated in October following a university college formal dinner. Menu details and contact details for attendees were obtained through the college and a retrospective cohort study was undertaken. Approximately 50 cases of gastroenteritis were identified, including 7 laboratory confirmed *C. jejuni* infections. Multivariate analysis identified chicken liver pâté as being significantly associated with illness (adjusted odds ratio [AOR] 7.2, 95% confidence interval [CI] 3.3–15.7, $P<0.0001$). Samples of leftover pâté were positive for both *C. jejuni* and *C. coli*. The outbreak was assumed to be due to insufficient cooking of chicken livers in the preparation of the pâté.

New South Wales

There were 4 outbreaks of foodborne or suspected foodborne illness reported in New South Wales during this quarter. The aetiological agents were identified as norovirus for 2 outbreaks and *S.* Typhimurium phage type (PT) 170/108* (multilocus variable number tandem repeat analysis [MLVA] profile 03-10-07-14-523) for 1 outbreak. The aetiological agent was unable to be determined for 1 outbreak.

Description of key outbreak

An outbreak of salmonellosis was investigated in October after 7 people presented to an emergency department with gastrointestinal symptoms. All seven reported eating Vietnamese style rolls from the same bakery. Active case finding identified 46 people who reported gastrointestinal illness after eating at this bakery in late October, almost half of whom were hospitalised (21, 46%) and 3 secondary cases. *S.* Typhimurium PT 170/108 (MLVA 03-10-07-14-523) was identified in 36 stool specimens, including the 3 secondary cases. The primary cases all reported eating Vietnamese style rolls with a variety of fillings. Mayonnaise made with raw egg was used in all the rolls. None of the mayonnaise was available at the time of environmental inspection but other samples: pâté, lettuce, and an environmental swab from the cool room were positive for the outbreak strain suggesting a cross-contamination incident. Due to a long chain of resale, the egg supplier could not be definitively identified by the trace-back investigation.

Northern Territory

There were 2 outbreaks of suspected foodborne illness reported in the Northern Territory during this quarter. The aetiological agent was unknown for both outbreaks.

Queensland

There were 15 outbreaks of foodborne or suspected foodborne illness reported in Queensland during this quarter. The aetiological agents were identified as ciguatoxin for 7 outbreaks, norovirus for 2 outbreaks, and *S.* Typhimurium PT 16 (MLVA 03-13-10-12-524), *S.* Typhimurium PT 170/108

* Classification of this organism differs between laboratories, with the Microbiological Diagnostic Unit using PT 170 to classify this type of *Salmonella* Typhimurium and SA Pathology using PT 108 due to a difference in the interpretation of one phenotypic characteristic.

(MLVA 03-09-07-14-524), *S.* Typhimurium (MLVA 03-12-12-09-524) *S.* Typhimurium (MLVA 03-10-07-09-524) and histamine for 1 outbreak each. The aetiological agent was unable to be determined for 1 outbreak.

Description of key outbreak

A large gastroenteritis outbreak was investigated in November among people who consumed meals at multiple Melbourne Cup functions that were catered for by a single catering company. People who ate at 63% (25/40) of the catered functions reported illness with an estimated 350 persons in total affected, including 12 hospitalisations and 1 associated death. *S.* Typhimurium PT 16 (MLVA 03-13-10-12-524) was identified in 83 faecal specimens that were collected from attendees of various functions. Both cohort and case control studies were conducted involving a total of 143 participants. A case control study comprising data collected from 4 catered groups found consumption of potato salad to be significantly associated with illness (odds ratio [OR] 5.5, 95% CI 1.3–23.4, $P=0.009$). A cohort study of a 5th catered function also found consumption of potato salad to be associated with illness but the association was not statistically significant (relative risk [RR] 2.2, 95% CI 0.7–7.0, $P=0.056$).

The outbreak strain was detected in a sample of cooked ham from an unopened catering pack leftover from one function, and in multiple leftover food items including chicken, potato salad and mayonnaise collected from another function. *Escherichia coli* was also detected in some of the leftover foods. Raw eggs used in the preparation of mayonnaise (without further cooking) and then used to dress salad items including the potato salad were considered the likely source of infection for this outbreak. Cross contamination of foods in the kitchen during preparation for these 40 functions was also considered highly likely. The eggs were traced back to a single farm and an audit conducted by SafeFood Queensland indicated compliance with the current egg standards; however, no drag swabs or microbiological testing at the farm level were conducted.

South Australia

There were 2 outbreaks of foodborne or suspected foodborne illness reported in South Australia during this quarter. The aetiological agents were identified as *S.* Typhimurium PT 9 (MLVA 03-24-12-10-523) and *Campylobacter*.

An investigation in November identified 11 cases of *S.* Typhimurium PT 9 (MLVA 03-24-12-10-523) in people who reported eating at the same restaurant over a 1 week period. Four of the cases required hospitalisation. Ten of the cases ate a salt and pepper squid dish that was served with a raw egg based aioli and 1 case was a chef at the restaurant who tried a variety of foods. The restaurant had been the subject of a previous investigation reported a month earlier where coleslaw made with the raw egg aioli base was statistically associated with illness.[1] An environmental inspection was conducted and found inadequate handling practices of the aioli. A sample of aioli was collected from the venue but *Salmonella* was not detected.

Tasmania

There was 1 outbreak of suspected foodborne illness reported in Tasmania during this quarter. The aetiological agent was *Shigella flexneri* and the cases were acquired whilst on camp overseas.

Victoria

There were 8 outbreaks of foodborne or suspected foodborne illness reported in Victoria during this quarter. The aetiological agents were identified as norovirus for 2 outbreaks, and *C. coli*, fish wax ester, *S.* Typhimurium PT 135, *S.* Typhimurium PT 9, *C. jejuni*, and *S.* Typhimurium PT 170/108 for 1 outbreak each.

Description of key outbreak

In early November, health authorities were notified of 6 aged care facility residents with onset of gastrointestinal illness over a 10-day period. It was found that all meals for the facility were prepared in a large central cook-chill kitchen that also supplied a large hospital, several other aged care facilities, a psychiatric unit, several cafes and businesses and a meals on wheels program. In total there were 27 cases (21 confirmed *S.* Typhimurium PT 135 infections and 6 suspected cases), including 2 deaths during the outbreak period. Twelve of 21 confirmed cases and all suspected cases had consumed meals prepared in a central cook-chill kitchen during their incubation period. *S.* Typhimurium PT 135 was detected in a sample of frittata mix collected from the central kitchen. The central kitchen now uses only pasteurised egg products.

Western Australia

There were 3 outbreaks of foodborne or suspected foodborne illness reported in Western Australia during this quarter. The aetiological agents were identified as norovirus for 2 outbreaks and the aetiological agent was unable to be determined for the remaining outbreak.

Description of key outbreak

An investigation was conducted into reports of gastrointestinal illness among 8 people who had independently eaten at a rural hotel in early December. A case series investigation found that ill people had diarrhoea (8/8) and vomiting (8/8) and fever (3/8), with an average incubation period of 28 hours and the average duration of illness of 34 hours. Three specimens were positive for norovirus. Cases had eaten a variety of meals, with hot chips and a side salad common to all cases. Three food handlers were ill and vomited in the staff toilets at the time the cases ate at the hotel. It is likely that a food handler with a norovirus-like illness had contaminated foods that were subsequently eaten by hotel patrons.

Multi-jurisdictional investigation

Salmonella *Typhimurium PT 29 MLVA 03-11-10-11-523*

OzFoodNet commenced a multi-jurisdictional outbreak investigation on 14 October 2013 upon identifying a cluster of *Salmonella* infections among persons from the Australian Capital Territory, New South Wales, South Australia and Victoria who all attended a national sporting institution in Canberra. A case was defined as any person consuming food at the institute between 23 September and 2 October who subsequently developed gastroenteritis, with a confirmed case having a faecal specimen positive for *S.* Typhimurium MLVA 03-11-10-11-523.

In total, 22 cases were linked to the outbreak, including 14 laboratory-confirmed *Salmonella* infections. A cohort study was conducted among the Victorian attendees (29/43 interviewed, 14 cases identified). Univariate analysis identified a number of food items associated with increased risk of illness including consuming fruit smoothies on 26 September (RR 3.1, 95% CI 1.3–7.6, $P=0.005$), muffins on 26 September (RR 2.9, 95% CI 1.6–5.0, $P=0.004$) and chicken and leek pie on 24 September (RR 2.6, 95% 1.1–5.7, $P=0.016$). Multivariate analysis did not identify any exposures associated with increased risk of illness.

Environmental investigations showed the on-site kitchen where these foods were prepared, to be well managed, with no obvious concerns noted. Due to case reports of egg consumption and the frequent implication of eggs as a vehicle for foodborne salmonellosis, trace back of eggs used by the kitchen was undertaken. This revealed the eggs were produced at a New South Wales farm. Environmental sampling performed by primary industry investigators yielded a number of exact or closely related *S.* Typhimurium isolates, including those from chicken faeces, laying sheds and grading areas. The probable cause of this outbreak is transfer of *Salmonella* from eggs used in the institute kitchen, however, a precise transfer mechanism or food vehicle could not be determined.

Cluster investigations

During the quarter, OzFoodNet sites conducted investigations into 13 clusters of infection for which no common food vehicle or source of infection could be identified. Aetiological agents identified during the investigations included 8 *S.* Typhimurium clusters, and 1 cluster each of: *S.* Newport; *S.* Saintpaul; *Listeria monocytogenes*; *Cryptosporidium*; and *C. jejuni*.

Comments

The majority of reported outbreaks of gastrointestinal illness in Australia are due to person-to-person transmission, and in this quarter 81% of outbreaks (n=543) were transmitted via this route, which was comparable with the same quarter in 2012 (n=559) but 54% higher than the 5-year mean (2008–2012) of 352 outbreaks.

S. Typhimurium was identified as the aetiological agent in 10 (27%) of the 37 foodborne or suspected foodborne outbreaks during the quarter (Appendix). Of the 8 confirmed foodborne outbreaks for which an analytical and/or microbiological link to a food vehicle was established, 63% (5/8) were due to *S.* Typhimurium and associated with the consumption of raw or minimally cooked egg dishes.

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 reference laboratories for conducting sub-typing of *Salmonella* species, *Listeria monocytogenes* and other enteric pathogens and for their continuing work and advice during the quarter.

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Reference

1. OzFoodNet Working Group. OzFoodNet quarterly report, 1 July to 30 September 2013. *Commun Dis Intell* 2015;39(2):E280–284.

Appendix: Outbreaks of foodborne or suspected foodborne disease reported by OzFoodNet sites, 1 October to 31 December 2013 (n=37)

State or territory	Month*	Setting prepared	Agent responsible	Number affected	Hospitalised	Evidence	Responsible vehicles
Multi-jurisdictional	Oct	Institution	*Salmonella* Typhimurium PT 29 MLVA 03-11-10-11-523	22	0	AM	Unknown
ACT	Oct	Commercial caterer	*C. jejuni*	50	0	AM	Chicken liver pâté
NSW	Oct	Grocery store/ delicatessen	Norovirus	14	0	A	Turkey, ham and salami wraps
NSW	Oct	Bakery	*S.* Typhimurium PT 170 MLVA 03-10-07-14-523	49	21	M	Vietnamese-style rolls containing raw egg mayonnaise
NSW	Nov	Restaurant	Unknown	8	0	D	Unknown
NSW	Dec	Restaurant	Norovirus	69	0	D	Unknown
NT	Oct	Primary produce	Unknown (suspected histamine)	4	0	D	Mackerel
NT	Dec	Restaurant	Unknown	3	0	D	Unknown
Qld	Oct	Restaurant	Unknown	9	Unknown	D	Unknown
Qld	Oct	Primary produce	Norovirus	4	Unknown	D	Unknown
Qld	Oct	Primary produce	Ciguatoxin	3	0	D	Coral trout
Qld	Oct	Primary produce	Ciguatoxin	3	Unknown	D	Coral trout
Qld	Nov	Restaurant	Histamine	4	0	D	Mahi mahi
Qld	Nov	Commercial caterer	*S.* Typhimurium PT 16 MLVA 03-13-10-12-524	350	12	AM	Potato salad containing raw egg mayonnaise
Qld	Nov	Restaurant	*S.* Typhimurium PT 170/108 MLVA 03-09-07-14-524	20	5	A	Chocolate mousse containing raw egg
Qld	Nov	Primary produce	Ciguatoxin	2	0	D	Cod
Qld	Nov	Commercial caterer	Norovirus	16	0	A	Sandwiches (multiple)
Qld	Nov	Restaurant	*S.* Typhimurium MLVA 03-12-12-09-524	12	4	D	Unknown
Qld	Dec	Restaurant	*S.* Typhimurium MLVA 03-10-07-09-524	9	2	D	Unknown
Qld	Dec	Primary produce	Ciguatoxin	4	0	D	Coral trout
Qld	Dec	Primary produce	Ciguatoxin	2	0	D	Coral trout
Qld	Dec	Primary produce	Ciguatoxin	3	0	D	Coral trout
Qld	Dec	Primary produce	Ciguatoxin	2	0	D	Blue spot coral trout
SA	Oct	Restaurant	*S.* Typhimurium PT 9 MLVA 03-24-12-10-523	11	4	D	Aioli made with raw egg
SA	Nov	Camp	*Campylobacter*	23	0	D	Undercooked chicken patties
Tas.	Oct	Camp	*Shigella flexneri*	7	1	D	Unknown

Appendix (cont'd): Outbreaks of foodborne or suspected foodborne disease reported by OzFoodNet sites, 1 October to 31 December 2013

State or territory	Month*	Setting prepared	Agent responsible	Number affected	Hospitalised	Evidence	Responsible vehicles
Vic.	Oct	Restaurant	Fish wax ester	4	0	D	Rudderfish
Vic.	Oct	Restaurant	*C. coli*	4	0	D	Chicken legs stuffed with a mousse made of raw chicken mince, raw egg and cream
Vic.	Nov	Hospital	*S.* Typhimurium PT 135	27	0	M	Suspected undercooked eggs
Vic.	Nov	Restaurant	Norovirus	34	0	D	Suspected person-to-food-to-person transmission
Vic.	Nov	Private residence	*S.* Typhimurium PT 9	3	0	D	Suspected pasta carbonara containing undercooked eggs
Vic.	Nov	Aged care	*C. jejuni*	11	0		Unknown
Vic.	Nov	Private residence	*S.* Typhimurium PT 170/108	5	1	D	Unknown
Vic.	Nov	Function Centre	Norovirus	178	0	D	Suspected person-to-food-to-person transmission
WA	Oct	Restaurant	Unknown	23	0	D	Unknown
WA	Dec	Restaurant	Norovirus	8	0	D	Salad
WA	Dec	Commercial caterer	Norovirus	28	0	D	Unknown
Total				1,028	50		

* Month of outbreak is the month of onset of first case or month of notification/investigation of the outbreak.

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

A Analytical epidemiological association between illness and 1 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

AM Analytical and microbiological evidence implicating the food vehicle

MLVA Multi-locus variable number tandem repeat analysis

PT Phage type

National Notifiable Diseases Surveillance System, 1 April to 30 June 2015

A summary of diseases currently being reported by each jurisdiction is provided in Table 1. There were 58,031 notifications to the National Notifiable Diseases Surveillance System (NNDSS) between 1 April and 30 June 2015 (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 except Queensland
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
STEC, VTEC*	All jurisdictions
Salmonellosis	All jurisdictions
Shigellosis	All jurisdictions
Typhoid fever	All jurisdictions
Quarantinable diseases	
Cholera	All jurisdictions
Highly pathogenic avian influenza (human)	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	
Chlamydia	All jurisdictions
Donovanosis	All jurisdictions
Gonococcal infection	All jurisdictions
Syphilis - congenital	All jurisdictions
Syphilis <2 years duration	All jurisdictions
Syphilis >2 years or unspecified duration	All jurisdictions

Table 1 continued: Reporting of notifiable diseases by jurisdiction

Disease	Data received from:
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
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

* Infection with Shiga toxin/verotoxin-producing *Escherichia coli*.

NEC Not elsewhere classified.

Table 2: Notifications of diseases received by state and territory health authorities, 1 April to 30 June 2015, by date of diagnosis*

Disease	State or territory								Total 2nd quarter 2015	Total 1st quarter 2015	Total 2nd quarter 2014	Last 5 years mean 2nd quarter	Ratio	Year to date 2015	Last 5 years YTD mean
	ACT	NSW	NT	Qld	SA	Tas.	Vic.	WA							
Bloodborne diseases															
Hepatitis (NEC)	0	0	0	0	0	0	0	0	0	0	0	0.0	0.0	0	0.0
Hepatitis B (newly acquired)†	0	9	0	11	3	1	7	8	39	40	45	45.6	0.9	79	101.6
Hepatitis B (unspecified)‡	22	536	35	263	91	11	423	142	1,523	1,612	1,620	1,628.8	0.9	3,122	3,256.0
Hepatitis C (newly acquired)†	4	4	0	0	11	9	32	45	105	104	135	111.4	0.9	209	223.4
Hepatitis C (unspecified)‡	31	815	41	633	114	57	544	237	2,472	2,618	2,570	2,522.0	1.0	5,072	5,033.6
Hepatitis D	0	3	0	3	3	0	2	0	11	11	17	12.4	0.9	22	23.6
Gastrointestinal diseases															
Botulism	0	0	0	0	0	0	0	0	0	1	1	0.6	0.0	1	1.0
Campylobacteriosis	129	NN	85	1,616	412	198	915	652	4,007	5,562	4,663	3,701.4	1.1	9,509	8,141.8
Cryptosporidiosis	6	210	34	351	124	3	175	58	961	1,507	664	715.4	1.3	2,464	1,755.0
Haemolytic uraemic syndrome	0	4	0	0	0	0	0	0	4	4	5	3.0	1.3	8	8.6
Hepatitis A	1	10	0	2	2	1	9	3	28	88	41	40.4	0.7	116	109.2
Hepatitis E	0	2	0	0	0	0	1	0	3	10	24	11.4	0.3	13	24.4
Listeriosis	0	7	1	2	1	0	7	0	18	16	20	18.2	1.0	34	44.0
STEC, VTEC§	0	2	0	11	17	0	3	0	33	27	33	22.4	1.5	60	56.4
Salmonellosis	32	887	170	1,322	354	42	927	386	4,120	6,524	4,216	3,032.6	1.4	10,587	7,448.2
Shigellosis	0	32	28	47	18	0	109	21	255	339	209	133.2	1.9	592	326.2
Typhoid fever	1	12	0	2	2	0	9	3	29	43	22	25.0	1.2	72	76.6
Quarantinable diseases															
Cholera	0	0	0	0	1	0	0	0	1	0	1	2.0	0.5	1	2.4
Highly pathogenic avian influenza (human)	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	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	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	0.5	0.0	0	0.5

Table 2 continued: Notifications of diseases received by state and territory health authorities, 1 April to 30 June 2015, by date of diagnosis*

Disease	State or territory								Total 2nd quarter 2015	Total 1st quarter 2015	Total 2nd quarter 2014	Last 5 years mean 2nd quarter	Ratio	Year to date 2015	Last 5 years YTD mean
	ACT	NSW	NT	Qld	SA	Tas.	Vic.	WA							
Sexually transmissible infections															
Chlamydia[‖,¶]	333	5,416	665	5,115	1,311	411	8	2,782	16,041	22,407	22,149	20,514.4	0.8	38,337	41,818.0
Donovanosis	0	0	0	0	0	0	0	0	0	0	0	0.0	0.0	0	0.2
Gonococcal infection[¶]	42	1,259	450	744	244	13	1,255	523	4,530	4,700	4,010	3,415.2	1.3	9,212	6,855.4
Syphilis – congenital	0	0	1	1	0	0	0	0	2	0	0	0.8	2.5	2	2.0
Syphilis < 2 years duration[¶]	3	152	51	123	10	4	213	34	590	603	503	379.8	1.6	1,189	763.6
Syphilis > 2 years or unspecified duration[‡,¶]	3	143	26	66	25	4	184	8	459	488	470	394.6	1.2	947	761.8
Vaccine preventable diseases															
Diphtheria	0	0	0	0	0	0	0	0	0	1	0	0.8	0.0	1	1.0
Haemophilus influenzae type b	0	3	0	1	0	0	0	0	4	2	6	5.4	0.7	6	8.2
Influenza (laboratory confirmed)	129	1,848	46	2,534	1,996	133	1,882	1,128	9,696	4,600	4,592	3,870.8	2.5	14,259	5,982.8
Measles	1	3	0	3	0	0	9	3	19	27	72	33.4	0.6	46	90.8
Mumps	0	14	0	13	8	4	2	56	97	62	40	47.2	2.1	157	93.4
Pertussis	136	2,019	10	333	262	8	1,002	320	4,090	4,116	2,116	4,699.2	0.9	8,166	10,611.6
Pneumococcal disease – invasive	5	132	19	65	33	10	89	43	396	191	407	464.4	0.9	586	684.0
Poliovirus infection	0	0	0	0	0	0	0	0	0	0	0	0.0	0.0	0	0.0
Rubella	1	2	0	0	2	0	0	0	5	5	3	8.8	0.6	10	20.4
Rubella – congenital	0	0	0	0	0	0	0	0	0	0	0	0.2	0.0	0	0.2
Tetanus	0	1	0	0	0	0	0	0	1	0	0	0.6	1.7	1	2.0
Varicella zoster (chickenpox)	27	NN	23	79	76	9	201	113	528	533	488	434.4	1.2	1,058	823.2
Varicella zoster (shingles)	44	NN	80	14	629	44	366	350	1,527	1,612	1,357	1,077.8	1.4	3,126	2,178.2
Varicella zoster (unspecified)	26	NN	1	1,581	37	41	1,190	340	3,216	3,133	2,827	2,307.2	1.4	6,318	4,671.2
Vectorborne diseases															
Barmah Forest virus infection	0	69	5	94	0	0	3	15	186	271	217	578.4	0.3	456	1,278.2
Chikungunya virus infection	0	5	2	8	1	0	8	2	26	56	22	17.4	1.5	82	35.6
Dengue virus infection	0	61	22	58	22	7	52	154	376	742	522	367.8	1.0	1,110	842.8
Flavivirus infection (unspecified)	0	0	0	1	1	0	0	0	2	4	8	3.6	0.6	6	8.4
Japanese encephalitis virus infection	0	0	0	0	0	0	0	0	0	2	0	0.4	0.0	2	0.6
Kunjin virus infection**	0	0	0	0	0	0	0	0	0	0	1	0.6	0.0	0	0.8
Malaria	1	8	1	15	0	1	10	11	47	64	85	86.0	0.5	110	190.4
Murray Valley encephalitis virus infection**	0	0	1	0	0	0	0	0	1	1	0	1.4	0.7	2	3.2
Ross River virus infection	3	423	62	1,087	27	2	88	305	1,997	5,745	1,306	1,337.4	1.5	7,723	3,325.0

Table 2 continued: Notifications of diseases received by state and territory health authorities, 1 April to 30 June 2015, by date of diagnosis

Disease	State or territory								Total 2nd quarter 2015	Total 1st quarter 2015	Total 2nd quarter 2014	Last 5 years mean 2nd quarter	Ratio	Year to date 2015	Last 5 years YTD mean
	ACT	NSW	NT	Qld	SA	Tas.	Vic.	WA							
Zoonoses															
Anthrax	0	0	0	0	0	0	0	0	0	0	0	0.0	0.0	0	0.2
Australian bat lyssavirus infection	0	0	0	0	0	0	0	0	0	0	0	0.0	0.0	0	0.2
Brucellosis	0	4	0	1	0	0	0	0	5	4	3	4.6	1.1	9	11.4
Leptospirosis	0	2	1	12	0	0	2	0	17	23	32	41.0	0.4	40	87.0
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	1	0	1	0	2	1	6	12.6	0.2	3	25.4
Q fever	0	47	1	63	2	0	11	3	127	153	119	103.8	1.2	279	205.2
Tularaemia	0	0	0	0	0	0	0	0	0	0	0	0.0	0.0	0	0.3
Other bacterial infections															
Legionellosis	0	27	3	24	3	2	22	17	98	95	112	100.6	1.0	192	186.8
Leprosy	0	1	0	0	0	0	1	0	2	1	4	3.4	0.6	3	4.8
Meningococcal infection – invasive††	0	13	1	8	7	0	15	3	47	29	41	49.4	1.0	74	89.4
Tuberculosis	5	117	11	45	6	3	70	31	288	285	301	290.6	1.0	569	608.4
Total	985	14,302	1,876	16,351	5,856	1,018	9,847	7,796	58,031	68,462	56,105			126,042	

* 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 reports hepatitis C newly acquired 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. From 1 July 2013 case definition changed to exclude all ocular infections.

¶ The national case definitions for chlamydial, 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).

** In the Australian Capital Territory, Murray Valley encephalitis virus infection and Kunjin virus infection are combined under Murray Valley encephalitis virus infection.

†† 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 2015, by state or territory. (Annualised rate per 100,000 population)*,†

Disease	State or territory								
	ACT	NSW	NT	Qld	SA	Tas.	Vic.	WA	Aust.
Bloodborne diseases									
Hepatitis (NEC)	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
Hepatitis B (newly acquired)‡	0.0	0.5	0.0	0.9	0.7	0.8	0.5	1.2	0.7
Hepatitis B (unspecified)§	22.8	28.5	57.2	22.3	21.6	8.5	29.0	22.1	26.0
Hepatitis C (newly acquired)‡	4.2	0.2	0.0	0.0	2.6	7.0	2.2	7.0	1.8
Hepatitis C (unspecified)§	32.2	43.4	67.0	53.6	27.1	44.3	37.3	37.0	42.1
Hepatitis D	0.0	0.2	0.0	0.3	0.7	0.0	0.1	0.0	0.2
Gastrointestinal diseases									
Botulism	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
Campylobacteriosis	133.9	NN	139.0	136.9	97.8	153.9	62.7	101.7	100.4
Cryptosporidiosis	6.2	11.2	55.6	29.7	29.4	2.3	12.0	9.0	16.4
Haemolytic uraemic syndrome	0.0	0.2	0.0	0.0	0.0	0.0	0.0	0.0	0.1
Hepatitis A	1.0	0.5	0.0	0.2	0.5	0.8	0.6	0.5	0.5
Hepatitis E	0.0	0.1	0.0	0.0	0.0	0.0	0.1	0.0	0.1
Listeriosis	0.0	0.4	1.6	0.2	0.2	0.0	0.5	0.0	0.3
STEC, VTEC‖	0.0	0.1	0.0	0.9	4.0	0.0	0.2	0.0	0.6
Salmonellosis	33.2	47.2	278.0	112.0	84.0	32.6	63.5	60.2	70.2
Shigellosis	0.0	1.7	45.8	4.0	4.3	0.0	7.5	3.3	4.3
Typhoid fever	1.0	0.6	0.0	0.2	0.5	0.0	0.6	0.5	0.5
Quarantinable diseases									
Cholera	0.0	0.0	0.0	0.0	0.2	0.0	0.0	0.0	0.0
Human pathogenic avian influenza (human)	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	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	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
Sexually transmitted infections									
Chlamydia¶,**	345.5	288.2	1,087.4	433.4	311.1	319.4	0.5	433.8	273.3
Donovanosis	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
Gonococcal infection**	43.6	67.0	735.9	63.0	57.9	10.1	86.0	81.5	77.2
Syphilis – congenital	0.0	0.0	1.6	0.1	0.0	0.0	0.0	0.0	0.0
Syphilis < 2 years duration**	3.1	8.1	83.4	10.4	2.4	3.1	14.6	5.3	10.1
Syphilis > 2 years or unspecified duration§,**	3.1	7.6	42.5	5.6	5.9	3.1	12.6	1.2	7.8
Vaccine preventable diseases									
Diphtheria	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
Haemophilus influenzae type b	0.0	0.2	0.0	0.1	0.0	0.0	0.0	0.0	0.1
Influenza (laboratory confirmed)	133.9	98.4	75.2	214.7	473.7	103.4	128.9	175.9	165.2
Measles	1.0	0.2	0.0	0.3	0.0	0.0	0.6	0.5	0.3
Mumps	0.0	0.7	0.0	1.1	1.9	3.1	0.1	8.7	1.7
Pertussis	141.1	107.5	16.4	28.2	62.2	6.2	68.6	49.9	69.7
Pneumococcal disease – invasive	5.2	7.0	31.1	5.5	7.8	7.8	6.1	6.7	6.7
Poliovirus infection	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
Rubella	1.0	0.1	0.0	0.0	0.5	0.0	0.0	0.0	0.1
Rubella – congenital	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
Tetanus	0.0	0.1	0.0	0.0	0.0	0.0	0.0	0.0	0.0

Table 3 continued: Notification rates of diseases, 1 April to 30 June 2015, by state or territory. (Annualised rate per 100,000 population)*,†

Disease	State or territory								Aust.
	ACT	NSW	NT	Qld	SA	Tas.	Vic.	WA	
Vaccine preventable diseases, *cont'd*									
Varicella zoster (chickenpox)	28.0	NN	37.6	6.7	18.0	7.0	13.8	17.6	13.2
Varicella zoster (shingles)	45.7	NN	130.8	1.2	149.3	34.2	25.1	54.6	38.3
Varicella zoster (unspecified)	27.0	NN	1.6	133.9	8.8	31.9	81.5	53.0	80.6
Vectorborne diseases									
Barmah Forest virus infection	0.0	3.7	8.2	8.0	0.0	0.0	0.2	2.3	3.2
Chikungunya virus infection	0.0	0.3	3.3	0.7	0.2	0.0	0.5	0.3	0.4
Dengue virus infection	0.0	3.2	36.0	4.9	5.2	5.4	3.6	24.0	6.4
Flavivirus infection (unspecified)	0.0	0.0	0.0	0.1	0.2	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
Kunjin virus infection††	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
Malaria	1.0	0.4	1.6	1.3	0.0	0.8	0.7	1.7	0.8
Murray Valley encephalitis virus infection††	0.0	0.0	1.6	0.0	0.0	0.0	0.0	0.0	0.0
Ross River virus infection	3.1	22.5	101.4	92.1	6.4	1.6	6.0	47.6	34.0
Zoonoses									
Anthrax	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
Australia 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	0.1	0.0	0.0	0.0	0.0	0.1
Leptospirosis	0.0	0.1	1.6	1.0	0.0	0.0	0.1	0.0	0.3
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	0.2	0.0	0.1	0.0	0.0
Q fever	0.0	2.5	1.6	5.3	0.5	0.0	0.8	0.5	2.2
Tularaemia	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
Other bacterial diseases									
Legionellosis	0.0	1.4	4.9	2.0	0.7	1.6	1.5	2.7	1.7
Leprosy	0.0	0.1	0.0	0.0	0.0	0.0	0.1	0.0	0.0
Meningococcal infection – invasive‡‡	0.0	0.7	1.6	0.7	1.7	0.0	1.0	0.5	0.8
Tuberculosis	5.2	6.2	18.0	3.8	1.4	2.3	4.8	4.8	4.9

* 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 reports hepatitis C newly acquired under hepatitis C 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. From 1 July 2013 case definition changed to exclude all ocular infections.

** The national case definitions for chlamydial, 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).

†† In the Australian Capital Territory, Murray Valley encephalitis virus infection and Kunjin virus infection are combined under Murray Valley encephalitis virus infection.

‡‡ 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.

Australian childhood immunisation coverage, assessed as at 31 March 2015

Brynley P Hull for the National Centre for Immunisation Research and Surveillance of Vaccine Preventable Diseases

Introduction

The National Centre for Immunisation Research and Surveillance of Vaccine Preventable Diseases (NCIRS) provides commentary on the trends in ACIR data. For further information please contact NCIRS at: telephone +61 2 9845 1435, email: brynley.hull@health.nsw.gov.au

Tables 1, 2 and 3 provide the latest quarterly report on childhood immunisation coverage from the Australian Childhood Immunisation Register (ACIR) for all children.

The data show the percentage of all children 'fully immunised' at 12 months, 24 months and 60 months, for four 3-month birth cohorts of children assessed at the stated ages between January and December 2014 using ACIR data as at 31 March 2015. 'Fully immunised' refers to vaccines on the National Immunisation Program Schedule, but excludes rotavirus, and is outlined in more detail below.

'Fully immunised' at 12 months of age is defined as a child having a record on the ACIR of three doses of a diphtheria (D), tetanus (T) and pertussis-containing (P) vaccine, 3 doses of polio vaccine, 2 or 3 doses of PRP-OMP containing *Haemophilus influenzae* type b (Hib) vaccine or 3 doses of any other Haemophilus influenzae type b (Hib) vaccine, 3 doses of hepatitis B vaccine, and 3 doses of 13-valent pneumococcal conjugate vaccine. 'Fully immunised' at 24 months of age is defined as a child having a record on the ACIR of 3 doses of a DTP-containing vaccine, 3 doses of polio vaccine, 3 or 4 doses of PRP-OMP Hib vaccine or 4 doses of any other Hib vaccine, 3 doses of hepatitis B vaccine, 1 dose of a measles, mumps and rubella-containing (MMR) vaccine, 1 dose of meningococcal C vaccine, and 1 dose of varicella vaccine. 'Fully immunised' at 60 months of age is defined as a child having a record on the ACIR of 4 doses of a DTP-containing vaccine, 4 doses of polio vaccine, and 2 doses of an MMR-containing vaccine.

A full description of the basic methodology used can be found in *Commun Dis Intell* 1998;22(3):36–37.

Results

The rolling annualised percentage of children 'fully immunised' by 12 months of age for Australia increased marginally from the previous quarter by 0.3 of a percentage point to 91.0% (Table 1). All jurisdictions experienced small increases in the percentage of children 'fully immunised' by 12 months of age. For individual vaccines due by 12 months of age all jurisdictions achieved coverage greater than 90%.

The rolling annualised percentage of children 'fully immunised' by 24 months of age for Australia decreased from the previous quarter by 1.1 percentage points to 90.1 (Table 2) This drop is likely to be due to the inclusion of the meningococcal C and varicella vaccines into the algorithm to calculate fully immunised coverage for this age group from the December 2014 quarter onwards. All jurisdictions experienced similar decreases

Table 1: Percentage of children immunised at 12 months of age, preliminary results by vaccine and state or territory for the birth cohort 1 January 2013 to 31 December 2013

	State or territory								
Vaccine	ACT	NSW	NT	Qld	SA	Tas	Vic	WA	Aust
Total number of children	5,598	98,764	3,860	62,691	19,857	5,967	75,919	34,050	306,706
Diphtheria, tetanus, pertussis (%)	93.9	91.4	90.5	92.3	91.4	91.2	92.0	92.1	91.8
Poliomyelitis (%)	93.9	91.3	90.5	92.3	91.4	91.1	92.0	92.1	91.8
Haemophilus influenzae type b (%)	93.6	91.2	90.4	92.2	91.3	91.0	91.8	91.9	91.7
Hepatitis B (%)	93.5	91.0	90.4	92.1	91.2	90.9	91.6	91.7	91.5
Pneumococcal	93.7	91.0	90.5	92.1	91.1	91.0	91.6	91.6	91.5
Fully immunised (%)	93.0	90.5	90.0	91.8	90.7	90.4	91.0	91.1	91.0

in fully immunised coverage for this age group. Coverage for individual vaccines due by 24 months remained high in all jurisdictions, except that coverage in all jurisdictions again decreased for the measles, mumps and rubella vaccine (by 0.7 to 1.3 percentage points). This is likely due to the introduction of the MMRV vaccine onto the National Immunisation Program Schedule in July 2013.

The rolling annualised percentage of children 'fully immunised' by 60 months of age for Australia increased marginally from the previous quarter by 0.1 of a percentage point to 92.2% (Table 3). This maintains the improvement in coverage for this age milestone. There were also only marginal changes in fully immunised coverage at 60 months of age in all jurisdictions. Coverage for individual vaccines due by 60 months remained greater than 90% in all jurisdictions.

Acknowledgment

These data were provided by Medicare Australia, to specifications provided by the Australian Government Department of Health.

Figure: Trends in vaccination coverage, Australia, 1997 to 31 December 2014, by age cohorts

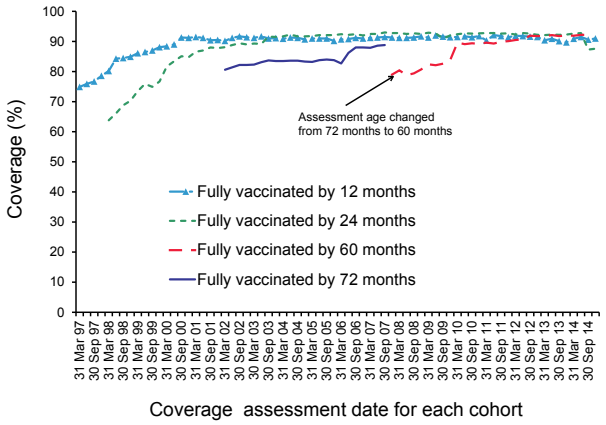


Table 2: Percentage of children immunised at 24 months of age, preliminary results by vaccine and state or territory for the birth cohort 1 January 2012 to 31 December 2012

Vaccine	State or territory								
	ACT	NSW	NT	Qld	SA	Tas	Vic	WA	Aust
Total number of children	5,544	101,638	3,668	63,503	20,163	5,892	76,780	34,257	311,445
Diphtheria, tetanus, pertussis (%)	96.0	94.9	95.3	95.0	94.7	95.1	95.5	94.5	95.0
Poliomyelitis (%)	96.0	94.8	95.3	95.0	94.7	95.0	95.4	94.5	95.0
Haemophilus influenzae type b (%)	94.9	93.5	94.9	94.2	93.4	93.5	94.2	93.3	93.8
Measles, mumps, rubella (%)	93.8	91.9	93.3	92.7	91.5	91.3	92.3	90.7	92.0
Hepatitis B (%)	95.6	94.5	95.3	94.6	94.3	94.8	95.0	93.9	94.6
Meningococcal C (%)	94.4	93.1	94.0	93.9	92.9	93.2	93.6	92.7	93.4
Varicella (%)	94.2	90.8	90.6	91.4	90.0	88.6	91.6	89.5	91.0
Fully immunised (%)	92.1	89.6	90.9	91.3	89.4	88.7	90.4	88.8	90.1

Table 3: Percentage of children immunised at 60 months of age, preliminary results by vaccine and state or territory for the birth cohort 1 January 2009 to 31 December 2009

Vaccine	State or territory								
	ACT	NSW	NT	Qld	SA	Tas	Vic	WA	Aust
Total number of children	5,382	100,561	3,559	64,904	20,337	6,418	75,318	33,956	310,435
Diphtheria, tetanus, pertussis (%)	94.1	93.1	92.8	92.8	91.5	92.9	93.1	91.2	92.7
Poliomyelitis (%)	94.1	93.0	92.8	92.7	91.4	92.9	93.0	91.1	92.7
Measles, mumps, rubella (%)	93.8	93.0	93.2	92.7	91.4	92.8	93.0	91.0	92.6
Fully immunised (%)	93.5	92.5	92.2	92.3	90.8	92.2	92.5	90.5	92.2

Australian Meningococcal Surveillance Programme quarterly report, 1 April to 30 June 2015

Monica M Lahra, Ratan Kundu for the Australian Meningococcal Surveillance Programme

Introduction

The reference laboratories of the Australian Meningococcal Surveillance Programme (AMSP) report data on the number of cases confirmed by laboratory testing using culture and by non-culture based techniques. Culture positive cases, where *Neisseria meningitidis* is grown from a normally sterile site or skin lesions, and non-culture based diagnoses, derived from results of nucleic acid amplification assays and serological techniques, are defined as invasive meningococcal disease (IMD) according to Public Health Laboratory Network definitions. Data contained in quarterly reports are restricted to a description of the number of cases by jurisdiction and serogroup, where known. Some minor corrections to data in the Table may be made in subsequent reports if additional data are received. A full analysis of laboratory confirmed cases of IMD in each calendar year is contained in the AMSP annual reports published in *Communicable Diseases Intelligence*. For more information see *Commun Dis Intell* 2015;39(1):E179.

Results

Laboratory confirmed cases of invasive meningococcal disease for the period 1 April to 30 June 2015 are shown in the Table.

Table: Number of laboratory confirmed cases of invasive meningococcal disease, Australia, 1 April to 30 June 2015, by serogroup and state or territory

State or territory	Year	Serogroup													
		A		B		C		Y		W135		ND		All	
		Q2	YTD	Q2	YTD	Q2	YTD	Q2	YTD	Q2	YTD	Q2	YTD	Q2	YTD
Australian Capital Territory	2015	0	0	0	0	0	0	0	0	0	0	0	0	0	0
	2014	0	0	0	0	0	0	0	0	0	0	0	0	0	0
New South Wales	2015	0	0	9	12	0	1	2	2	0	1	1	1	12	17
	2014	0	0	7	10	0	0	6	6	1	3	0	0	14	19
Northern Territory	2015	0	0	1	1	0	0	0	0	0	0	0	0	1	1
	2014	0	0	0	2	0	0	0	0	0	0	0	0	0	2
Queensland	2015	0	0	8	13	0	0	0	1	2	2	0	0	10	16
	2014	0	0	7	13	0	0	0	0	0	1	2	2	9	16
South Australia	2015	0	0	4	6	0	0	0	0	0	0	0	0	4	6
	2014	0	0	4	9	0	0	0	0	0	0	0	0	4	9
Tasmania	2015	0	0	0	0	0	0	0	0	0	0	0	0	0	0
	2014	0	0	0	0	0	0	0	0	0	0	0	0	0	0
Victoria	2015	0	0	8	15	0	0	2	2	5	6	0	1	15	24
	2014	0	0	5	8	0	0	1	1	1	2	0	0	7	11
Western Australia	2015	0	0	2	4	0	0	1	1	0	0	0	0	3	5
	2014	0	0	6	7	0	2	0	0	1	1	0	0	7	10
Total	2015	0	0	32	51	0	1	5	6	7	9	1	2	45	69
	2014	0	0	29	49	0	2	7	7	3	7	2	2	41	67

Australian Sentinel Practices Research Network, 1 April to 30 June 2015

Monique B-N Chilver, Daniel Blakeley, Nigel P Stocks for the Australian Sentinel Practices Research Network

Introduction

The Australian Sentinel Practices Research Network (ASPREN) is a national surveillance system that is funded by the Australian Government Department of Health, owned and operated by the Royal Australian College of General Practitioners and directed through the Discipline of General Practice at the University of Adelaide.

The network consists of general practitioners who report 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 alert public health officials of epidemics in their early stages as well as play a role in the evaluation of public health campaigns and research of conditions commonly seen in general practice. Electronic, web-based data collection was established in 2006.

Since 2010, ASPREN GPs have been collecting nasal swab samples for laboratory testing, allowing for viral testing of 20% of influenza-like illness (ILI) patients for a range of respiratory viruses including influenza A, influenza B and A(H1N1) pdm09.

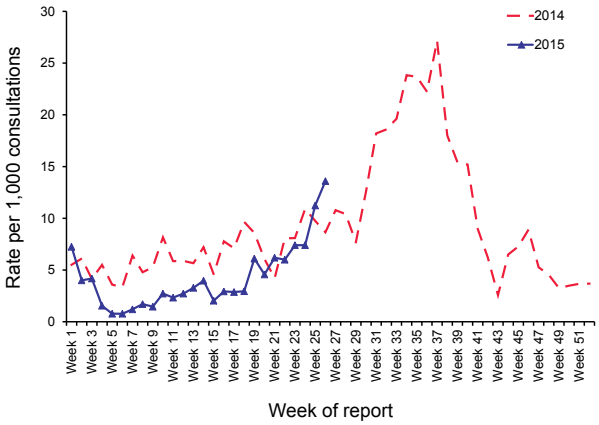
The list of conditions reported is reviewed annually by the ASPREN management committee. In 2015, 4 conditions are being monitored. They include ILI, gastroenteritis and varicella infections (chickenpox and shingles). Definitions of these conditions are described in Surveillance systems reported in CDI, published in *Commun Dis Intell* 2015;39(1):E180.

Results

Sentinel practices contributing to ASPREN were located in all 8 states and territories in Australia. A total of 241 general practitioners regularly contributed data to ASPREN in the 2nd quarter of 2015. Each week an average of 201 general practitioners provided information to ASPREN at an average of 16,862 (range 13,411 to 18,560) consultations per week and an average of 163 (range 96 to 262) notifications per week.

ILI rates reported from 1 April to 30 June 2015 averaged 6 cases per 1,000 consultations (range 2–13 cases per 1,000 consultations). This was lower compared with rates in the same reporting period in 2014, which averaged 8 cases per 1,000 consultations (range 4–11 cases per 1,000 consultations, Figure 1). ILI rates sharply increased above baseline in week 25.

Figure 1: Consultation rates for influenza-like illness, ASPREN, 2014 and 1 January to 30 June 2015, by week of report



The ASPREN ILI swab testing program continued in 2015 with 746 tests being undertaken from 1 April to 30 June. The most commonly reported virus during this period was rhinovirus (16.1% of all swabs performed, Table), with the 2nd most common virus being influenza B (10.3% of all swabs performed).

From the beginning of 2015 to the end of week 27, 132 cases of influenza were detected with 84 of these typed as influenza B (9.1% of all swabs performed) and the remaining 48 being influenza A (5.2% of all swabs performed) (Table). Overall respiratory virus positivity was 52% compared to 43% for the same period last year.

During this reporting period, consultation rates for gastroenteritis averaged 3 cases per 1,000 consultations (range 2–5 cases per 1,000, Figure 2). This was slightly lower than the rates in the same reporting period in 2014 where the average was 5 cases per 1,000 consultations (range 3–6 cases per 1,000).

Table: Influenza-like illness swab testing results, ASPREN, 1 January to 30 June 2015, by week of report

Week ending	Influenza A %	Influenza B %	RSV %	Para-influenza virus type 1 %	Para-influenza virus type 2 %	Para--influenza virus type 3 %	Adenovirus %	Rhinovirus %	Metapneumovirus %	*Mycoplasma pneumoniae* %	Pertussis %	Proportion positive for Influenza %
4 Jan	0	0	0	0	0	0	0	25	0	0	0	0
11 Jan	50	0	0	0	50	0	0	0	0	0	0	50
18 Jan	0	13	13	0	0	13	0	0	0	0	0	13
25 Jan	0	11	0	0	0	33	0	11	11	0	0	11
1 Feb	13	38	0	0	13	0	13	0	0	0	0	50
8 Feb	0	0	0	0	0	0	50	25	0	0	0	0
15 Feb	14	0	0	0	14	0	0	7	7	0	0	14
22 Feb	10	0	0	0	10	0	0	14	0	0	0	10
1 Mar	8	15	8	0	0	8	0	23	0	0	0	23
8 Mar	14	0	0	0	9	0	0	18	5	5	0	14
15 Mar	10	0	0	0	10	5	0	10	0	5	0	10
22 Mar	13	0	0	4	4	0	0	22	4	0	0	13
29 Mar	0	0	0	0	0	0	0	23	5	0	0	0
5 Apr	15	0	5	0	10	0	0	5	10	5	0	15
12 Apr	10	0	0	0	5	5	5	25	0	0	0	10
19 Apr	0	9	14	0	0	5	5	9	0	0	0	9
26 Apr	3	0	0	3	0	0	0	20	0	0	0	3
3 May	8	3	5	0	3	3	0	14	0	3	0	11
10 May	4	11	9	0	2	5	5	13	2	0	0	14
17 May	7	20	5	2	5	5	4	14	2	2	0	27
24 May	0	13	3	0	2	5	0	20	2	0	0	13
31 May	0	10	10	0	2	5	5	23	3	0	0	10
7 Jun	4	9	6	0	4	10	6	19	3	1	0	13
14 Jun	6	13	4	1	4	6	7	17	1	1	0	20
21 Jun	6	7	9	0	2	1	4	14	3	0	0	13
28 Jun	3	15	10	0	2	12	4	14	2	1	0	18
Total proportion positive by virus	5.2	9.2	5.8	0.4	3.4	5.3	3.5	15.9	2.3	0.9	0.0	14

Figure 2: Consultation rates for gastroenteritis, ASPREN, 2014 and 1 January to 30 June 2015, by week of report

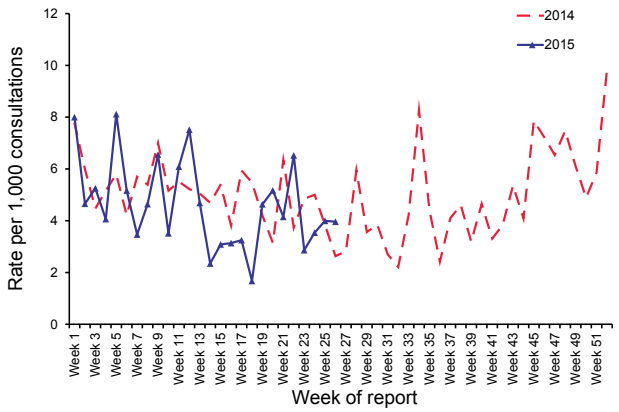
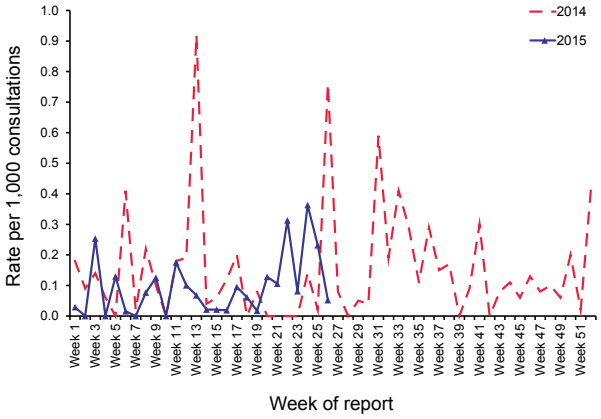


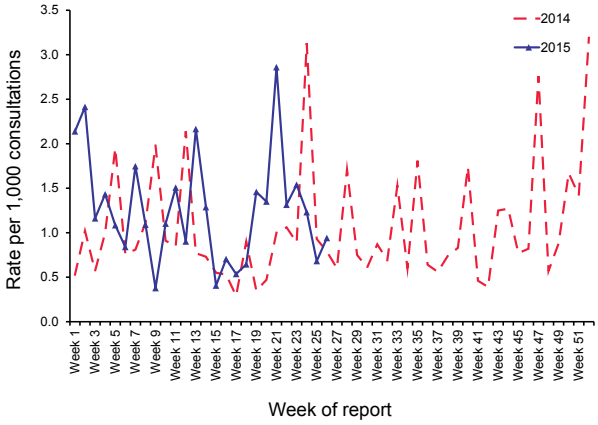
Figure 3: Consultation rates for chickenpox, ASPREN, 2014 and 1 January to 30 June 2015, by week of report



Varicella infections were reported at a similar rate for the 2nd quarter of 2015 compared with the same period in 2014. From 1 April to 30 June 2015, recorded rates for chickenpox averaged 0.11 cases per 1,000 consultations (range 0.02–0.31 cases per 1,000 consultations, Figure 3).

In the 2nd quarter of 2015, reported rates for shingles averaged 0.92 cases per 1,000 consultations (range 0.46–2.49 cases per 1,000 consultations, Figure 4), which was slightly higher compared with the same reporting period in 2014 where the average shingles rate was 0.89 cases per 1,000 consultations (range 0.29–3.13 cases per 1,000 consultations).

Figure 4: Consultation rates for shingles, ASPREN, 2014 and 1 January to 30 June 2015, by week of report



Invasive pneumococcal disease surveillance Australia, 1 April to 30 June 2015

Rachel de Kluyver 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 2nd quarter of 2015 was more than the previous quarter but less than the number of notified cases in the 2nd quarter of 2014. 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.

Key points

In the 2nd quarter of 2015, there were 397 cases of IPD reported to the National Notifiable Diseases Surveillance Scheme. This was a 2% reduction on the number of cases reported for the same period in 2014 (n=407) (Table 1). Most common serotypes affect all age groups, with serotype 19A continuing to be the most common cause of IPD overall (Table 2).

In non-Indigenous Australians, the number of notified cases was highest in the under 5 years age group followed by the over 85 years age group. In Indigenous Australians, notified cases were highest in the under 5 years age group followed by the 50–54 years age group (Table 3). Compared with the second quarter of 2014 (http://www.health.gov.au/internet/main/publishing.nsf/Content/cda-cdi3803l.htm), the proportion of cases reported as Indigenous increased from 11% to 15%. Data completeness was comparable at 94% for both periods.

There were 67 cases of IPD reported in children aged under 5 years, of which 42% (n=25) were due to a serotype included in either the 7vPCV or the 13vPCV (Figure 1). Serotype 19A, which is included in the 13vPCV, continued to be the most common serotype affecting this age group (Table 2). The number of cases in this age group was 10% higher than the 2nd quarter of 2014 (n=61) while the serotype distribution remained similar.

There were 21 cases of IPD reported in Indigenous Australians aged 50 years or over. Of those cases with a reported serotype, 57% (n=12) were due to a serotype included in the 23-valent polysaccharide pneumococcal vaccine (23vPPV) (Figure 2). The number of notified cases of IPD in this age group was almost 40% higher than in the 2nd quarter of

Table 1: Notified cases of invasive pneumococcal disease, Australia, 1 April to 30 June 2015, by Indigenous status, serotype completeness and state or territory

	ACT	NSW	NT	Qld	SA	Tas.	Vic.	WA	Total Qtr 2 2015	Total Qtr 1 2015	Total Qtr 2 2014	Year to date 2015
Indigenous status												
Indigenous	0	8	17	13	4	0	2	16	60	33	46	60
Non-Indigenous	5	99	2	50	29	10	65	27	287	129	315	287
Not stated/ unknown	0	26	0	2	0	0	22	0	50	28	46	50
Total	5	133	19	65	33	10	89	43	397	190	407	587
Indigenous status completeness* (%)	100	80	100	97	100	100	75	100	87	–	–	–
Serotype completeness† (%)	100	86	100	94	73	100	97	100	91	–	–	–

* Indigenous status completeness is defined as the reporting of a known Indigenous status, excluding the reporting of not stated or unknown Indigenous status.

† Serotype completeness is the proportion of all cases of invasive pneumococcal disease that were reported with a serotype or reported as non-typable. Serotype incompleteness may include when 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; the isolate was not referred to the reference laboratory or was not viable; typing was pending at the time of reporting, or no serotype was reported by the notifying jurisdiction to the National Notifiable Diseases Surveillance System.

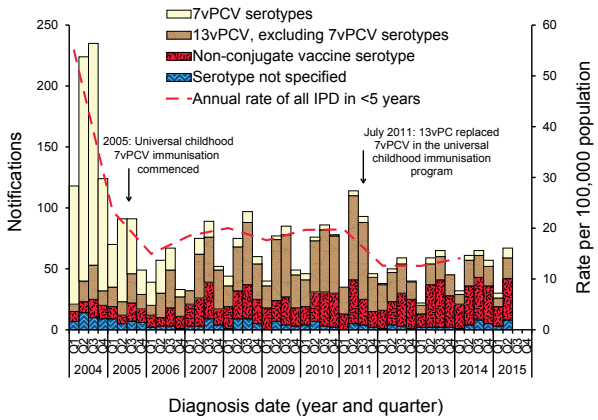
Table 2: Frequently notified serotypes of invasive pneumococcal disease, Australia, 1 April to 30 June 2015, by age group

Serotype	Age group			Serotype total*
	Under 5 years	5 to 64 years	Over 65 years	
19A	11	17	10	38
3	6	12	7	25
9N	4	13	8	25
22F	2	13	7	22
23B	4	11	5	20
7F		13	4	17
8		14	3	17
19F	7	4	4	15
35B	2	8	5	15
11A	2	7	4	13
15A		6	7	13
16F	3	3	7	13
23A	1	1	11	13
12F		6	4	10
33F		9	1	10
38	3	6	1	10
15B	1	4	3	8
15C	3	1	3	7
6C	1	5	1	7
10A	1	2	3	6
4		5		5
Other	6	27	19	52
Serotype unknown†	10	16	10	36
Total	67	203	127	397

* Serotypes that only occur in less than 5 cases per quarter are grouped as 'Other' and include 'non-typable' samples this quarter.

† 'Serotype unknown' includes those serotypes reported as 'no isolate', 'not referred', 'not viable', 'typing pending' and 'untyped'.

Figure 1: Notifications (2004 to 30 June 2015) and annual rates (2004 to 2014) of invasive pneumococcal disease in children aged less than 5 years, Australia, by vaccine serotype group



2014 (n=13) and double that of the previous quarter (n=10). Compared with the previous quarter, the proportion of 23vPPV serotypes increased from 44% to 57% of cases with a reported serotype.

There were 124 cases of IPD reported in non-Indigenous Australians aged 65 years or over. Of those cases with a reported serotype, 50% (n=62) were due to a serotype included in the 23vPPV (Figure 3). The number of notified cases of IPD in this age group was 7% higher than in the 2nd quarter of 2014 (n=115) and more than double that of the previous quarter (n=56). Compared with the previous quarter, the proportion of IPD due to 23vPPV serotypes increased from 41% to 55% of cases with a reported serotype.

In this quarter, there were 22 deaths attributed to 13 different IPD serotypes, which was similar

Figure 2: Notifications (2004 to 30 June 2015) and annual rates of all invasive pneumococcal disease (2004 to 2014) in Indigenous Australians aged 50 years or over, Australia, by vaccine serotype group

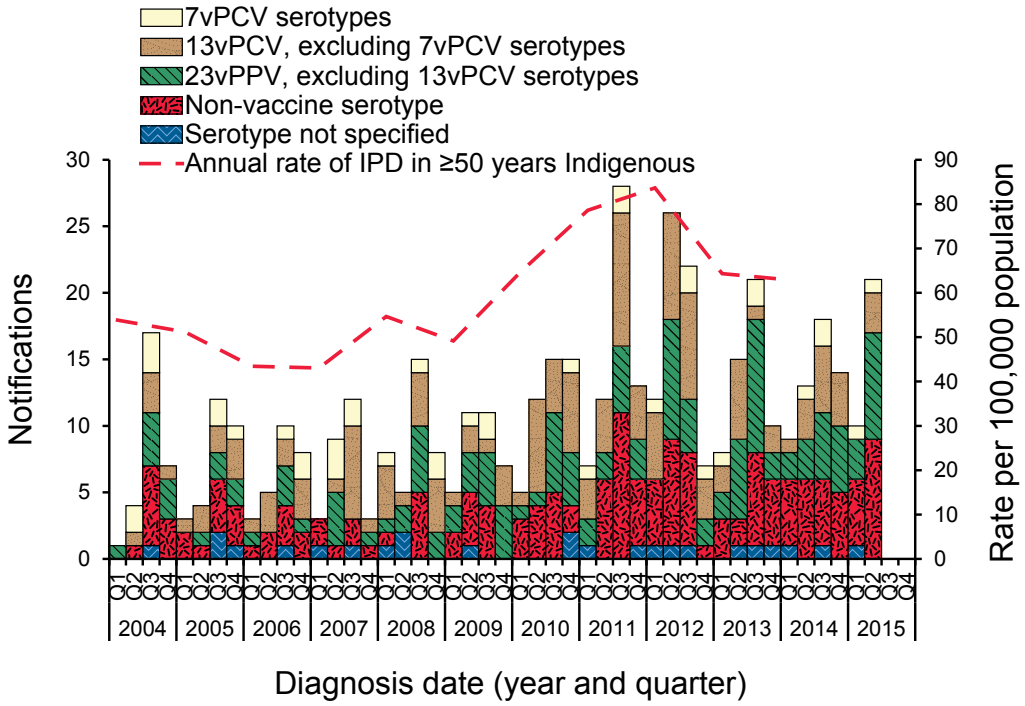


Table 3: Notified cases of invasive pneumococcal disease, Australia, 1 April to 30 June 2015, by Indigenous status and age group

Age group	Indigenous status			Total
	Indigenous	Non-Indigenous	Not reported	
0–4	12	51	4	67
5–9	3	7	4	14
10–14	2	1	2	5
15–19	2	1	2	5
20–24	3	1	1	5
25–29	3	2	3	8
30–34	4	2	4	10
35–39	1	11	6	18
40–44	6	6	9	21
45–49	3	7	7	17
50–54	9	22	3	34
55–59	5	26	2	33
60–64	4	27	2	33
65–69	1	20		21
70–74		27		27
75–79	1	19		20
80–84	1	23		24
85+		34	1	35
Total	60 (15%)	287 (72%)	50 (12%)	397

to the same quarter in 2014 (n=21). There was 1 death reported in a child aged under 5 years, which was associated with serotype 6C.

During this reporting period, the Northern Territory notified a case of IPD in a 43-year-old female due to serotype 32F. This is the first time this globally rare serotype has been identified in Australia.[1]

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*.

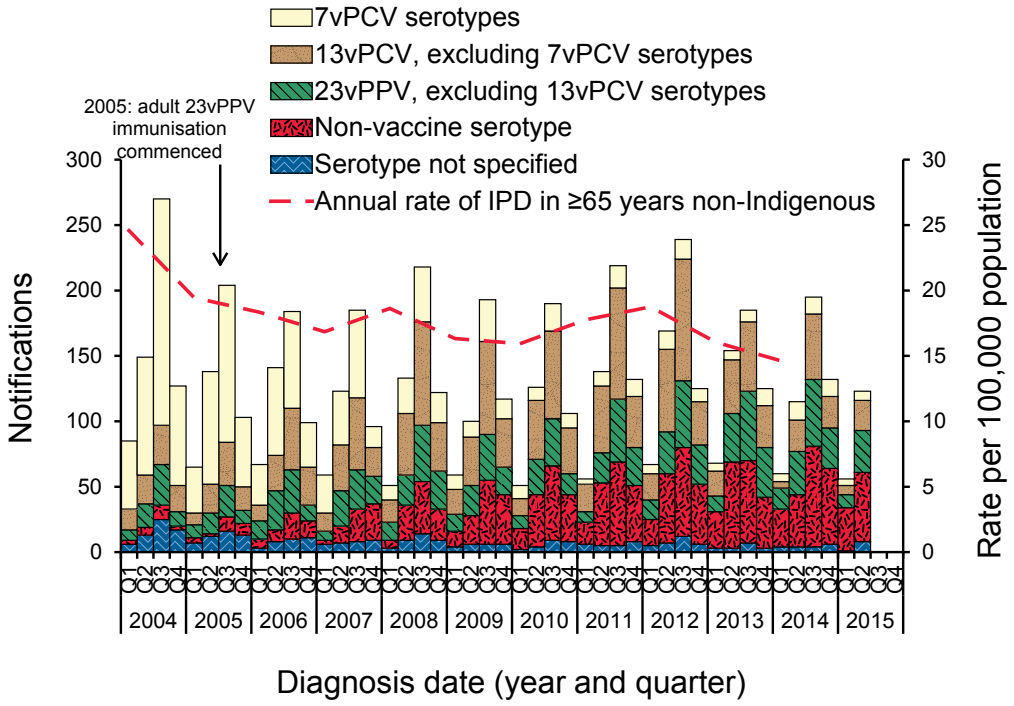
In Australia, pneumococcal vaccination is recommended as part of routine immunisation for children, the medically at risk and older Australians. More information on the scheduling of the pneumococcal vaccination can be found on the Immunise Australia Program website (www.immunise.health.gov.au).

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.

Table 4: *Streptococcus pneumoniae* serotypes targeted by pneumococcal vaccines

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

Figure 3: Notifications (2004 to 30 June 2015) and annual rates of all invasive pneumococcal disease (2004 to 2014) in non-Indigenous Australians aged 65 years or over, Australia, by vaccine serotype group



Acknowledgements

Report compiled by Dr Rachel de Kluyver on behalf of the Enhanced Invasive Pneumococcal Disease Surveillance Working Group.

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Reference

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Conference abstracts

Communicable Disease Control Conference 2015

An era of change: global threats and harnessing new technologies

1–2 June 2015, Brisbane

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E506 Speaker: Professor Peter Horby, Associate Professor, Group Leader Epidemic Research Group Oxford (ERGO), Group Head / Principal Investigator and Fellow, Nuffield Department of Medicine, University of Oxford, UK

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E567 Authors: C Sotomayor, Q Wang, P Howard and V Sintchenko

E568 Authors: L Garton, TW Yip, M Gunathilake, JY Su, A Ishwar, J Creighton, R Sherry, A Hope, C Beatson, H Goodwin, N Ryder, M Thalanany, V Krause

E568 Authors: LM Nissen, ETL Lau, C Campbell, H Kastrissios, BD Glass, A Drovandi and M Rosenthal

E569 Presenter and author: Neil Franklin, Health Protection NSW

E569 Authors: C Mills, R Kremer, G Caleo and K Lokuge

These abstracts are provided unedited

Day 1: Controlling global infectious threats

Plenary 1: Global threats from infectious diseases
Chair: Professor Jeremy McAnulty

Novel epidemiological approaches to surveillance of emerging infectious disease threats in the Asia–Pacific region

Speaker: Professor Archie Clements, Director and Professor, Research School of Population Health, ANU College of Medicine, Biology and Environment, The Australian National University, Canberra

The Asia–Pacific region is the global epicentre of emerging infectious diseases, and novel approaches to surveillance are required to effectively mitigate the growing and high potential burden of these diseases. With climate change, predictions of increasing frequency and severity of cyclones in the Pacific might increase flooding risk, exacerbating the disease burden from emerging disease such as leptospirosis and typhoid. Similarly, dengue emergence in the region is linked to environmental change, including climate change and rapid, unplanned urbanisation. Earth observation technologies, which use satellite and earth surface monitoring data provide information on the natural and built environment, including climate and weather (temperature, precipitation, humidity, cloud cover, etc.), land cover and land use, vegetation, soil and elevation, underpin one novel approach; data from clinical surveillance systems, and entomological surveillance, can be integrated with earth observation data to augment existing surveillance approaches, whereby associations between environmental factors, measured using earth observation, and disease patterns can be used to predict the spatial and/or temporal distribution of disease. This approach has greatest potential for vectorborne and zoonotic infectious diseases that are sensitive to environmental factors. Digital surveillance using internet search engine queries and social media is another non-traditional approach with some promise. Digital surveillance and earth observation have the potential to be integrated within early warning systems that can trigger timely resource mobilization in the face of an emerging epidemic. In the field, systems that support operational decision-making, such as spatial decision support systems, can provide accessible, low-tech methods to enable more effective targeted intervention to combat disease outbreaks, including surveillance-response. Each of these approaches will be illustrated and critically evaluated using examples from recently conducted studies in the region.

Immunisation strategies to reduce vaccine preventable diseases: using the New Zealand example

Speaker: Associate Professor Nikki Turner, Department of General Practice and Primary Health Care, Director, CONECTUS and The Immunisation Advisory Centre, University of Auckland, New Zealand

NZ traditionally had poor immunisation coverage in their national immunisation programme. However over the past 15 years there has been dramatic improvement in both coverage and timeliness of delivery of the childhood schedule. The main reasons behind this improvement have been the ability to collect and utilise data via the NZ National Immunisation Register, the creation of national targets, feedback loops on progress and provider buy in.

NZ now has a much more integrated approach to surveillance with the ability to connect the main surveillance data bases monitoring immunisation coverage and disease surveillance, utilising encrypted data from the unique national individual identifier. Data can be used much more effectively for monitoring the programme and answering translational questions.

However gaps still remain, particularly in the areas of maternal vaccination, older children and adult vaccination. There is still significant morbidity from pertussis and influenza in young infants and some localised measles outbreaks particularly in older children and young adults. Immunisation programmes need to take a whole of life approach, having the right tools, utilising linked data sets and reporting mechanisms can support a more effective approach across the whole population.

Clinical research in the midst of an Ebola outbreak

Speaker: Professor Peter Horby, Associate Professor, Group Leader Epidemic Research Group Oxford (ERGO), Group Head / Principal Investigator and Fellow, Nuffield Department of Medicine, University of Oxford, UK

The size and scale of the on-going Ebola Virus Disease (EVD) outbreak is unprecedented, and has been declared a Public Health Emergency of International Concern. The pathogenesis of EVD is incompletely understood but high levels of viral replication and the detection of virus in multiple body tissues is typical of severe disease. Coagulopathy, disruption of endothelial function, and increased inflammatory responses are also associated with severe EVD. The association between high levels of viraemia and EVD severity suggests that therapies that target viral replication may benefit patients.

Whilst several experimental therapeutic interventions have shown promise in the laboratory and in animal studies, none have been tested for efficacy and safety in humans with EVD. In August 2014 a World Health Organization expert panel concluded unanimously that 'investigators have a moral duty to evaluate these interventions in the best possible clinical studies that can be conducted under the circumstances of the epidemic.'

I will describe the therapeutics options that are being considered for EVD, the efforts that have been made to evaluate experimental therapeutics for EVD, and some of the challenges faced.

Emerging Infectious Diseases and International Health
Chair: Vitali Sintchenko

Endgame strategies for lymphatic filariasis elimination in American Samoa

Presenter: Colleen Lau, The University of Queensland, Queensland

Authors: C Lau, R Soares Magalhaes, S Hundessa, S Sheridan, M Schmaedick, S Fuimaono, J Tufa and P Graves

Background: After seven rounds of mass drug administration (MDA) in American Samoa, lymphatic filariasis (LF) antigen prevalence dropped from 16.5% (1999) to 2.3% (2007). In 2011, prevalence in 6–7 year old children was <1%, the WHO threshold used to cease MDA. Our study aims to inform the risk of resurgence by identifying any residual infection clusters and/or high-risk populations.

Methods: A serum bank collected from 807 adults in 2010 was tested for LF antigen (Og4C3) and antibody (Bm14). Demographic data and spatial analysis were used to identify high-risk populations and residual infection clusters. In 2014, a follow up study was conducted to verify the findings.

Results: In 2010, Og4C3 prevalence was 0.8–3.2%, with two possible hot spots (estimated diameters of 1.2–1.5 km). Prevalence was higher (3.5–14.3%) in recent (<5 years) migrants compared to longer-term residents. In 2014, ICT prevalences at the two hot spots (N=125) were 4.4% and 12.5%, confirming the presence of residual clusters. Recent migrants (N=674) were tested at a workplace and clinic, and found to have ICT prevalence of 1.2% and 2.9%.

Conclusion: LF transmission appears to be still occurring in American Samoa. Further investigation is required to determine if current guidelines for certifying interruption of transmission are valid.

An outbreak of healthcare-associated cutaneous melioidosis in temperate Western Australia

Presenter: Carolien Giele, Communicable Disease Control Directorate, Department of Health, Western Australia

Authors: C Giele, T Inglis, A Merritt, B Clark, L Manning, A Peterson, P Armstrong and G Dowse

Background: Melioidosis is a serious disease caused by the environmental saprophyte *Burkholderia pseudomallei*. The organism is endemic in northern Australia and southeast Asia, and appears to be variably distributed elsewhere in the tropics.

Methods: Outbreak investigation.

Results: Six cutaneous melioidosis infections were notified among residents of a community south of Perth between January 2012 and December 2013. The index case preceded by 21 months a cluster of five cases reported over a 4-month period. All cases had attended the same medical clinic for wound dressings, the

latter five clearly prior to developing infection. A single *B. pseudomallei* MLST type was isolated from all six cases and was identical to that subsequently isolated from a bottle of saline irrigation fluid used to clean wounds of the last five cases.

Conclusion: This is the first reported healthcare-associated outbreak of cutaneous melioidosis in a temperate region. A contaminated bottle of saline was the likely source for at least five cases of infection, but the primary source of *B. pseudomallei*, the mode of contamination of the bottle, and how the organism persisted for an extended period at the clinic remain uncertain. This incident highlights the importance of maintaining infection control standards in primary healthcare settings.

Dengue infection acquired in Western Australia: evidence of importation of infected mosquitoes?

Presenter: Andrew Jardine, Department of Health, Western Australia

Authors: M Lindsay, D Smith, J Nicholson, P Neville, A Jardine, S Harrington, A Whittle, H Lyttle, A Levy and P Armstrong

A male with laboratory-confirmed dengue infection was notified to the Department of Health Western Australia (WA) in October 2013. He had a dengue-like illness and primary DENV-1 infection was confirmed by serology and PCR. He had never travelled outside the State and the most likely location of infection was Wickham or Point Sampson in the Pilbara region.

Retrospective testing of sera from other patients with suspected arbovirus illness did not identify any further cases of local infection. Intensive adult and larval mosquito surveillance undertaken immediately after notification and after the start of the wet season in January 2014 did not detect dengue virus or any vector species in the local region.

This is the first locally acquired case of dengue in WA for more than 70 years. While the source of infection in this case is unknown, it is most likely due to transient introduction of an infected mosquito by air or sea, which subsequently transmitted dengue to the patient but failed to establish a local breeding population.

This case highlights the high public health importance of vigilance in identifying possible local dengue cases, and in maintaining efforts to ensure dengue vectors do not become established in WA.

Tuberculosis in the Pacific Islands region, 2000 to 2013

Presenter: K Viney, Australian National University, Australian Capital Territory

Authors: D Hoy, A Roth, PM Kelly, D Harley and A Sleigh

Background: Tuberculosis (TB) poses a significant public health challenge in the 22 countries and territories of the Pacific Islands region.

Methods: We ascertained TB cases reported annually by Pacific Island national TB programmes to the World Health Organization, Western Pacific Region Office and calculated various standard measures for TB occurrence (i.e. case notifications, incidence, prevalence, mortality), comparing the year 2013 to the year 2000.

Results: In 2013, 24,145 TB cases were notified, most (94% or 22,657) were from Papua New Guinea (PNG). Kiribati reported the highest TB case notification rate (398 per 100,000 population). Notification rates per 100,000 population were also high in PNG, Marshall Islands and Tuvalu (309, 283 and 182, respectively). Between 2000 and 2013, TB case notification rates increased by 58%, from 146 to 231 per 100,000 population. Incidence has remained high but stable from 2000 to 2013; prevalence and mortality have fallen by 20% and 47%, respectively.

Conclusion: Although TB incidence is stable across the region, many countries have high rates and the Pacific regional burden is increasing. To halt and reverse this trend, TB control efforts and health systems must be strengthened.

What do we know about enterovirus-D68 in Australia?

Presenter: Avram Levy, PathWest Laboratory Medicine WA and School of Pathology and Laboratory Medicine, University of Western Australia

Authors: A Levy, J Roberts, J Lang, S Tempone, A Kesson, A Daly, B Thorley and D Speers

Enterovirus D-68 (EV-D68) has emerged as a common cause of respiratory illness worldwide including acute and severe disease. Recent outbreaks have involved numerous cases of respiratory disease, followed by acute flaccid paralysis (AFP) or other neurological disease in a small proportion of cases. Western Australia (WA) has experienced two peaks of EV-D68 activity during July–October (winter–spring) in 2011 and 2013 with sporadic cases recorded from 2008–2015. The majority of cases involved hospitalised patients with upper respiratory infections, particularly lower airway disease or asthma/wheeze. EV-D68 was also identified in cerebrospinal fluid from one patient. Phylogenetic analysis suggests circulation of some strains within WA as well as sporadic detections of other strains, one of which appears closely related to the Californian AFP cases. It is likely that EV-D68 circulation has not been limited to WA, but has been occurring throughout Australia with detection in faeces from two patients through nationwide AFP and enterovirus surveillance in 2010. EV-D68 should be recognised as an emerging respiratory pathogen capable of causing large outbreaks, with severe neurological disease following infection on rare occasion. Ongoing surveillance including the use of EV-D68-specific assays should be considered by large public health laboratories.

Simulation training: preparing staff for managing a person with Ebola virus disease

Presenter: Wendy Morotti, Communicable Diseases Unit, Queensland

Authors: W Morotti, D El Saadi, J Gerard, B McCall, H Carroll and S Bennett

The current Ebola virus outbreak in West Africa and subsequent potential for cases to present in other countries has required Queensland health care services to evaluate their ability to manage a person presenting with Ebola virus disease (EVD). The level of personal protective equipment required to manage a person with EVD is much higher than that used for other more commonly managed infectious conditions in Queensland, posing new challenges for staff and hospital management in meeting patient's health care needs.

To assist staff to become more confident in the safe management of an EVD case, simulated clinical training in the care of an EVD case was developed and offered through one day workshops. Medium fidelity simulation training involves the use of manikins or actors trained to demonstrate a condition. The training covered a range of scenarios including general patient care, undertaking invasive techniques and managing contaminated environments.

This presentation will describe how the health staff performed in the simulated clinical training scenarios and discuss common challenges they faced. In particular it will highlight areas of care that pose a particular risk to staff or the patient when being undertaken in full PPE and discuss how these risks might be mitigated.

Vaccine Preventable Diseases 1
Chair: Stephen Lambert

Community Acquired Pneumonia Immunisation Trial in Adults (CAPiTA)

Presenter: Marieke Bolkenbaas, University Medical Center Utrecht, the Netherlands

Authors: M Bonten, S Huijts, M Bolkenbaas, C Webber, S Gault, W Gruber, S Patterson and D Grobbee, for the CAPiTA study team

Background: Conjugate vaccines have shown to prevent pneumococcal disease in children, but efficacy in elderly has not been established yet.

Methods: The CAPiTA study was a randomised, double-blind clinical trial in 84,496 immunocompetent community-dwelling participants of 65 years and older in the Netherlands. The primary objective of the study

was to determine the efficacy of a 13-valent pneumococcal conjugate vaccine (13vPnC) in preventing a first episode of vaccine-type (VT) pneumococcal community-acquired pneumonia (CAP). Secondary objectives were to determine the efficacy in preventing a first episode of non-bacteremic/noninvasive (NB/NI) VT pneumococcal CAP and a first episode of VT-IPD. Participants were randomised 1:1 to receive either 13vPnC or placebo. Surveillance for CAP and IPD cases was conducted at hospitals in areas of enrolment. A serotype-specific urinary antigen detection assay and/or cultures of pneumococci from blood or other sterile sites were used to identify vaccine-type CAP and IPD episodes.

Results: Per protocol analysis showed a vaccine efficacy for a first episode VT-CAP of 45.56% (95.2% 21.82%–62.49%, P=0.0006); for a first episode of NB/NI VT-CAP 45.00% (95.2% 14.21%–65.31%, P=0.0067), and for a first episode of VT-IPD 75.00% (95.2% 41.43%–90.78%, P=0.0005).

Conclusions: 13vPnC was effective in preventing vaccine-type pneumococcal CAP and IPD in adults >65 years of age.

Impact and effectiveness of 13vPCV among Australian children

Presenter: Sanjay Jayasinghe, National Centre for Immunization Research and Surveillance, Sydney, Australia, Discipline of Paediatrics and Child Health, Sydney Medical School, University of Sydney

Authors: C Chiu, H Quinn, R Menzies and P McIntyre

Background: In 2011 13-valent pneumococcal conjugate vaccine (13vPCV) replaced 7vPCV in the publicly-funded immunisation program for Australian children, with the '3+0' schedule (3 primary doses without booster) maintained. The 13vPCV was licensed based on immune correlates of protection. We assessed the coverage, impact and effectiveness (VE) against IPD three years into the 13vPCV program.

Methods: We assessed the serotype-specific IPD incidence rate ratios (IRRs) post (2011/12–2013/14) versus pre (2008/09–10/11) 13vPCV introduction in children aged <5 years using national laboratory-based notification data, and vaccine uptake from the Australian Childhood Immunization Register. We employed conditional logistic regression analysis to determine VE based on odds ratios for 13vPCV vaccination in cases versus age-matched controls.

Results: For children aged <5 years, the 3-year average annual incidence rate (per 100,000) of 13v-non-7v IPD declined from 10.7 (n=143, 2008/09–10/11) to 3.8 (n=54, 2011/12–2013/14). The greatest impact was against serotype 19A (IRR: 0.23, 95%CI 0.15–0.33). In 2013, 90% of children were fully vaccinated by 12 months of age. VE of ≥1 dose of 13vPCV against 13-non-7v IPD was 95.6% (77.8–99.1%).

Conclusion: A substantial reduction occurred in IPD due to 13v-non-7v serotypes in Australian children since 13vPCV ('3+0' schedule) introduction. Overall, 13vPCV VE is consistent with predictions based on immune correlates.

Birth outcomes among Australian women who receive an influenza vaccine in pregnancy, 2012–2014

Presenter: Lisa McHugh, MAE scholar, Queensland Children's Medical Research Institute and Communicable Diseases Unit, Queensland Health and Australian National University, ACT

Authors: L McHugh, RM Andrews, K Viney, SB Lambert and KA O'Grady

Background: Although recommended for all pregnant women, there is a shortage of Australian data on the safety and effectiveness of influenza vaccination in pregnancy. We aimed to determine whether there are any differences in relation to birth weight and gestation at birth of infant between groups based on pregnancy vaccination status.

Methods: A cohort study of women aged >17 years recruited between February 2012 and December 2014 at less than 8 weeks postpartum. The primary exposure of interest was self-reported influenza vaccination in pregnancy. The primary outcomes of interest were infant birth weight in grams and gestation (weeks) at birth of infant.

Results: There were 7175 women enrolled. Mean maternal age at infant birth was 31.7 years (range 17–51 years). The mean birth weight of infants born to vaccinated mothers was 3325 grams (95% CI 3301, 3350) compared to 3336 (95% CI 3317, 3356) in unvaccinated mothers, $P=0.50$; the mean gestation at birth was 38.7 weeks (95% CI 38.6, 38.7) compared to 38.7(95% CI 38.6, 38.8) $P=0.43$ respectively.

Conclusion: We found no association between influenza vaccination during pregnancy and either birth weight or gestational age. These data will be an important contribution to evaluating the impact of influenza vaccine in pregnancy.

Post-exposure passive immunisation for preventing rubella and congenital rubella syndrome

Presenter: Megan Young, Griffith University, Queensland

Authors: MK Young, AW Cripps, GR Nimmo and ML van Driel

Background: Primary studies examining the effectiveness of polyclonal immunoglobulins for post-exposure prophylaxis of rubella have small sample sizes and varying results.

Methods: We conducted a systematic review as per standard Cochrane methodological procedures.

Results: Twelve studies (430 participants) were included; seven RCTs and five controlled clinical trials (CCTs). The result of meta-analysis (11 studies) of gammaglobulin versus control (saline or no treatment) for preventing rubella cases favoured the intervention group (RR 0.61, 95%CI 0.45–0.83) but studies were heterogeneous. Heterogeneity was explained by subgrouping studies according to the estimated volume of gammaglobulin administered by weight and then removing studies where the intervention was given >5 days after exposure ("0.027–0.037 ml/lb" RR 1.60 (95%CI 0.57–4.52); "0.1–0.15 ml/lb" RR 0.53 (95%CI 0.29–0.99); "0.2–0.5 ml/lb" RR 0.20 (95%CI 0.04–1.00)).One study examined the incidence of congenital rubella syndrome among pregnant participants (<9/40 weeks) who were randomised to one of two gammaglobulin groups ('high' or 'low' rubella titre). No cases were identified.

Conclusion: Compared to no treatment, polyclonal immunoglobulins are of benefit for preventing rubella if given in sufficient dose up to five days after exposure. There is insufficient evidence to make direct conclusions about the effectiveness of polyclonal immunoglobulins for preventing congenital rubella syndrome.

The 2012/3 measles serosurvey: at odds with Australia's elimination status?

Presenter: Heather F Gidding, University of New South Wales and National Centre for Immunisation Research and Surveillance, New South Wales

Authors: HF Gidding, HE Quinn, L Hueston, DE Dwyer and PB McIntyre

Background: Australia's measles elimination status was verified in 2014, but notifications have increased since 2011.

Aim: Estimate population immunity to measles using the 2012/3 national serosurvey and R (the effective reproduction number), and compare with previous serosurveys.

Methods: A similar national sample to previous serosurveys was tested (residual sera and plasma from 1–49 year olds obtained from public and private laboratories) using the Enzygnost anti-measles IgG enzyme immunoassay. R was calculated from weighted estimates of the proportion seronegative by age using established methods.

Results: The weighted proportion seronegative increased progressively from 3.7% (95%CI: 3.3–4.2%) in 1999 to 10.3% (9.11–1.4%) in 2012/3. Similarly, the proportion equivocal increased from a low of 1.7% (95%CI: 1.4–1.9%) in 2002 to 8.9% (95%CI: 7.8–10.0%) in 2012/3. These changes were most pronounced in 10–34 year olds and suggest waning vaccine induced immunity. R increased from 0.57 in 1999 to above the epidemic threshold of 1 in 2012/3 (R=1.7)

Conclusion: The 2012/3 serosurvey results are consistent with the age distribution of notified measles cases, but at odds with other indicators (e.g. number and importation status of notifications) which support the conclusion that measles elimination is being maintained. Steps to further investigate these serologic findings are being considered.

Using SMS technology to actively monitor the safety of vaccines in general practice

Presenter: Alan Leeb, Illawarra Medical Centre

Authors: A Leeb, AK Regan, I Peters, G Leeb, L Tracey and PV Effler

Introduction: Ongoing monitoring of adverse events following immunisation (AEFI) is essential for detecting safety signals and maintaining public confidence. SmartVax is a general practice initiative using software to send short message service (SMS) texts to query vaccines about AEFI.

Methods: All persons immunised at a WA general practice during 2012–2014 were sent an SMS 3 days post-vaccination asking if they experienced any reaction(s). Patients who replied "Yes" were invited to complete an online survey to assess nature, severity and duration of the reaction.

Results: 8,868 vaccination encounters received SMS follow-up and an SMS response was received for 75%. The overall reaction rate was 7% (9% in persons under 16 years and 5% in others); most were local reactions. Differences by vaccine brand were identified. The change to combined MMR-V did not increase reported reactions.

Conclusions: This prototypic system provides a timely, efficient way to monitor vaccine safety. While the data presented are from a sole large group site, a network of practices has been established and currently 19 sites across five jurisdictions are participating. A network of GP sites uploading de-identified data to an aggregated database could serve as an early warning system for unanticipated safety signals.

Sexually Transmissible and Bloodborne Infections
Chair: Linda Selvey

Human papillomavirus control: registry data support an impact of incomplete vaccine courses

Presenter: Julia Brotherton, National HPV Vaccination Program Register, VCS, Victoria

Authors: J Brotherton, M Malloy, D Gertig, A Budd, K Drennan and M Saville

Background: Optimised two-dose HPV vaccine schedules are now endorsed for young adolescents by WHO. Limited data are available about effectiveness of <3 doses using a standard dose schedule.

Methods: Deterministic data linkage was undertaken between the Victorian Cervical Cytology Registry and National HPV Vaccination Program Register to determine vaccination status and incidence of cervical pathology among vaccine eligible women screened in Victoria between April 2007 and December 2011. Proportional hazards regression was used to estimate hazard ratios (HR) adjusted for age, socioeconomic status and area of residence. Women were stratified into those vaccinated before or after first screen.

Results: Any number of doses (1, 2 or 3) were associated with lower rates of high grade and low grade cytology diagnoses *as long as doses were given prior to screening commencement* (one dose HR high grade 0.44 (95%CI 0.32 to 0.59), one dose low grade 0.48 (95%CI 0.40 to 0.58); two doses HR high grade 0.63 (95%CI 0.50 to 0.80), HR low grade 0.52 (95%CI 0.44 to 0.61); three doses HR high grade 0.53 (95%CI 0.47 to 0.60), HR low grade 0.73 (95%CI 0.68 to 0.78)).

Conclusion: Our data suggest vaccine effectiveness of less than three doses against cervical intraepithelial neoplasia.

Trends in testing for chlamydial infection in the ACT, 2003 to 2012

Presenter: Lucas Mills, Australian National University and ACT Health Protection Service

Authors: L Mills, R Hundy and E Fearnley

Keywords Sexually transmissible infections, chlamydia, surveillance

Background: Around Australia, notifications for chlamydial infection have increased rapidly over recent years. Notification data is susceptible to changes in testing in the community. This study sought to describe testing trends to assist the interpretation of disease notifications.

Methods: We used pathology testing data provided by two major pathology providers in the ACT (accounting for 77% of laboratory notifications) from 2003 to 2012. Pathology results were analysed by sex, age, clinical setting and year of test, using Stata13.

Results: The overall proportion of positive tests was higher in males than females (5.7% compared with 3.1%) with no statistically significant change in positivity over this period. The number of tests performed annually increased from 10,997 in 2003 to 20,139 in 2012, with increased testing in both a general practice and sexual health setting. Approximately two-thirds of all tests were performed on women, although the proportion of tests performed on men has increased over time from 27.7% to 38.1%.

Conclusions: Analysis of ACT data suggests the increase in chlamydia notifications is associated with increased testing. This study has demonstrated that it is feasible to utilise pathology testing data to interpret trends in notification data.

Peer-led STI testing and early HIV diagnosis among MSM in Western Australia

Presenter: Byron Minas, Department of Health Western Australia

Authors: BC Minas, CM Giele, SC Laing, L Bastian, AW Burry, KJ Sales and DB Mak

Background: We describe trends in newly acquired HIV notifications among men who have sex with men (MSM) in Western Australia (WA) since commencement of the M Clinic, a peer-led STI testing service for MSM established by the WA AIDS Council in 2010.

Methods: Numbers and proportions of MSM HIV cases with newly acquired infection were compared for the 2004–2006, 2007–2009 and 2011–2013 time periods. Data from 2010 were excluded as the M Clinic opened that year.

Results: Between the 2004–2006 and 2007–2009 periods, the number of MSM with newly acquired HIV increased by 50% (23 to 33 cases) and the number of newly acquired cases as a proportion of new HIV diagnoses among MSM increased from 27% to 35% (30% increase) (P= NS). In the 2011–2013 period, the number of newly acquired HIV cases among MSM more than doubled compared to 2007–2009, and comprised 53% (n=70) of new HIV diagnoses among MSM (P< .05).

Conclusions: The proportion of MSM HIV notifications that were newly acquired increased between 2004 and 2013 in WA, with the greatest increase seen after the M Clinic's commencement. Peer-led approaches to HIV testing can contribute to early diagnosis among MSM.

Mapping progress towards Australia's National Hepatitis B Strategy targets, 2013

Presenter: Jennifer MacLachlan, The Doherty Institute, Victoria

Authors: J MacLachlan, N Allard and B Cowie

Background: The National Hepatitis B Strategy includes specific targets to achieve in order to improve the response to chronic hepatitis B (CHB). We assessed progress towards these targets in the areas of diagnosis, monitoring, treatment and vaccination, for Australia's 61 Medicare Locals.

Methods: Data for the period 2012–13 were analysed, including the Australian Childhood Immunisation Register, Pharmaceutical Benefits Schedule prescribing data, National Notifiable Diseases Surveillance System notifications, and previously derived estimates of hepatitis B prevalence.

Results: National immunisation uptake at one year in 2012–13 was 91.5%, with no Medicare Locals reaching the Strategy's target of 95%. Several areas with uptake <90% have been previously identified as having high CHB prevalence. Progress towards the target of 80% of people with CHB being diagnosed is static, being 57% nationally in 2013 and the number of notifications remaining stable.

No Medicare Local achieved the 15% treatment target in 2013, and only twelve MLs achieved uptake above the national average of 5.3%, all in Sydney and Melbourne.

Conclusions: This analysis demonstrates that Australia's response to CHB is falling short of the targets identified in the National Strategy, and that identifying high and low performing areas can highlight effective strategies and areas of greatest need.

Molecular detection of *Neisseria gonorrhoeae* antimicrobial resistance: moving towards individualised treatment strategies

Presenter: Ella Trembizki, QCMRI, The University of Queensland

Authors: E Trembizki and D Whiley on behalf of GRAND investigators

N. gonorrhoeae antimicrobial resistance (AMR) is a major global concern. There are limited new treatment options and because of increased use of Nucleic Acid Amplification Testing (NAATs) for diagnosis, there is a lack of AMR data. As a part of the Gonorrhoea Resistance Assessment by Nucleic acid Detection (GRAND) study, we examined the molecular basis of AMR in 2,228 *N. gonorrhoeae* isolates from throughout Australia in the first half of 2012. Mutation profiles were then correlated with minimum inhibitory concentrations and geographical location. Through this screening we identified candidate sequences that predict resistance to penicillin, ciprofloxacin and azithromycin, and have now developed real-time PCR methods to predict resistance to ciprofloxacin and azithromycin. These Cipro-NAAT and azithro-NAAT methods were then applied to NAAT positive clinical samples (N=576) from the Northern Territory for the first half of 2014, and the results compared with bacterial culture-based surveillance. Our data confirms a lack of azithromycin resistance in the NT, but suggest that ciprofloxacin resistance may be overestimated. We are further investigating these results. These data show how molecular AMR surveillance can enhance bacterial culture-based capabilities. In addition, our assays provide promising steps toward individualised treatment strategies for *N. gonorrhoeae* infection.

Epidemiology of gonorrhoea notifications in Australia, 2007–2012

Presenter: April Roberts-Witteveen, ACT Health

Authors: A Roberts-Witteveen, K Pennington, N Higgins, C Lang, M Lahra, R Waddell and J Kaldor

Introduction: In Australia, gonorrhoea is rare apart from among some populations of Aboriginal and Torres Strait Islander people and men who have sex with men. An investigation into the increase in notifications in Australia from 2009 was undertaken.

Methods: Notified gonorrhoea cases reported between 2007 and 2012 were obtained from the National Notifiable Diseases Surveillance System. Analyses undertaken included time trends in counts and rates, according to jurisdiction, gender, Aboriginal status and sexual orientation.

Results: The largest increases in notification rates over the study period were in both sexes in New South Wales (2.9 and 3.7 times in 2012 compared to 2007) and Victoria (2.4 and 2.7), men in the Australian Capital Territory (2.3) and women (2.3) in Queensland. Although their rates decreased over time, the highest notification rates were in Aboriginal women in the Northern Territory and Western Australia. Changes in age and sex distribution, antimicrobial resistance or patterns of exposure and acquisition were not observed.

Conclusion: There is an ongoing gonorrhoea epidemic affecting Aboriginal people in Australia, but the increases in notifications have primarily occurred in non-Aboriginal populations in the larger jurisdictions. Interpretation of these data would be enhanced by laboratory testing data.

Indigenous Populations and Other Vulnerable Groups
Chair: Vicki Krause

Trachoma prevalence according to treatment strategy in Australia between 2007 and 2013

Presenter: John Kaldor, University of New South Wales, New South Wales

Authors: B Liu, C Cowling, A Hayen, G Watt, D Mak, S Lambert, H Taylor and J Kaldor

Background: We examined how different azithromycin treatment strategies have affected trachoma prevalence.

Methods: Annual community-level data on trachoma prevalence and treatment in 5–9 year old children from 3 Australian jurisdictions between 2007–2013 were examined according to the trachoma treatment strategy implemented (no treatment, active case only, household and community-wide). Changes in community trachoma prevalence according to treatment strategy were estimated using random-effects meta-analysis.

Results: 182 communities (42.3% from the Northern Territory, 11.5% from South Australia and 46.2% from Western Australia) had 881 treatment and corresponding trachoma prevalence records. The greatest annual fall in trachoma prevalence was in communities implementing community-wide treatment: absolute reductions ranged from –8%(95%CI –17% to 1%) to –31%(–26% to –37%); these communities also had the highest baseline trachoma prevalence (15.4%–43.9%). Including only communities with moderate trachoma prevalence (5–<20%) at initial measurement, the change in trachoma prevalence from the first to the last year of data recorded was similar for communities implementing community-wide or those using more targeted treatment strategies: absolute reductions were –11%(–8% to –13%) and –7%(–5% to –10%) respectively.

Conclusion: Community-wide azithromycin administration reduces trachoma prevalence. Less intensive treatment in moderate prevalence communities may lead to similar reductions in prevalence. Facial cleanliness and environmental interventions should also be implemented.

Diversity of *emm* types causing Invasive group A streptococcal disease, Northern Territory

Presenter: Rowena Boyd, Centre for Disease Control, Northern Territory Department of Health

Authors: R Boyd, B J Currie, D C Holt, T Harris and V Krause

Background: Northern Australia has high rates of invasive group A streptococcal (GAS) disease, with highest incidence in the Indigenous population. Multivalent GAS vaccines will potentially reduce rates of GAS-related diseases however only cover up to 30 of over 200 *emm* types identified. Our study adds to the limited knowledge of *emm* types circulating and disease transmission which has implications for public health response.

Methods: Laboratory isolation of *Streptococcus pyogenes* from a normally sterile site is notifiable in the Northern Territory. Demographic and clinical data were collected from medical records for notifications between 2011–2013. Molecular typing characterised GAS isolates according to sequencing of *emm* genes.

Results: Of 128 notifications, 81(64%) isolates were available for typing and 28 different *emm* types were identified. Geographical clusters were not evident and no associations were shown between *emm* type and clinical manifestation or illness severity. Typing confirmed transmission of *emm* 207.1 between infant twins and refuted transmission in two household-case pairs.

Conclusions: The diversity of *emm* types limits potential vaccine coverage and highlights the importance of educational, social and environmental interventions that limit spread of GAS in community. Typing of isolates was important to verify transmission but did not point to illness presentation or severity.

Malnutrition increases the risk of lower respiratory infection with respiratory syncytial virus

Presenter: Stuart Paynter, School of Public Health, University of Queensland

Authors: S Paynter, R Ware, L Yakob, M Lucero, V Tallo, H Nohynek, P Weinstein, P Sly, G Williams and E Simoes

Although malnutrition is a well known risk factor for clinical pneumonia in children, it is not clear whether or not malnutrition increases the risk of lower respiratory infection due to viruses. Resolving this question requires large scale longitudinal studies.

We followed up a cohort of 12,191 infants who were part of a pneumococcal vaccine trial in the Philippines. Children who had lower than median growth between their first and third pneumococcal vaccinations had a higher rate of subsequent hospital admission with lower respiratory infection due to respiratory syncytial virus (RSV), compared to those with growth above the median (hazard ratio: 1.34; 95% confidence interval: 1.02–1.76)

In addition, we used a mathematical transmission model to examine whether seasonal malnutrition in children in the study setting had any impact on RSV incidence. The results of the mathematical model indicate that peak RSV transmissibility was highest during the period of seasonal malnutrition.

Poor infant growth appears to increase the risk of hospital admission with lower respiratory infection due to RSV

Reducing rheumatic fever in New Zealand

Presenter: Chrissie Pickin, Ministry of Health, New Zealand

Authors: C Pickin and N Stefanogiannis

Indigenous Mâori and Pacific people living in New Zealand experience high rates of rheumatic fever and rheumatic heart disease. The New Zealand Government has committed to tackling this inequity and reducing the incidence of rheumatic fever by two-thirds by 2017. The New Zealand Rheumatic Fever Prevention Programme encompasses a range of innovative activities aimed at preventing the transmission of Group A streptococcal throat infections and ensuring that these throat infections are treated quickly and effectively. The activities include a 'primordial' prevention programme focussed on reducing household crowding in priority communities and improving access to sore throat management services in primary care and community settings.

2014 rheumatic fever hospitalisation data suggest that the programme is having an effect with a statistically significant reduction between 2013 (4.3 per 100,000 hospitalisations) and 2014 (3.4 per 100,000 hospitalisations). The decrease was achieved in both Mâori and Pacific people and was statistically significant for Mâori.

This presentation will include an overview of this multi-faceted programme and will share lessons for successful programme design and delivery that can reduce health inequity.

The use of routinely-collected primary care data for improving the control of sexually transmissible infections in Aboriginal communities

Presenter: Kathryn Taylor, Aboriginal Health Council of South Australia (AHCSA)

Authors: K Taylor, K Marshall, S Betts and D Scrimgeour

Aboriginal people continue to experience a high prevalence and incidence of sexually transmissible infection (STI), especially in remote locations. We describe the development of an innovative system of collection and analysis of STI testing data from Aboriginal community-controlled health services (ACCHSs) in South Australia to facilitate sexual health quality improvement initiatives and develop a greater understanding of STI epidemiology in Aboriginal communities across the state.

The Aboriginal Health Council of SA (AHCSA) is the peak body representing 10 ACCHSs in South Australia. AHCSA's Sexual Health Program (SHP) began in 2010, building capacity within ACCHSs to deliver sexual health services for Aboriginal communities. This includes supporting ACCHSs to run a 6-week community screening activity, for which testing rates and prevalences are reported annually. In 2013, a partnership was negotiated with SA Pathology, whereby results of all tests conducted at participating ACCHSs for chlamydia, gonorrhoea and trichomoniasis were reported to AHCSA. This has enabled STI data to be analysed and disseminated to health services throughout the year, to identify gaps in service delivery and guide the work of the SHP. We will present the development of the surveillance system, its current uses, findings to date and future directions for the program.

Person–to-person engagement – achieving exceptional program outcomes in remote Indigenous communities

Presenter: Gabrielle Watt, Department of Health, Northern Territory

Authors: J Arnold, S Cooney, B Johnson, D Miller, A Wilson, P Wines and L Wing

Achieving high coverage rates for health service delivery in remote communities is often difficult due to factors including high population mobility, language barriers and cultural differences.

The Northern Territory trachoma program uses a unique approach to delivering health care in remote communities by speaking individually with each person in the community. Community members are provided with individual information to make decisions on screening and treatment options for themselves and their families. Novel in a time of media, texts and computers, this intensive approach has resulted in exceptional screening and treatment coverage rates. In 2014, >95% of 5–9 year old Indigenous children living in at risk remote communities were screened for active trachoma, while >90% of community members received treatment with azithromycin as indicated by the *CDNA Guidelines for the Public Health Management of Trachoma in Australia*.

Australia, the only high income country to still have blinding trachoma, has made a commitment to the World Health Organisation to eliminating blinding trachoma by 2020. Trachoma (*Chlamydia trachomatis*) infection has been around since the Bronze Age. This presentation will outline how community engagement and education can contribute to excellent program outcomes.

Public Health Surveillance 1
Chair: Sonya Bennett

Validity of antenatal influenza vaccination surveillance systems in Western Australia

Presenter: Annette Regan, Department of Health Western Australia

Authors: AK Regan, DB Mak, HC Moore, L Racey, R Saker, C Jones and PV Effler

Background: Although influenza vaccination has been available to pregnant women since 2009 in Australia, data on vaccine coverage are limited. Our aim was to evaluate the validity of existing databases in Western Australia for measuring antenatal influenza immunisations.

Methods: Self-reported vaccination status of 563 women who delivered between March and October 2013 was compared against three data sources: a state-wide antenatal influenza vaccination database maintained by the Department of Health, a public maternity hospital database, and a private health service database. Sensitivity, specificity, and positive and negative predictive values were calculated for each system using self-report as the "gold standard."

Results: The state-wide antenatal vaccination database detected 45.7% (95% CI 40.1–51.4%) of influenza vaccinations, the public maternity hospital database detected 66.7% (55.1–76.9%), and the private health service database detected 29.1% (20.5–39.4%). The specificity of each system exceeded 90% and positive predictive values exceeded 80%. Sensitivity was lower in women whose antenatal care was provided by a private obstetrician.

Conclusions: Existing influenza vaccination surveillance systems for monitoring influenza vaccine uptake during pregnancy detect between 29% and 67% of vaccinations. Considering the importance of influenza immunisation as a public health intervention, particularly in pregnant women, alternative sources of vaccination information for surveillance should be explored.

Using SMS to monitor communicable disease contacts: the EbolaTracks program

Presenter: Lauren Tracey, Department of Health, Western Australia

Authors: LE Tracey, AK Regan, PK Armstrong, GK Dowse and PV Effler

Background: Monitoring contacts in a disease outbreak requires substantial time, resources, and coordination between health agencies. WA Health built an automated SMS system to facilitate active monitoring of persons travelling from Ebola virus-affected countries for 21 days following their last potential exposure.

Methods: EbolaTracks participants receive automatic SMS messages twice daily to ascertain if they are feeling unwell and to solicit their recorded temperature by SMS reply. If a participant reports symptoms, or does not respond, EbolaTracks automatically alerts the on-call officer via SMS and email, leading to a direct response

Results: EbolaTracks has been used successfully to monitor all travellers to WA from Ebola virus-affected countries. The current response rate is 84% for all SMS sent, saving substantial time and resources that would otherwise be spent on telephone follow-up.

Conclusions: EbolaTracks has demonstrated proof-of-concept for the use of SMS technology to actively monitor contacts exposed to a significant communicable disease. While this system has been successfully used in WA for Ebolavirus, the system could also be applied to more routine disease control contexts (e.g. measles contacts, gastroenteris outbreaks), as well as for contacts of emerging diseases of public health concern (e.g. MERS-CoV, avian influenza), in both developed and developing country settings.

Ross river virus notifications – challenges in interpreting data

Presenter: Linda A Selvey, School of Public Health, Curtin University

Authors: LA Selvey, J Donnelly, M Lindsay, SP Boddu, V D'Abrera and DW Smith

An increase in Ross River virus (RRV) notifications in South-West Western Australia in the off-season from 2006–2009 prompted an investigation of RRV notifications from Perth and the adjacent Peel region. This included a review of enhanced surveillance, and analysis of laboratory data from the state reference laboratory (PathWest) and from a private laboratory.

We identified some challenges in interpreting RRV notifications data. These include: 34% of winter notifications in the Perth/Peel regions were incorrectly ascribed to either Perth/Peel region or season (vs 5% of notifications from summer months); laboratories don't consistently adhere to the nationally agreed case definition for RRV when notifying; and a single IgM positive laboratory test had a very low PPV –the estimated PPV was 4.0% in the off-season and 24.6% in the RRV season (commercial test kit); and 39.3% in the off season, and 81% in the RRV season for the in-house immunofluorescence test performed by PathWest.

We recommend that IgM positive, IgG negative RRV test result be excluded from the national RRV case definition. Based on our data, this would remove 17% of PathWest notifications and 76% and 65% of notifications from private laboratories in the off-season and RRV season respectively in South-West Western Australia.

Evaluation of *healthdirect* Australia as a surveillance tool for seasonal influenza

Presenter: Tove Fitzgerald, Clinical Nurse Consultant, Hunter New England Population Health, New South Wales, MAE scholar, Australian National University, Australian Capital Territory

Authors: T Fitzgerald, C Dalton, S Carlson, PD Massey and K Viney

Background: *Healthdirect* is a national nurse triage helpline. This is the first evaluation of *healthdirect* for influenza surveillance using CDC guidelines for evaluating public health surveillance systems.

Methods: We described and compared demographics and age standardised influenza-like illness rates by week of *healthdirect* patients to laboratory notified influenza cases and Flutracking participants with fever and cough from 2009–2012. We also described distribution of *healthdirect* calls by weekday in addition to frequency of clinical pathway use and triage outcomes. Stakeholder interviews were conducted.

Results: A total of 180,076 *healthdirect* patients with influenza-like illness were triaged from 2009–2012. Of these, 100,775 (55.9%) were aged 0–4 years, the highest daily call count was 31,743 calls (17.6%) on Sundays and the 'paediatric colds' clinical pathway was the most frequently assigned pathway (62,472; 34.7%). The *healthdirect* data was consistent with Flutracking and laboratory data but diverged during the pandemic. There was strong support for jurisdictional level data.

Conclusion: *Healthdirect* is a useful surveillance tool; however high influenza-like illness rates and lower severity calls in 2009 suggests data may be biased in pandemics by community concern. State level data analysis should be explored.

Implementation of enhanced surveillance for early HIV treatment in NSW

Presenter: Melanie Middleton, Communicable Diseases Branch, Health Protection NSW, New South Wales

Authors: M Middleton, B Telfer, V Bowden, V Sheppeard, J McAnulty and C Selvey

Background: The *NSW HIV Strategy 2012–2015* set a target for HIV treatment uptake as it has been shown to significantly reduce the risk of HIV transmission. To monitor this, we introduced a system to collect information on early HIV treatment uptake for people newly diagnosed in NSW.

Methods: Questionnaires were mailed to the treating doctor of all NSW residents whose first diagnosis was in NSW each quarter, prospectively for people diagnosed after 1 September 2013 and retrospectively for people diagnosed between 1 January 2013 and 1 September 2013. Information was collected on the treatment start date, other clinical variables, and the reason for treatment deferral or loss to follow-up where indicated.

Results: Of 534 new HIV diagnoses, 1 January 2013 to 30 June 2014, 451 (84%) had a questionnaire returned. Of these, 7% had been lost to follow-up by the reporting doctor. After excluding 31 people who were no longer residing in NSW, 83% (n=352/420) were retained in care in NSW at least six months post diagnosis and 292 (55%) had commenced treatment within six months of diagnosis.

Conclusions: Enhanced surveillance for early HIV treatment was successfully implemented in NSW, providing useful indicators with which to monitor progress against the HIV Strategy.

One Health in practice

Presenter: Keith Eastwood, Hunter New England Population Health, New South Wales

Authors: B Paterson, K Eastwood, P Massey, D Durrheim, K Cox-Whitton, T Grillo, P Cashman, S Britton, A Lee, B Moloney, T Fitzgerald and P Corben

The concept of One Health is often viewed as an ideal philosophy; rhetorical rather than functional. We demonstrate that a collaborative partnership undertaken in northern NSW between NSW Health, the NSW Department of Primary Industries and allied non-government agencies, contributes to Australia's

human and animal health security and provides pragmatic 'One Health' solutions. The group is an informal but active partnership which shares zoonotic and public health information. We identify ways to minimise zoonotic risks; ensure members are alerted to emerging risks; jointly manage zoonotic outbreaks; undertake research and participate in disaster preparedness exercises. We have co-published journal articles; contributed to policy development across agencies and drafted fact sheets for the public, doctors and vets. The group meets quarterly by teleconference and maintains regular email contact. Collaborative work completed through the partnership includes public health awareness, applied research and outbreak investigation and has included particular focus on Hendra virus, Australian Bat Lyssavirus, Q Fever, Brucellosis (*Brucella suis*), Cryptosporidiosis and Leptospirosis. In the event of an emergency zoonotic event, such as an outbreak of highly pathogenic avian influenza in New South Wales, established communication and operational mechanisms allows for a rapid and coordinated response. This group is One Health in action.

Vaccine Preventable Diseases 2
Chair: Frank Beard

Is cocooning as a vaccination strategy effective in preventing pertussis in infants?

Presenter: Stacey Rowe, Department of Health, Victoria, Australia

Authors: SL Rowe, EL Tay, LJ Franklin, N Stephens, RS Ware, M Batchelor, RA Lester, M Kaczmarek and SB Lambert

Background: Between June 2009 and June 2012, the Victorian Department of Health implemented a cocooning program whereby parents of new babies were offered a free pertussis-containing vaccination at or around the time of birth. Here we report vaccine effectiveness of the program in reducing the risk of pertussis in infants.

Methods: A matched case–control study was conducted. Notified cases of pertussis aged less than 12 months were matched to controls by area of residence and date of birth. Telephone interviews were conducted with parents to ascertain whether they were vaccinated against pertussis, and when vaccination took place.

Results: Vaccination of both parents after delivery and ≥28 days of onset of illness was effective in reducing pertussis infection after adjusting for potential confounding variables: adjusted vaccine effectiveness (VE) 76% (95% CI, 5%–94%). The unadjusted effectiveness of vaccinating mothers after delivery and ≥28 days of onset of illness (VE 60%, 95%CI 18%–80%) was lost after adjustment for fathers' vaccination status and other factors (aOR 1.01, 95%CI 0.36–2.86).

Conclusion: In our population, cocooning as a vaccination strategy—when defined as vaccination after delivery and ≥28 days of onset of illness—appeared to be effective only when both parents receive timely vaccination.

Pattern of pertussis in a children's hospital in the post PCR era

Presenter: Helen Quinn, NCIRS, Sydney Australia

Authors: S Hale, H Quinn, N Wood and P McIntyre

Background: The recent pertussis epidemic was marked by high rates of notified disease, associated with widespread PCR use and the detection of milder cases. However little information is available for children who presented to hospital with pertussis in this period, particularly those ≥12 months of age.

Methods: Retrospective observational study of pertussis cases presenting to hospital during a 6 year period 2007–2012. Laboratory testing method, disease severity, vaccination status and prevalence of comorbidities by age were examined.

Results: Children ≥12 months of age accounted for 42% of cases 2007–2012, usually fully immunised and if hospitalised, had comorbidities in 56%, including 80% requiring ICU admission. In 2007–2012, despite PCR having become available, annual culture requests were higher than during the pre PCR 1997–1999

epidemic period. Comparing the two periods, the average annual number of culture positive hospitalisations in infants was similar in 2007–2012 to 1997–1999, but was significantly higher among children ≥12 months of age.

Conclusion: Comorbidities were common among older immunised children requiring hospitalisation. Increased numbers of hospitalisations in children ≥12 months of age, even when restricted to culture positive cases, is consistent with waning vaccine effectiveness in the absence of a booster dose at 18 months of age.

Clinical severity comparisons between pertactin deficient and pertactin positive *Bordetella pertussis* variants

Presenter: Michelle Clarke, Women's and Children's Hospital, South Australia

Authors: H Quinn, S Octavia, R Lan, N Wood, L Gilbert, V Sintchenko, G Hanly, C Blyth, P McIntyre and H Marshall

Background: Pertussis control remains challenging in Australia despite high coverage for infant pertussis immunisation. Recent literature highlights changes to the circulating *Bordetella pertussis* genotypes, including absence of pertactin (PRN) antigens, one of the primary antigens included in acellular pertussis vaccines. This study aimed to compare clinical disease severity indicators between PRN+ve and PRN–ve infections in children.

Methods: Genotyping of *B*. pertussis isolates was conducted using published methods between 2008 and 2012. Clinical records of culture positive cases with genotype data available were reviewed for participating hospitals in WA, SA and NSW. Severity indicators were compared between PRN+ve and PRN-ve *B. pertussis* infections.

Results: Among 199 *B. pertussis* isolates, 71 (36%) were PRN-ve and 128 PRN+ve. The proportion requiring admission as opposed to emergency department management only was similar (40/71, 56.3% vs 71/128, 55.5%, p=0.906) as was median length of stay for admitted cases (4 vs 5 days, p=0.385) for PRN-ve vs PRN+ve cases. There was a non-significant trend for PRN-ve cases to require intensive care less frequently than PRN+ve cases (5/71;7.0% vs16/128;12.5%, *P*=0.185)

Conclusions: Culture positive cases of pertussis predominantly occur in young unimmunised infants. In this opportunistic sample from 3 hospitals, we did not find evidence of significantly different clinical severity between PRN-ve and PRN+ve *B. pertussis* strains.

Factors associated with pertussis hospitalisation in adults-a population based nested case-control study

Presenter: Surendra Karki, University of New South Wales, NSW, Australia

Authors: S Karki, P McIntyre, AT Newall, CR MacIntyre, E Banks and B Liu

Background: Few studies have looked at risk factors for notified pertussis but not for pertussis-related hospitalisation in adults.

Methods: We examined the association between various factors and pertussis-related hospitalisations in a cohort of older adults in the 45 and Up study in New South Wales, Australia, between 2006 to 2012 using record linkage and a nested case-control design.

Results: Among 265,287 participants, the incidence of pertussis notifications and hospitalisations was 77.8 (95% CI, 73.0–82.8) and 2.9 per 100,000 person years, respectively. Defining cases as those hospitalised with pertussis and control as those notified but not hospitalised, we found that increasing age , smoking, and more remote residence were associated with a greater likelihood of pertussis hospitalisation [65–74 years aOR 5.98 (95%CI 1.65–21.68), 75+ years aOR 10.88 (95%CI 2.58–45.77) compared to age category 45–54 years], geographical residence [remote/very remote aOR 10.65 (95%CI 2.28–49.70), outer regional 3.56 (95%CI 1.23–10.29) compared with residence in major city] and smoking [smoking in the past, aOR 2.23 (95%CI 1.01–4.92) compared with people who never smoked].

Conclusions: The primary risk factors for pertussis hospitalisation among those with a pertussis notification were older age, smoking and region of residence. Other potential risk factors such as asthma and high BMI had non-significantly higher odds of hospitalisation.

Factors associated with pertussis vaccination in a cohort of older Australian adults

Presenter: Amalie Dyda, School of Public Health and Community Medicine, UNSW

Authors: A Dyda, S Karki, P McIntyre, CR MacIntyre, J Kaldor and B Liu

In Australia adult pertussis vaccination is not funded by the National Immunisation Program, but is recommended for some. In NSW between 2009–2012 a government program provided free pertussis vaccination to this group.

We used data from the 45 and Up study, a cohort of NSW residents aged >45 years. Using a questionnaire, participants were asked about pertussis vaccination. We estimated coverage and investigated characteristics associated with pertussis vaccination using multivariate logistic regression.

Among 27036 participants, overall 17.8% (95% CI 17.3–18.2) reported pertussis vaccination in the previous 5 years. Reported vaccination coverage was (14%, 95%CI 12.5–14.7) in those aged 45–54 years, peaked in those 60–64 years (23%, 95% CI 21.5–23.7), and was lowest in those >85 years (5%, 95% CI 3.9–6.4). In adjusted analyses other factors associated with vaccination included being female (aOR 2.06, $P<0.01$) and higher income (aOR 1.23, $P<0.01$). Groups less likely to report vaccination included those born in a non-English speaking country (aOR 0.44, $P<0.01$), smokers (aOR 0.58, $P<0.01$) and those living alone (aOR 0.71, $P<0.01$).

The association of vaccination with income, together with the rising burden of adult pertussis, increasing use of grandparent care, warrants consideration of pertussis vaccination strategies for adults.

Determining best strategies for maternally-targeted pertussis vaccination using an individual based model

Presenter: Patricia Campbell, The University of Melbourne, Victoria

Authors: PT Campbell, J McVernon and N Geard

Control of pertussis remains problematic, despite implementation of high-coverage mass vaccination more than sixty years ago. Cocoon and/or maternal vaccination have been implemented in Australia, the United States and the United Kingdom as emergency measures to protect vulnerable infants, who are at greatest risk of severe outcomes. Questions remain about the relative benefit of maternal vaccination over cocooning, and whether repeat immunization is required for every pregnancy.

We simulated pertussis transmission and maternally-targeted vaccination strategies within an individual-based model framework characterizing individuals by their sex, age and family composition. We compared infant disease in vaccinating and non-vaccinating households and estimated household and population impacts of maternal and cocoon vaccination strategies.

At the population level, maternal vaccination at 80% coverage more than halved pertussis incidence in infants under 2 months old. The risk of pertussis among first-born infants of mothers not antenatally immunised was 5 times that of immunised mothers. Revaccination with each pregnancy was required to ensure equivalent protection to subsequent siblings. Compared to maternal vaccination, cocooning was less effective at reducing infant incidence at the population level and provided no direct protection to infants once pertussis was introduced into a household. Our results inform design of these maternally-targeted strategies.

Late Breakers
Chair: Paul Armstrong

A multi-jurisdictional outbreak of hepatitis A associated with consumption of frozen berries

Presenter: Marion Easton, Department of Health and Human Services, Victoria and OzFoodNet

Authors: M Easton, F Romanes, M Antoniou, J Gregory, B Polkinghorne, R Stafford, S Bowden, L Tracy, J McMahon and the OzFoodNet Network

Introduction: Routine follow-up of three locally acquired hepatitis A (HAV) infections in Victoria in January and February 2015 identified a common risk factor of consuming the same brand of frozen mixed berries. An additional case notified in NSW also reported this risk factor which led to the recall of the product and triggered a multi-jurisdictional outbreak investigation co-ordinated by OzFoodNet.

Methods: All jurisdictions reviewed HAV cases with onset of symptoms after 1 October 2014 who had spent any time during their acquisition period in Australia. Sera from cases were submitted for genotyping and sequencing. A food safety investigation and case control study were conducted.

Results: As of 23 April 2015, there were 31 HAV cases who met the definition for a confirmed outbreak case (based on genotyping and sequencing), 12 probable outbreak cases and 39 possible cases. Twenty-six confirmed cases had consumed the same recalled brand of mixed berries and three were secondary cases. Five probable cases also consumed this same brand of berries. A statistically significant association with illness and consumption of the mixed berries was demonstrated in the preliminary univariate analysis (OR 165; 95%CI 17 – 2000; $p<0.0001$).

Conclusion: There is strong epidemiological and laboratory evidence for an association with consumption of mixed berries and HAV.

Case-study: A retrospective assessment of the impact of Ebola virus disease (EVD) on mortality and health seeking behaviours in a rural village in Kailahun District, Sierra Leone, 2015

Presenter: Clair Mills, Medecins Sans Frontieres

Authors: G Caleo, J Duncombe, K Lokuge, C Mills, F Jephcott, E Looijen, R Kremer, JS Squire and J Greig

Introduction: A state of emergency due to Ebola virus disease (EVD) was declared in Kailahun District on 12 June 2014; Medecins Sans Frontieres (MSF) opened an Ebola Management Centre (EMC) on 29 June. A total of 565 confirmed EVD cases were reported in the district; however official figures do not reflect the true magnitude of the outbreak.

Methods: An exhaustive retrospective mortality survey of one highly affected rural village was conducted. A mixed-method case study approach was employed. All consenting households in the selected village were interviewed. In households with suspected EVD cases or deaths, records were cross-matched against the EMC patient list and the Ministry of Health and Sanitation community burials database. Qualitative interviews were carried out with key local informants and in 20 randomly selected households.

Results: All households (n=240) in the village participated in the study, a total of 1120 people. 39 deaths were reported during the recall period. There were 28 EVD confirmed and probable cases. 15 were confirmed in the EMC; 13 of those (86.7%) died. A further thirteen probable cases died in the community; 7 (54%) of these were not detected by the surveillance system.

Qualitative findings will also be presented.

Conclusions: This case study presents, on a small scale, the impact of EVD in rural Sierra Leone. Overall mortality rates were above emergency thresholds; there was significant under-notification of cases. These findings underline the importance of early engagement with the community.

Public health perspective of phase III results of an investigational herpes zoster vaccine

Presenter: Michael Nissen, GSK, Vaccines Value and Health Sciences, Singapore

Authors: L Varghese, M Nissen, A Olivieri and D Curran

A new investigational subunit vaccine (HZ/su) containing the varicella-zoster virus glycoprotein E and the AS01B Adjuvant System has shown a reduction in herpes zoster (HZ) incidence by 97.2% (95% CI: 93.7%–99.0%; $P<0.0001$) compared to placebo in subjects ≥50 years old. Vaccine efficacy did not decrease with age. The potential public health impact of the vaccine was assessed in the Australian population.

A multi-cohort static model was developed in MS Excel. Model inputs included Australian specific data for (1) demographics and natural mortality rates, (2) incidence of HZ and post-herpetic neuralgia (PHN); and vaccine efficacy estimates from a recently published phase III clinical trial. The model projected the number of HZ and PHN cases avoided by vaccinating the Australian population aged ≥60 using a lifetime time horizon.

The projected number of HZ cases avoided would be: 1,077,966; 841,149; 679,542, assuming 0%, 2% and 4% annual waning rates of HZ/su efficacy, respectively. The corresponding PHN cases avoided would be: 191,061; 146,977 and 117,328. The number needed to vaccinate to prevent one case of HZ ranged from 4.25–6.74.

If introduced, the new candidate vaccine could prevent between 0.68 and 1.1 million cases of HZ compared to no vaccination in ≥60 year olds.

Risk Assessment of Hepatitis A and Transfusion–Transmission associated with Multi-jurisdictional Berry Outbreak

Presenter: Veronica Hoad, the Australian Red Cross Blood Service, Perth

Authors: V Hoad, J Pink, C Seed, P Kiely and A Keller

Background: Hepatitis A is transfusion-transmissible, although case reports are rare. On 14 February 2015 an imported berry mix recall was issued in Australia due to potential hepatitis A contamination. The Blood Service implemented recall and deferral interim instructions for exposed donors as a precaution until a risk assessment was completed.

Methods: Modelling of the risk to the blood supply was undertaken using the following parameters: the number of fresh donations during the at risk period (385,889), a 2% berry consumption, 0.01% or 0.1% attack rate, asymptomatic viraemia of 21 days, 30% asymptomatic infection, 0.41% immunity in recipients and 1.5% severe infection rate.

Results: The risk of an infected donation was negligible at approximately 1 in 3,000,000 donations. Using a 10 factor safety margin the risk of a symptomatic infection occurring in a recipient was estimated at 1 in 735,688.

Conclusion: The risk of a case of transfusion–transmission of hepatitis A associated with this outbreak was not deemed to exceed tolerable risk and thus interim risk management actions were ceased.

Novel MRSA outbreak in the Illawarra Shoalhaven district, August 2013–June 2014

Presenter: Chatu Yapa, Health Protection NSW and National Centre for Epidemiology and Population Health, The Australian National University

Authors: C Yapa, D Cordery, M O'Sullivan, J Harris, D Mayne, J McAnulty and C Boutlis

Methicillin-resistant *Staphylococcus aureus* (MRSA) is a significant hospital-acquired pathogen. A novel MRSA strain was identified in the Illawarra–Shoalhaven district in August 2013, causing an outbreak in nine hospitals. By June 2014, this strain accounted for 45% of all hospital MRSA. We conducted a case-control study to assess risk factors for transmission of this strain.

Cases, defined as hospital in-patients with the novel strain were compared with control patients with other hospital-acquired MRSA strains isolated between 29 August 2013 and 30 June 2014. We collected demographic, clinical and hospitalisation information. Whole genome sequencing (WGS) was conducted on available novel isolates.

We compared 67 cases with 112 controls. The median age of cases was 79 years, 66% were male and 24% lived in nursing homes. In the 90 days before diagnosis, 73% of cases were admitted to ≥2 hospitals compared with 47% of controls ($P< 0.01$). WGS for 60 isolates identified nine unique clades. Analysis of these identified several clusters – two associated with one ward and two with a single clinician.

Despite the lack of specific risk factors, the novel strain was rapidly transmitted through several facilities. WGS may allow for better definition of transmission pathways and characterise MRSA outbreaks.

Psittacosis outbreak in veterinary university demonstrating novel source of transmission

Presenter: Bridget Doyle, Public Health Unit, Murrumbidgee Local Health District, Albury NSW.

Authors: J Chan, B Doyle, V Sheppeard, J Branley, M Gabor, K Viney, H Quinn and J Heller

A local public health unit was notified in November 2014 of a cluster of respiratory illness in 4 people, all associated with a veterinary school. Active case finding identified another case at a local horse stud. It was identified that all cases had a common exposure to the abnormal equine foetal membranes of Mare A. This tissue subsequently tested PCR positive for *Chlamydophila psittaci*. Active case-finding did not identify any cases of atypical pneumonia not linked to the membranes.

We interviewed university and stud farm staff exposed to the abnormal membranes regarding clinical symptoms, investigations and potential exposures.

Our investigation found that nine people were exposed to the foetal membranes of Mare A. Of these, five cases of psittacosis (three probable, two suspected) were identified. Contact with birds was not associated with illness. People who had direct contact with the abnormal membranes were more likely to develop disease.

Spread from horses has not previously been considered an important pathway for transmission. Previous studies have demonstrated the presence of *C. psittaci* infection in horses. To our knowledge, this is the first report of *C. psittaci* transmission from horse to humans.

Day 2: Harnessing new technologies

Responding to Ebola virus disease: Australia and beyond
Chair: Dr Sonya Bennett

This Breakfast Forum will provide an opportunity for discussion about all things Ebola – from International and National preparedness, to clinical trials of vaccines and therapeutics, and front line experiences of working in West Africa. Short presentations will be followed by a lively question and answer session. Not to be missed.

Conducting clinical trials in high transmissions settings and international issues

Professor Peter Horby

Operation Uplift and planning exercises for EVD

Dr Jeannette Young, QLD CHO

Border preparedness and implementation issues

Dr Jenny Firman, Principal Medical Adviser, Office of Health Protection, Department of Health

Clinical work and other experiences in an EVD affected country

Dr John Gerrard, IDP GCH

Plenary 2. Microbiological challenges and opportunities for controlling infectious diseases
Chair: John Bates

Foodborne and zoonotic disease surveillance: genomics, metagenomics, and the road ahead

Speaker: Dr John Besser, PhD, Deputy Chief, Enteric Diseases Laboratory Branch, Centers for Disease Control and Prevention, Atlanta USA

Advanced technology is changing the field of microbiology at an unprecedented rate, opening up opportunities and challenges for public health that were not imaginable a few years ago. In the area of foodborne disease surveillance, successive laboratory and epidemiology innovations during the last 20 years have increasingly made it possible to detect and solve distributed outbreaks caused by problems in the food supply that would not otherwise have been recognised. Will the real-time use of NGS technology make it possible to detect more outbreaks more quickly and make them easier to solve? The U.S. Centers for Disease Control and Prevention, in collaboration with multiple U.S. and International agencies and all 50 U.S. states, initiated a nationwide real-time whole genome sequencing (WGS)-based surveillance project for *Listeria monocytogenes* in late 2013. Impacts were immediate, with *Listeria* outbreaks dominating headlines. Lessons learned will be presented, along with a description of the nationwide wgMLST-based infrastructure developed to monitor foodborne diseases. Uses of NGS technology for other diseases such as Ebola and influenza will be briefly mentioned along with applications for metagenomics that are rapidly emerging from the realm of science fiction into clinical diagnostic and public health practice.

Winds of change: the evolving diagnostic microbiology laboratory – a personal reflection

Speaker: Dr Jenny Robson, Sullivan Nicolaides Pathology (SNP), Brisbane

Clinical microbiology is undergoing rapid change not only in the way things are done but also the physical organization of facilities. Microbiology laboratory networks are consolidating with centralisation of many of their functions. This talk will examine the changing Australian landscape with respect to private microbiology and describe those forces that have reshaped laboratories recently. These include the widespread application of molecular technologies including multiplexed panels, and more recently microarrays and automation of molecular tests; matrix-assisted laser desorption ionization–time of flight mass spectrometry methods and their replacement of other identification systems for microorganisms; and the shift to liquid-based specimens enabling the introduction of partial and full laboratory automation. The impact on patient care and on public health surveillance including enteric pathogens, respiratory viruses, and sexually transmitted infections will be discussed together with the challenges that these changes pose. No doubt even newer technologies such as next generation sequencing will appear on the scene and be embraced in the clinical laboratory armamentarium at a rapidly evolving pace.

Communicable Diseases Surveillance and Response in Pacific Island countries and territories: Challenges and Opportunities

Speaker: Dr Salanieta Taka Saketa, Epidemiologist, Research, Evidence and Information Programme, Public Health Division, Secretariat of the Pacific Community, Suva Office, Fiji

High speed global communication and travel poses risks of spread of communicable diseases within a matter of days and weeks and the natural protective factors of distance and islands is inconsequential. Pacific Island Countries and Territories (PICTs) are faced with the challenges and opportunities of implementing surveillance systems that can communicate information and prepare communities; faster than the spread of these diseases. Innovative strategies and technologies are needed to enhance surveillance and response capabilities in PICTs both at the national and regional level, bearing in mind the unique challenges of the region.

Foodborne Diseases and Outbreaks
Chair: Ben Polkinghorne

Whole genome sequencing can assist in monitoring community outbreaks of salmonellosis

Presenter: Anastasia Phillips, Centre for Infectious Disease and Microbiology, ICPMR, Westmead, New South Wales

Authors: A Phillips, Q Wang, N Holmes, C Furlong, C Sotomayor, P Howard, K Ward and V Sintchenko

Background: *Salmonella* Typhimurium (STM) is an important aetiological agent in foodborne outbreaks. Subtyping is critical to outbreak investigation, yet current techniques (e.g.. multilocus variable number tandem repeat analysis, MLVA) often do not provide sufficient discrimination. Whole genome sequencing (WGS) of STM may have greater discriminatory power to support disease control.

Methods: STM isolates of a single, endemic MLVA type causing two independent outbreaks along with sporadic cases in NSW during 2014 were selected for WGS. DNA extracted from case and environmental isolates was sequenced (HiSeq, Illumina) and variation was assessed by single nucleotide polymorphism (SNP) analysis. SNP analysis was compared to known epidemiology.

Results: Thirty-four outbreak, 17 sporadic, one secondary and 10 environmental isolates were included in the analysis. WGS analysis supported known epidemiological hypotheses and genomes of within-outbreak isolates were nearly identical. Sporadic cases differed from outbreak cases by a small number of SNPs. Among sporadic cases, WGS detected previously unrecognised mini-clusters.

Conclusions: Clustering of STM cases based on WGS data correlates well with epidemiology and further differentiates sporadic from outbreak cases. WGS offers the opportunity to further cluster sporadic isolates within endemic MLVA types of STM and to improve the resolution of public health laboratory surveillance.

Incidence and risk factors for hospitalisation with gastroenteritis in a cohort of older Australians

Presenter: Yingxi Chen, The Australian National University

Authors: Y Chen, B Liu, K Glass and M Kirk

Background: Infectious gastroenteritis is an important cause of morbidity in Australia. We aimed to estimate the incidence and risk factors for gastroenteritis related hospitalisation in older Australians.

Methods: The 45 and Up Study is a large-scale Australian cohort of NSW aged ≥45 years in 2006–2008. Self-reported demographic data from 265,440 participants were linked to hospitalisation data. We used Stata 12 to estimate hazard ratios (HR) of incident hospitalisation for gastroenteritis using Cox regression, adjusting for socio-demographic, health and behavioural status, with age as the underlying time variable.

Results: There were 6,077 incident gastroenteritis admissions over 1,111,000 person years. Incidence increased exponentially with increasing age; incidence was 0.24 per 1,000 (95% CI 0.22–0.25) in 45–54 year olds and 2.18 per 1,000 (95% CI 2.02–2.36) in those aged 85+ years. After adjustment, gastroenteritis hospitalisation was significantly associated with proton pump inhibitors (PPIs) usage (HR 1.57, 95% CI 1.49–1.67), and no fruit and vegetable consumption (HR 1.93 95%CI 1.36–2.75). In addition, the risk was over 300% greater for those with poor health than those with excellent health.

Conclusion: Hospitalisation for gastroenteritis is more common in the elderly, those in poor health and those using PPIs. Prevention strategies could potentially focus on dietary modification.

Outbreak of *Salmonella* Typhimurium 9 infection amongst Christmas function attendees, December 2014

Presenter: Caitlin Graham, Communicable Disease Control Branch, SA Health, South Australia

Authors: JH Stephens, C Graham, EJ Denehy and AP Koehler

On 15 December 2014, the Communicable Disease Control Branch (CDCB) South Australia was notified of an outbreak of gastrointestinal illness amongst attendees of a recent Christmas function. The objective of the investigation was to identify the potential source of infection and establish appropriate intervention strategies to prevent further illness. A retrospective cohort study was conducted among 24 guests of the function. A telephone questionnaire was used to collect information on demographics, illness, and menu items consumed. Descriptive statistics, univariate analyses, and Poisson regression were performed. The questionnaire response rate was 96%. The attack rate was 58.3%. Seven cases were confirmed as having a *Salmonella* Typhimurium phage type 9 infection. There was a significant association between the consumption of tiramisu and illness (RR 11.2, 95% CI 1.65–75.93, $P=0.0004$). The ingredients of the homemade tiramisu included raw eggs, and this was identified as a high risk food item. This outbreak adds to the ongoing evidence that produce made from raw egg is hazardous. Education should be extended to cooking in the home.

Illness in children associated with the consumption of raw unpasteurised milk

Presenter: Nectaria Tzimourtas, Department of Health and Human Services, Victoria

Authors: D Thaker, N Tzimourtas and J Gregory

Background: In October 2014, a cluster of Haemolytic Uraemic Syndrome (HUS) cases and a separate cluster of Cryptosporidiosis cases, all living in the same outer Melbourne metropolitan area were detected in Victoria. An investigation was initiated to determine if there was a common source.

Methods: Cases of HUS were interviewed using the national OzFoodNet STEC/HUS questionnaire. The Department used its own Cryptosporidiosis questionnaire with food exposures added to interview the cases of Cryptosporidiosis. Responses were compared for a common link. Case finding was conducted with other state and territories through OzFoodNet and CDNA.

Results: Three cases of HUS and two cases of Cryptosporidiosis were found to have consumed the same brand of unpasteurised milk, sold as 'bath' milk, in their incubation period. One case of HUS died. Public health warnings and subsequent media resulted in Dairy Food Safety Victoria tightening controls on the availability of dairy products not intended for human consumption such as unpasteurised milk.

Conclusion: The consumption of unpasteurised milk is a well-documented risk factor for gastrointestinal diseases including Cryptosporidiosis and Shiga toxin producing E. coli infection and was the suspected source of illness. The sale of unpasteurised milk for human consumption in Australia is prohibited.

Epidemiology of bacterial toxin-mediated foodborne outbreaks in Australia

Presenter: Fiona May, Australian Government Department of Health and Australian National University, Australian Capital Territory

Authors: B Polkinghorne, E Fearnley and F May

Foodborne outbreaks caused by bacterial toxins such as *Clostridium perfringens, Staphylococcus aureus* and *Bacillus cereus* are an often overlooked cause of morbidity. We aim to describe the epidemiology of these outbreaks in Australia, establish those groups at highest risk and identify where preventative measures should be focussed.

All foodborne and suspected foodborne outbreaks in Australia are collated in the OzFoodNet Outbreak Register. We extracted data on all toxin mediated outbreaks notified to the register between 2001 and 2013. Descriptive analyses were undertaken using Stata 13.

There were 272 toxin mediated outbreaks reported, of which 39% were laboratory confirmed. These outbreaks affected 4,066 people, including 70 hospitalisations and 13 deaths, 12 of which occurred in aged care facility residents. Clostridium perfringens was suspected or confirmed as the causative agent in 63% of outbreaks. Restaurants (31%) and aged care facilities (32%) were the most commonly reported settings for outbreaks and inadequate temperature control of pre-cooked foods was the most commonly reported contributing factor (60%).

Toxin mediated outbreaks cause significant preventable morbidity in Australia, and disproportionately affect those living in aged care facilities. Public health efforts aimed at improving pre-cooked food storage habits could help to reduce the magnitude of this problem.

First outbreak of locally acquired hepatitis E virus infection in Australia

Presenter: Catriona Furlong, NSW Health Protection

Authors: C Yapa, C Furlong, A Rosewell, K Ward, S Adamson, J Kok, S Bowden, C Shadbolt, L Smedley, M Ferson, T McNeill, M Staff, V Sheppeard and J McAnulty

Background: In May 2014, an outbreak of hepatitis E linked to a single restaurant was identified.

Methods: A case series was conducted using a standardised questionnaire. Co-diners were also interviewed and tested. Further cases were identified testing patients in whom viral hepatitis screening was requested at a major private and public reference laboratory where other infections had been excluded, and by alerting medical practitioners to the outbreak. Serum isolates were genotyped and sequenced. Implicated foods were traced back to the source.

Results: We identified 17 cases who reported dining at a single restaurant in their incubation periods. Consumption of the pork liver pâté was identified as the highest risk for developing HEV. Seven additional locally acquired cases were detected due to enhanced surveillance. All had consumed pork or pork liver in their incubation period. Seventeen of 24 cases were genotype 3. Pork livers were traced back to a single farm.

Conclusions: This is the first HEV outbreak in Australia. We recommend that clinicians in Australia consider the diagnosis of HEV in patients presenting with a compatible illness in the absence of overseas travel history. Further, the food service industry should ensure that pork liver products are thoroughly cooked.

Public Health Surveillance 2
Chair: Vicky Sheppeard

Trends in species, serotype and antimicrobial susceptibility of *Shigella* isolates, Victoria, 2000–2015

Presenter: Mary Valcanis, University of Melbourne

Authors: M Valcanis, CR Lane, A Kuzevski and B Howden

Aim: To report the trends in species, serotype and antimicrobial resistance in isolates of *Shigella* in Victoria.

Methods: We performed identification, speciation, serotyping and *S. sonnei* biotyping on all isolates of *Shigella* submitted to the MDU PHL between 2000 and 2015. Susceptibility testing to 12 antimicrobials (azithromycin since 2014) was performed by agar dilution for epidemiological purposes.

Results: Over 1,600 confirmed *Shigella* isolates were included in the analysis, ranging from 51(2003) to 297(2014) annually. The dominant species was *S. sonnei*, of which biotype g was most common in all years, followed by *S. flexneri*. Less than 10 cases of *S. boydii* and *S. dysenteriae* were seen in any year. Emerging serotypes, such as *S. flexneri* 1c and 4d, have been identified in Victoria. Ninety-six per cent of *Shigella* isolates were resistant to at least one antimicrobial. Decreased susceptibility to ciprofloxacin was noted in 2003; by 2014, 62% (75/122) of *S. sonnei* isolates exhibited full ciprofloxacin resistance. In late 2013, azithromycin resistance was first observed in *S. flexneri* 3a among men who have sex with men. Resistant strains, previously seen in overseas travellers, have been increasingly identified amongst locally acquired infections.

Conclusions: Isolate characterisation is vital to monitor and track *Shigella* types circulating and emerging in the population. Resistance profiles must be available to guide appropriate treatment.

A NSW program to improve management of people newly diagnosed with HIV

Presenter: Jeremy McAnulty, Communicable Diseases Branch, Health Protection NSW, New South Wales

Author: B Telfer, V Bowden, M Middleton, V Sheppeard, J McAnulty and C Selvey

Background: In NSW half of new HIV diagnoses are made by general practitioners often inexperienced in HIV. The *NSW HIV Strategy 2012–2015* calls for enhanced case support at the time of diagnosis to help achieve prevention and treatment targets. In May 2013 the HIV Support Program (HSP) commenced.

Methods: A laboratory notification of HIV infection triggers a phone call to the diagnosing doctor from a local HSP Coordinator with HIV expertise. The Coordinator provides support and advice so that the newly diagnosed patient receives appropriate clinical management, psychosocial support, counselling about HIV treatment and prevention of transmission of HIV to others, contact tracing assistance and linkage to specialist, community and peer support services.

Results: From 9 May 2013 to 30 September 2014 the HSP supported 186 doctors of whom 156 (84%) were HIV-inexperienced primary care doctors. Of 186 doctors supported, 114 intervention forms were returned by Coordinators; 103 (90%) doctors welcomed support by the HSP, 61 (54%) had just made their first HIV diagnosis, 59 (52%) were aware of how to do contact tracing and 49 (43%) were interested in shared care.

Conclusions: The HSP has been well received by diagnosing doctors. Further evaluation will be done from 2015.

The National Notifiable Diseases Surveillance System (NNDSS) 1991–2011: Expanding and improving

Presenter: Katherine Gibney, Monash University, Victoria

Authors: K Gibney, A Cheng, K Leder and R Hall

Background: We reviewed the system characteristics, data quality and performance of the NNDSS over its first 21 years of operation.

Methods: Line listed data for all cases notified to NNDSS from 1991–2011 were reviewed. Changes in conditions notified, data fields included, data completeness, and notification delays were examined at national and jurisdictional levels.

Results: The number of notifiable conditions increased from 34 to 65, with 22 conditions notifiable by all jurisdictions from 1991–2011. Demographic data were near complete except Indigenous status which was missing for 56% of cases nationally (jurisdictional range 13%–81%). Vaccination status and travel data were missing for 89% and 76% of relevant notifications. Data completeness improved over the study period for 24/26 (92%) variables. Median delay to NNDSS notification was 8 days (interquartile range 4–17 days), varying between jurisdictions from 5 to 15 days ($P<0.001$) and decreasing from 11 days in 1991–1997 to 5 days in 2005–2011 ($P<0.001$).

Conclusion: NNDSS expanded during its first 21 years. Overall, data completeness and notification timeliness improved, but there were marked inter-jurisdictional differences. Examination of delays and low completion of specific data field is important for devising strategies to improve future data collection.

Implementation of an automated notifiable disease surveillance tool in Victoria

Presenter: Stacey Rowe, Communicable Disease Epidemiology and Surveillance, Department of Health, Victoria

Authors: S Rowe, J Lawrie, B Cowie, J Carlin and N Stephens

Background: The increasing volume of communicable disease notifications presents a challenge to conventional surveillance programs. In Victoria this led to the development of an automated outbreak detection tool, the Public Health Outbreak Surveillance System (PHOSS).

Methods: Based on the model developed by Farrington et al in the UK Health Protection Agency, PHOSS is a web application written in R that automatically receives new notifications from the surveillance database daily, with retrospective data back to 1991. It provides a list of notifiable diseases ordered by how much they exceed the calculated warning threshold ('Exceedance score'), together with a range of user interfaces for examining time series and disease maps.

Results: PHOSS has been implemented in the weekly surveillance meeting in Victoria and is used to highlight possible outbreaks, and to examine temporal and geographic trends of conditions of interest. A number of worked examples of outbreaks detected by PHOSS and demonstration of the tool's use in public health practice will be presented.

Conclusion: Together with existing communicable disease surveillance approaches, automated outbreak detection tools can act as a safety net in detecting additional outbreaks, and can also augment the utility and accessibility of existing datasets to support daily public health practice.

Can adverse event signals be detected by electronic GP data?

Presenter: Rob Menzies, National Centre for Immunisation Research and Surveillance

Authors: L Trihn, K Macartney, A Dey, C Chiu and P McIntyre

Background: In early 2011 there was an increase in reports of severe injection site reactions (ISR) in adults following receipt of pneumovax.

Methods: Data from the General Practice Research Network (GPRN) were analysed retrospectively to determine whether the increase in ISR could be detected.

Results: Electronic clinical records of 95,760 pneumococcal and 683,829 influenza vaccine doses administered between 2002 and 2012 were received, from 1,088 participating general practitioners (GPs) in 402 practices. The seasonality and age distribution of vaccinations, dose numbers and revaccination intervals were consistent with recommended usage of both vaccines. Following receipt of Pneumovax, there were 233 ISR (243 per 100,000), including 40 severe ISR (42). As expected, pneumovax ISR for second or subsequent doses (398 per 100,000), were higher than the rate for first doses (239). However, the number of local reactions following receipt of Pneumovax between January and March 2011 (just before the public announcement of a batch recall) was only 3 cases, similar to the same period of the previous year (5 cases between January and March 2010).

Conclusions: The GPRN data appear to be broadly coherent and consistent with recommended usage. However, a larger GP network would be needed for rapid AEFI surveillance.

A web-based system for mass-gathering surveillance: Experience from the 8th Micronesian Games, 2014

Presenter: Paul White, Research Evidence and Information Programme, Public Health Division, Secretariat of the Pacific Community

Authors: S Saketa, E Johnson, S Gopalani, E Edward, C Loney, A Mercier, T Toata, C Leppers, R Wojcik, S Lewis, A Roth, Y Souares and D Hoy

Background: Web-based surveillance tools provide near-real time updates of disease patterns, facilitating prompt investigation and response. A web-based surveillance platform based on SAGES developed by JHU/APL and SPC was used in partnership with Pohnpei public health to enhance surveillance at the 8th Micronesian Games hosted by Pohnpei State in July 2014' a mass gathering of 1,700 participants from nine Micronesian states and countries.

Methods: The mass gathering surveillance comprised daily reporting of 8 syndromes using standardised case definitions from 11 sentinel points-of-care with data entered in a centralised location and summarised in daily situation reports (Sitreps).

Results: 5,640 encounters and 408 syndromes were recorded. The Sitreps helped monitor public health threats, and in particular an ongoing measles outbreak, identifying early increases of acute fever and rash as well as diarrhoea.

Conclusion: The enhanced surveillance helped mitigate public health risks. SAGES allowed easy data entry, analysis and accelerated Sitrep production while cloud storage was advantageous in reducing local server costs, facilitating remote support and analysis and increasing off-site data security. As a successful tool, SAGES features in the public health surveillance sustainability plan of Pohnpei State.

Influenza and Other Respiratory Diseases
Chair: Rhonda Owen

Simulating enhanced pandemic influenza surveillance strategies

Presenter: Nicholas Geard, Melbourne School of Population and Global Health, Victoria

Authors: N Geard, A Black, J Ross, J McCaw and J McVernon

The collection of enhanced epidemiological data in the early stages of an influenza pandemic ("first few hundred" studies) can help to estimate its likely impact, and inform decision making about the appropriate scale of response. The key characteristic of interest is the reproduction number R_0, which provides information on how quickly a pathogen is likely to spread through a population.

An effective surveillance plan must balance the need to collect sufficient data to enable accurate estimation of disease parameters against the sustainability of obtaining quality data alongside other response activities. Here we describe a method for estimating epidemic parameters from imperfectly observed data, together with a computational model that simulates both the disease outbreak and the surveillance strategy. The output of the disease model provides a 'true' picture of all cases occurring in the outbreak, while the output of the surveillance model provides the subset of cases that we anticipate will be detected using that particular strategy, and hence which are available for the purposes of parameter estimation.

Thus we can evaluate and compare alternative enhanced surveillance strategies, and gain insight into the required duration of enhanced data collection, based on the precision of the estimated epidemic parameters.

Influenza vaccine effectiveness for hospital and community patients, Auckland, New Zealand, 2014

Presenter: Heath Kelly, Victorian Infectious Diseases reference Laboratory, Melbourne, Australia

Authors: N Pierse, H Kelly A Bissielo, S Radke, QS Huang, M Baker and N Turner on behalf of the SHIVERS investigation team

Background Trivalent seasonal influenza vaccines have been used for more than 60 years but published estimates of vaccine effectiveness (VE) from observational studies vary widely. We aimed to estimate the protection afforded by trivalent inactivated influenza vaccines, in both primary care and hospital settings, in a well characterised urban population in Auckland during 2014.

Methods Using two different comparison groups, patients with no virus detected and those with a non-influenza respiratory virus detected, VE estimates were made using a test-negative study design. VE was stratified by community and hospital patients, age group, and influenza type and subtype.

Results 1039 hospitalised and 1154 community patients met all the study inclusion criteria and had a respiratory sample tested for influenza and other respiratory viruses. The adjusted VE using all influenza negative controls was 42% (95% CI: 16%–60%) for hospital patients and 56% (95% CI: 35%–70%) for community patients. No significant difference was seen when patients testing positive for a non-influenza respiratory virus were used as the control group. No protection was found against influenza A(H3N2), although power was limited.

Conclusion This study contributes to the validation of the test negative design and confirms that inactivated influenza vaccines continue to provide modest but significant protection against laboratory-confirmed influenza.

FluMum: Influenza and pertussis vaccine uptake in pregnancy among a cohort of 7000 women recruited from six study sites in Australia, 2012–2014

Presenter: Ross Andrews, Deputy Director, Menzies School of Health Research, Charles Darwin University, Casuarina, NT

Authors: R Andrews, KA O'Grady, T Nolan, P Richmond, N Wood, H Marshall, S Lambert, M Chatfield, L McHugh, SA Le-Gros Wilson, P Binks, K Watson, J Zenchyson, E Clarke, M Kefford, V Hill and C Talbot

Background: Influenza vaccine is recommended in pregnancy (nationally funded), whilst pertussis vaccination is now proposed for every pregnancy in recent Immunisation Handbook changes. Uptake of neither of these vaccines is systematically monitored in Australia.

Methods: We analysed self-reported data on influenza and pertussis vaccine uptake within our ongoing "FluMum" cohort study of mother-infant pairs recruited from participating sites in Darwin, Brisbane, Sydney, Melbourne, Adelaide and Perth from 2012–2014 inclusive. The data presented here are from preliminary analyses that may be subject to change.

Results: Among 7184 women, self-reported uptake of influenza vaccine in pregnancy was 33.2%, most women (66.7%) had not been vaccinated, 0.4% were unsure and 0.7% were not recorded. Coverage rates were similar in 2012 (32%) and 2013 (30%) but marginally higher in 2014 (36%). Factors associated with influenza vaccine uptake will be presented. In 2014, 6.4% of 1324 women self-reported receiving pertussis vaccination in pregnancy.

Conclusion: Immunisation of pregnant women is a significant weakness in immunisation programs where uptake is poor. There is a critical need to drive improvements, particularly for influenza and pertussis, where potential to prevent death and disability from these infectious diseases is paramount.

Electronic health record extraction from general practice for monitoring influenza vaccine coverage and effectiveness

Presenter: Annette Regan, Department of Health Western Australia

Authors: AK Regan, R Gibbs, L Tracey and PV Effler

Background: Influenza vaccination is recommended annually to prevent serious infection. Reliable and timely estimates of vaccine uptake are necessary to better inform prevention programs; effectiveness estimates inform annual antigen selection. However, limited annual data are available to produce these estimates. We explored the use of electronic health record (EHR) extraction from general practice (GP) for these purposes.

Methods: Data from 142 GPs were extracted by the Western Australia Department of Health. Data were aggregated to estimate the proportion of patients immunised and vaccine effectiveness based on pathology results. In 2015, a validation study was conducted to determine the validity of estimates derived from EHRs.

Results: According to EHRs extracted from 650,701 patients, 16.3% of the population received an influenza vaccination between 2012 and 2014. Vaccination coverage was highest in adults ≥65 years with medical conditions (63.1%) and lowest in children <2 years (7.3%). A total of 7,207 pathology testing results were extracted; vaccine effectiveness ranged from 30–56% (2012:30%; 2013: 56%; 204: 46%). Validation results will be available in March 2015.

Conclusions: Systems employing data extraction of EHRs could provide more timely estimates of influenza vaccination coverage and effectiveness. Such information is valuable for improving prevention programs.

Peer-led, student-centered interventions the key to student health care worker influenza vaccination

Presenter: Munyaradzi G Nyandoro, Alumnus School of Medicine University of Notre Dame, Fremantle (UNDF), Western Australia

Authors: MG Nyandoro, D Kelly, D Macey and DB Mak

Background: Vaccination is the most effective influenza prevention strategy recommended for all health care workers (HCW), including students. However Australian HCWs' uptake is poor (16.3% – 58.7%).

Methods: Self-reported influenza vaccination uptake among student HCWs at UNDF was measured using on-line surveys to assess the impact of a peer-led vaccination campaign on uptake.

Results: In 2013 (pre-campaign) influenza vaccination uptake was 36.3% (95% CI = 31.8%–40.8%); students identified awareness, cost and convenience as key barriers. The campaign used posters of, and presentations by, current HCW students and key staff to raise awareness of the importance and benefits of vaccination, and provide information on how to access free and low-cost vaccination. In 2014 (post-campaign), vaccination rate increased to 55.9% in 2014 (95% CI = 52.2% – 59.6%). Multivariate logistic regression showed that HCW students in 2014 (OR 2.2, CI 1.7–2.9, $P<0.001$), and those who were eligible for government-funded vaccine (OR 12.3, 6.3–24.0, $P<0.001$), enrolled or enrolled in nursing/midwifery (OR 1.6, 1.1–2.4, $P=0.016$), or employed as a HCW (OR 1.6, 1.5–2.6, $P<0.001$) were more likely to be vaccinated.

Conclusion: HCW students' influenza vaccination uptake improved significantly following a low-cost, peer-led promotional campaign. This approach can be adapted to other settings.

Seasonal influenza vaccine effectiveness against medically-attended influenza in Victoria, 2014

Presenter: James Fielding, Victorian Infectious Diseases Reference Laboratory

Authors: J Fielding, K Grant, K Carville, J Druce, I Barr and H Kelly

Background: We used a test negative case control design to estimate effectiveness of the seasonal trivalent influenza vaccine against medically-attended influenza during the 2014 influenza season in Victoria.

Method: Patients presenting with influenza-like illness (ILI) to general practitioners (GPs) in a sentinel surveillance network during 2014 were tested for influenza. Cases were influenza positive by PCR and controls tested negative for influenza. Vaccination status was recorded by sentinel GPs. Vaccine effectiveness (VE) was calculated as [(1 – adjusted odds ratio) x 100%].

Results: A total of 427 ILI patients were included in the study. There were 170 cases of influenza, of which 164 (96%) were type A. Receipt of the 2014 vaccine was reported for 123 patients (29%). Adjusted VE against subtype A/H1N1 infection was 23% (95% CI, –40% to 58%) and against A/H3N2 infection was 3% (95% CI, –105% to 54%).

Conclusions: This study suggests that in 2014, the seasonal trivalent influenza vaccine provided relatively low protection against influenza A/H1N1 infection, despite apparent vaccine and circulating strain match. Consistent with other studies, the vaccine conferred no protection against influenza A/H3N2.

Project launch: Linkage of the Australian Childhood Immunisation Register (ACIR) data to state-based health datasets to evaluate and inform Australia's immunisation program

We would like to invite conference delegates to help us celebrate the successful linkage of the ACIR to state-based health datasets in Western Australia and New South Wales. Over the past 3.5 years a strong collaboration between researchers and state and national agencies has led to the linkage of a 17-year birth cohort (over 2 million children) to ACIR and health outcome data, particularly about vaccine preventable diseases. This was achieved through project funding from the Population Health Research Network, as part of the National Collaborative Research Infrastructure Strategy, and researcher support from the National Health and Medical Research Council. We will provide an overview of the project, highlight the achievements to date (including the establishing a process for data release under the Commonwealth data integration policy), and describe the main objectives and planned priorities for analysis. This will be an opportunity to better understand the process of conducting linkage using Commonwealth datasets, propose specific questions that could be answered with our study data, and identify preferred mechanisms for stakeholder feedback.

Short Oral Presentations
Chair: Paul Armstrong

Forensic surveillance: are we missing fatal communicable disease cases?

Presenter: Keith Eastwood, Hunter New England Population Health, Newcastle NSW

Authors: Keith Eastwood, Bev Paterson, Rexson Tse, Leah Clifton, Rod Givney, Allan Cala, Yvonne Tin, Carly Pedersen and Stephen Graves

Background: Communicable disease surveillance systems are geared for cases that present through conventional health services with laboratory confirmation, however, cases of serious infection resulting in rapid death and proceeding directly for coronial examination may be missed.

Methods: A partnership between northern NSW forensic medicine, pathology and public health services was commenced in 2014. It reviewed existing systems for identifying deaths of potential public health significance and developed a case definition, designed an algorithm to aid pathologists in selecting optimal specimens and diagnostic tests, drafted a questionnaire for use by grief counsellors to collect medical information from relatives, modified the forensic patient database to flag patients of interest and set up a pathology alert to facilitate access to laboratory results.

Results: Overall 37 patients of interest have been identified in the initial 10 month review period. Causes of death included pneumonia (15 cases), meningitis (2), non-localised sepsis (5), gastroenteritis (3). Cases study examples will be presented.

Conclusion: Unless forensic surveillance is integrated into notification systems, it is possible for fatal communicable disease cases to go unreported. Our partnership has had broad benefits in optimising specimen collection, selecting appropriate pathology tests, interpreting results, informing public health authorities and improving response times.

The epidemiology of hepatitis A in Queensland, 1988–2014

Presenter: Lisa McHugh, MAE scholar, Queensland Children's Medical Research Institute and Communicable Diseases Unit, Queensland Health and Australian National University, ACT

Authors: L McHugh, SB Lambert and K Viney

Background: In Australia, where hepatitis A (hepA) has low endemicity, the most likely mode of transmission is from common source outbreaks. However this does not appear to be the case in Queensland, where most notified cases occur following international travel. The introduction of hepA vaccine for Indigenous children in north Queensland in 1999 has also contributed to this change in epidemiology over time.

Methods: We present a retrospective case-series analysing the source and incidence of notified cases of hepA infections in Queensland. We examine epidemiologic and virus genotype data to assess if there remains a circulating endemic strain in Queensland.

Results: From 1988–2014 there were 6,748 hepA notifications in Queensland, with a median age of 25 years (28% aged 20–29 years). Major risk factors for hepA acquisition were overseas travel (47%) and locally-acquired, foodborne-related (shellfish/ sun-dried tomato, 25%). Of those who acquired infection from international travel, only 3% reported being vaccinated prior to travelling.

Conclusion: Based on a combination of epidemiologic and genotype data, hepA infections in Queensland are largely acquired from international travel. With targeted immunisation allowing for enhanced disease control, improved vaccination rates in prospective overseas travellers could reduce current case numbers markedly.

Australia's meningococcal C immunisation program: long term impact and implications

Presenter: Glenda Lawrence, University of New South Wales

Authors: G Lawrence and P McIntyre

Background: Australia's national meningococcal C program commenced in 2003 targeting those born after 1983 and aged ≥ 12 months. Many developed countries also implemented programs using a range of strategies and several recently recommended an adolescent booster dose to enhance disease control. Several have recently introduced an adolescent booster dose.

Methods: Age– and region-specific serogroup C and non-serogroup C notification rates were estimated from NNDSS data after adjusting for untyped notifications, and were compared for the pre (2000–2002) and post (2010–2012) program periods. Confirmed vaccine failures 2003–2012 were identified.

Results: Adjusted serogroup C notifications have declined by 96% (95% CI 94–98) compared with an independent reduction in non-serogroup C notification of 55% (95% CI 51–59). Sustained reductions in both C and non-C serogroup categories occurred in all regions and age groups. Serogroup C disease declined in both immunised and non-immunised age groups including those aged <12 months (88% (61–97)). Five vaccine failures were confirmed: two vaccinated in adolescence and three aged 1–3 years. The median period to disease onset was 2 years.

Conclusion: A substantial post-program reduction in serogroup C meningococcal disease with widespread herd effects has occurred concurrently with an independent reduction in non-serogroup C disease. The small number of serogroup C vaccine failures suggests that a booster dose is not yet required. Ongoing monitoring and evaluation of population immunity is essential.

2012–13 HPV serosurvey

Presenter: Alexis Pillsbury, National Centre for Immunisation Research and Surveillance (NCIRS)

Authors: A Pillsbury, H Quinn, L Hueston, S Lesic and P McIntyre

Background: Australia introduced funded HPV vaccination in 2007 for girls aged 12–13 years with catch-up to 26 years of age. Early surveillance data demonstrated genital wart and *cervical intraepithelial neoplasia (*CIN) incidence reduction in women, and herd immunity benefits for males. The program expanded to adolescent boys in 2013. We assessed HPV seroprevalence among males aged 15–39 years in 2012–13, comparing results to those from a 2005 serosurvey prior to female program commencement.

Methods: Residual diagnostic sera were obtained and serum antibody levels to HPV types 6, 11, 16 and 18 measured using Luminex immunoassay. Proportion seropositive was calculated for age groups 15–19, 20–29 and 30–39 years according to pre-defined cut-off values.

Results: Among those aged 20–29 and 30–39 years, proportion seropositive was lower for all types in the 2012–13 serosurvey compared to the 2005 serosurvey. The most notable decrease occurred for HPV6 among those aged 30–39 years; 25.9% to 7.0%. There was no difference in seroprevalence between serosurveys among those aged 15–19 years.

Conclusion: Results provide further evidence of the positive herd immunity impact of female HPV vaccination upon males. Results demonstrate the utility of serosurveillance as a non-invasive indicator of population infection/immunity trends.

The impact of changes in healthcare-seeking behaviour and testing on influenza surveillance.

Presenter: Lisa McCallum, Hunter New England Local Health District

Authors: L McCallum, C Dalton, S Carlson, H Kelly and D Durrheim

Background: There were 37,000 more influenza notifications to the National Notifiable Diseases Surveillance System (NNDSS) in May–October 2014 than during the same period in 2013. However, this increase was not reflected in the Flutracking online community survey of influenza-like illness.

Methods: We used changes in healthcare-seeking behaviour and health provider testing from 2013 to 2014 among Flutracking participants to explore potential impacts on laboratory notified influenza cases. The proportion of Flutracking participants reporting being tested for influenza, and the proportion testing positive were calculated for 2013 and 2014. We calculated the ratio of the 2013:2014 proportions of Flutracking participants reporting being tested for influenza and testing positive for influenza. We applied these ratios to the number of NNDSS notifications to estimate the additional number of notifications due to the increased health-care seeking and testing.

Results: Approximately two-thirds of the increase (24,000) was due to additional people seeking medical care and being tested; the remaining third (approximately 10,000) was due to the increased proportion testing positive.

Conclusion: Accounting for changes in healthcare-seeking behaviour and testing practices enables trends in surveillance data to be interpreted more fully.

Maternal and birth characteristics associated with pertussis during early infancy

Presenter: Lisa McCallum, University of Western Sydney, Hunter New England Health

Authors: L McCallum, B Liu, P McIntyre and L Jorm

Background: Birth and maternal characteristics have been associated with an increased risk of infection in childhood. However, few studies have evaluated maternal or birth characteristics as risk factors for pertussis.

Methods: We constructed a cohort of mothers and their infants using routinely collected population-based datasets in New South Wales (NSW), Australia. All singleton infants born to NSW resident mothers from 01 Jan 1994 – 31 Dec 2008 were included in the study.

Results: Characteristics associated with increased risk of pertussis during early infancy included having a young (aged 10–19 years) mother, being born in a high prevalence year and mother having hypertension during pregnancy. Infants whose mothers had pertussis during pregnancy or soon after their birth had significantly increased risk of pertussis during early infancy, particularly when aged less than three months (RR73.8; 95%CI 45.4 – 120.1; $P<0.001$).

Conclusion: Socioeconomic factors appeared to be associated with an increased risk of pertussis during early infancy. Protecting mothers from pertussis is likely to reduce the risk of pertussis during early infancy. This work provides additional evidence to support immunising mothers against pertussis during pregnancy, especially during periods of increased pertussis activity.

Challenges in managing a school-based measles outbreak—Melbourne, Victoria, 2014

Presenter: Katherine Gibney, Department of Health and Human Services, Victoria

Authors: A Brahmi, L Franklin and K Gibney

Background: In 2014, Victoria experienced the highest number of confirmed measles cases since 2001, including an outbreak with school-based transmission.

Methods: Confirmed measles cases notified in 2014 were reviewed to identify affected school students and their immunisation status. Surveillance data, correspondence and investigation notes related to a primary school-based measles outbreak were analysed to assess the adequacy and usefulness of school-based immunisation records in the management of this outbreak.

Results: Of the 75 confirmed measles cases notified in Victoria in 2014, 23 cases (31%) were among school-aged students (defined as aged 5–18 years), of whom three had a documented history of measles vaccination, 17 were unvaccinated, and three had unknown vaccination history. Seven measles outbreaks were identified in Victoria in 2014, including a primary school-based outbreak with nine confirmed measles cases. Of the six unvaccinated pupils in the affected school, five contracted measles. The proportion of the school's prep students with documented vaccination records, as required by law, ranged from 39% in 2013 to 97% in 2014.

Conclusion: Inadequately vaccinated students constitute a vulnerable population and schools are a potential site for measles outbreaks. Inadequate enforcement of school-based immunisation records impact the management and control of school-based measles outbreaks.

Verification of measles elimination in Australia

Presenter: Nicolee Martin, Department of Health, Canberra, ACT

Authors: N Martin, D Durrheim, V Stambos, H Gidding, A Dey, T Tran, M Chiew, H Kelly G Dowse, E Denehy and S Lambert

In 2013, World Health Organization (WHO) Western Pacific Region member countries, including Australia, were invited to address five lines of evidence for measles elimination outlined in WHO's *Guidelines on Verification of Measles elimination in the Western Pacific Region*: 1. A detailed description of measles epidemiology following introduction of measles vaccine in the national immunisation program (NIP); 2. Quality of epidemiological and laboratory surveillance; 3. Population immunity; 4. Sustainability of the NIP; 5. Genotyping supportive of measles virus transmission interruption.

In its submission Australia demonstrated: low measles incidence; decreasing outbreak size/duration over time; 84% of cases documented as imported or import-related; childhood measles vaccine coverage >90% for two doses; national estimates of effective reproduction number (R) well below the epidemic threshold; high levels of population seropositivity; and the absence of sustained transmission of any single measles genotype. These findings provided strong evidence for interruption of endemic measles virus transmission.

In March 2014, Australia was formally verified by the WHO Regional Verification Commission as having achieved measles elimination for at least 36 months from 2009. Continuing high-quality surveillance, virus genotyping, tight outbreak control and maintenance of uniformly high levels of routine two dose measles vaccine coverage are required to maintain Australia's measles elimination status.

Genotyping evidence to support measles elimination in Australia

Presenter: Thomas Tran, Viral Identification Laboratory, Victorian Infectious Diseases Reference Laboratory, The Doherty Institute, Melbourne

Authors T Tran, V Stambos, H Kelly and J Druce

Background: It has been previously argued that Australia had interrupted the transmission of endemic measles (that is, had achieved measles elimination) in the years between 1999 and 2005 but it was not until March 2014 that measles elimination was officially declared in Australia. Genotyping evidence was a critical to this declaration. We aim to review the genotyping evidence from 2008 to 2012 as evidence for measles elimination.

Methods: Scientists at the WHO measles regional reference laboratory in Victoria tested measles virus samples from around Australia to differentiate wild-type virus from vaccine-associated virus, to genotype circulating wild-type viruses and to monitor the absence of endemic measles transmission of any one genotype for ≥12 months. Measles viruses were identified by RT-PCR and characterised by nucleic acid sequence analysis of the nucleoprotein gene. Sequencing allowed construction of phylogenetic trees.

Results: From 2008–2012, 354 cases of measles were detected with genotype data available for 309 (87%) cases. Of these, 293 were identified as wild-type strains and 16 were vaccine-associated. A diverse range of genotypes was identified, including B3, D4, D5, D8, D9, G3 and H1. Only genotype D9 was detected over a prolonged period, for 33 weeks in 2010–2011, but phylogenetic studies combined with epidemiological investigations demonstrated recurrent importations of D9 measles viruses rather than persistence of a single lineage. No single genotype has persisted since 2012.

Conclusion: Genotyping data from 2008–2012 were critical in supporting evidence for measles elimination in Australia. Ongoing monitoring, required to maintain elimination status, has confirmed no single genotype has persisted in Australia since 2012.

Short Oral Presentations 2
Chair: Jeremy McAnulty

The effect of prior vaccination on estimates of seasonal influenza vaccine effectiveness

Presenter: Kylie Carville, Epidemiology Unit, Victorian Infectious Diseases Reference Laboratory

Authors: H Kelly, C Lane and K Carville

Background Influenza vaccination is recommended annually, although effects of repeated annual vaccination have been questioned over the last 35 years. We explored the effect of vaccination in the previous year on estimates of influenza vaccine effectiveness (VE) using data from Victorian sentinel general practices, 2011 to 2013.

Methods The test negative design was used to estimate influenza VE, defined as 1– adjusted OR. Patients testing positive for influenza were cases and those testing negative were non-cases (controls). Analysis was adjusted for year, age group, comorbidity and calendar time and restricted to patients presenting within 7 days of symptom onset during the influenza season. We calculated VE for patients vaccinated in consecutive seasons, in the current season only, previous season only, and in neither season (reference).

Results 1369 patients were included, 487 (36%) were influenza positive. In 2012, VE was highest amongst those vaccinated in the current season only (VE=83% (95%CI 24, 96)), followed by those vaccinated in both the current and previous seasons (VE=41% (–4, 67)) and lowest among those vaccinated in the previous season only (VE=19% (–43, 55)). No consistent trend was seen in the other years.

Conclusion Results from this study, although limited by small numbers, are consistent with prior hypotheses and with other recent studies.

Transmission of the first influenza A(H1N1)pdm09 pandemic wave in Australia was driven by undetected infections: pandemic response implications

Presenter: James Fielding, Victorian Infectious Diseases Reference Laboratory

Authors: J Fielding, H Kelly and K Glass

Background: During the first wave of influenza A(H1N1)pdm09 in Victoria, Australia the rapid increase in notified cases and the high proportion with relatively mild symptoms suggested that community transmission was established before cases were identified. This lead to the hypothesis that those with low-level infections were the main drivers of the pandemic.

Methods: A deterministic susceptible-infected-recovered model was constructed to describe the first pandemic wave in a population structured by disease severity levels of asymptomatic, low-level symptoms, moderate symptoms and severe symptoms requiring hospitalisation. The model incorporated mixing, infectivity and duration of infectiousness parameters to calculate subgroup-specific reproduction numbers for each severity level.

Results: With stratum-specific effective reproduction numbers of 1.82 and 1.32 respectively, those with low-level symptoms, and those with asymptomatic infections were responsible for most of the transmission. The effective reproduction numbers for infections resulting in moderate symptoms and hospitalisation were less than one. Sensitivity analyses confirmed the importance of parameters relating to asymptomatic individuals and those with low-level symptoms.

Conclusion: Transmission of influenza A(H1N1)pdm09 was largely driven by those invisible to the health system. This has implications for control measures – such as distribution of antivirals to cases and contacts and quarantine/isolation – that rely on detection of infected cases. Pandemic plans need to incorporate milder scenarios, with a graded approach to implementation of control measures.

Influenza pandemic planning – How prepared are you?

Presenter: Elizabeth Birbilis, Department of Health and Human Services, Victoria

Authors: E Birbilis and R Lester

Influenza pandemic planning is a key activity for most health departments and ministries throughout the globe. Effective preparation and planning are essential elements to mitigate the health, social and economic impacts of influenza pandemics.

In November 2014 the Victorian Department of Health and Human Services published the *Victorian health management plan for pandemic influenza*. Based on the *Australian health management plan for pandemic influenza,* and using lessons learnt from the 2009 pandemic, the Victorian plan takes a unique approach by providing operational guidance to health and primary care services using a settings-based approach. Specific planning considerations are included for educational and child care facilities, laboratories, local government, emergency services and for residential facilities, such as aged care, disability and custodial.

The guidance is delivered through a series of operational appendices which are written within the context of the existing policy environment. A staged approach is used, based on the Australian pandemic stages and is consistent with Victoria's strategic approach to emergency management.

This up to date plan provides a framework to minimise the overall impact of an influenza pandemic on the Victorian community.

Visualising severity of influenza seasons in Australia using the online Flutracking.net survey

Presenter: Sandra Carlson, Hunter New England Health

Authors: C Dalton, M Butler, S Carlson, L McCallum, D Durrheim

Background: Flutracking is an online weekly survey of influenza-like illness (ILI), health seeking behaviour and laboratory investigation, and illness severity that has operated nationally in Australia since 2008. In 2014, approximately 18,000 participants responded to the survey each week.

Methods: We compared two indices of ILI severity to develop a visual display of the relative severity of annual influenza seasons: the average percentage of respondents with cough and fever by week (on x axis), and the average number of days off normal duties (on the y axis) from 2008 to 2014 for NSW and Australia.

Results: The influenza seasons of years 2009, 2012 and 2014 cluster in the upper right quadrant indicating greater severity whereas the 2010 and 2011 seasons cluster in the lower left quadrant consistent with lesser severity. The visualisation produces an intuitive insight into the relative severity of influenza seasons that anecdotally accords with clinical and public health practitioner perceptions. The visualisation overcomes some of the biases and misperceptions regarding the severity of a particular influenza season associated with variations in influenza laboratory testing frequency.

Conclusion: The Flutracking community data provides a useful and intuitive visualisation to communicate influenza season severity. (Graphic available to view at: http://goo.gl/m3xC8H)

Disproportionate burden of influenza virus infections among Aboriginal infants

Presenter: Faye Janice Lim, Wesfarmers Centre of Vaccines and Infectious Diseases, Telethon Kids Institute, The University of Western Australia, Western Australia

Authors: FJ Lim, CC Blyth, P Fathima, N de Klerk and HC Moore

Background: Influenza viruses are an important cause of respiratory infections in children and pose a significant burden on families and the community. Using linked administrative data, we describe the burden of influenza virus among children.

Methods: We selected records with a notification or diagnosis of influenza in 2000–2012 for all children born in Western Australia, 1996–2012. We linked these with state-wide routine laboratory records of specimens collected within 48 hours of notification or hospitalisation. Using person-time-at-risk as the denominator, we calculated the incidence of influenza virus-positive detections by age and Aboriginal status.

Results: In total, there were 5168 notifications (1.5 per 1000 child-years) and 2303 hospitalisations (0.6 per 1,000 child-years) for influenza. 81.7% of notifications and 60.7% of hospitalisations linked to an influenza virus test record. Infants less than 12 months had the highest influenza virus-positive notification and hospitalisation rates. Incidence rates among Aboriginal infants less than 6 months were 4 times higher than non-Aboriginal infants for influenza virus-positive notifications (IRR=4.6, 95% CI=3.3,6.2) and hospitalisations (IRR=4.3, 95% CI=2.9,6.1).

Conclusion: Aboriginal young children bear a disproportionate burden of influenza virus infections. These data will enable us to track the impact of including influenza vaccine for Aboriginal children in the National Immunisation Program.

Interseasonal influenza in the Australian Capital Territory, 2012–2013

Presenter: April Roberts-Witteveen, ACT Health

Authors: A Roberts-Witteveen and L Ford

Background: An increase in influenza notifications was investigated in the summer of 2012/2013 in the Australian Capital Territory.

Methods: Notified cases of influenza with specimen collection between 24/12/2012 and 30/6/2013 were interviewed using a standardised questionnaire. Data about diagnosis method, symptom profile and duration, delay between onset and diagnosis, other laboratory testing and vaccination history were collected. Adjusted odds ratios, *P*-values and confidence intervals were calculated in StataIC v12.1.

Results: Of 100 notified cases in the study period, 98 were interviewed. Compared to serology (n=67), cases diagnosed by PCR (n=31) were statistically significantly more likely to have a fever, cough and fever, or an emergency department presentation and less likely to have been vaccinated in the previous 12 months. Fifty-eight (87%) cases diagnosed by serology met one or more of the following categories: a delay between onset and diagnosis of 10 or more days, influenza vaccination in the previous 12 months, or a negative PCR test in conjunction with the positive serology test. These cases were assessed as unlikely to be true recent infection.

Conclusion: Characteristics of influenza cases differed by diagnosis method. The value of serology in the interseasonal period could be enhanced by case follow-up.

Declining seasonal influenza vaccine effectiveness for pandemic A(H1N1)pdm09, 2010–2014, Victoria, Australia

Presenter: Kylie Carville, Epidemiology Unit, Victorian Infectious Diseases Reference Laboratory

Authors: K Carville, J Fielding, C Lane and H Kelly

Background: Since its emergence in 2009, influenza A(H1N1)pdm09 has become the predominant H1N1 influenza virus circulating globally. In subsequent years the World Health Organization has recommended that the annual influenza vaccine contain viruses that are antigenically and genetically similar to influenza A(H1N1)pdm09.

Methods: Seasonal influenza vaccine effectiveness (VE) is calculated annually in Victoria, using data from the GP Sentinel Surveillance System in the test-negative design. Patients testing positive for influenza are classified as cases and those testing negative are classified as non-cases (controls).

Results: VE against pH1N1 in 2010 was 78% (95%CI 33, 93), in 2011 74% (95%CI –69, 100), in 2012 59% (–95, 91), in 2013 64% (95%CI–89, 93), and in 2014 23% (95% –44, 59).

Discussion: Our data are limited by small numbers, but the decline is unexpected as circulating and vaccine virus strains have been the same. In Canada the pandemic A(H1N1)pdm09 VE estimate for 2013–2014 was 74% (95%CI 58–83). However, a European hospital network has also reported a low pandemic A(H1N1) pdm09 VE of 24% for 2013–2014. We hypothesise that VE may be moderated by past infection or previous immunisation.

Predictors of sustained participation in an online influenza-like illness surveillance system

Presenter: Sandra Carlson, Hunter new England Population Health, New South Wales

Authors: S Carlson, C Dalton, L McCallum, M Butler and D Durrheim

Background: This analysis seeks to identify the factors that have led to Flutracking becoming the largest, most rapid growing, and highest participation rate online influenza-like illness surveillance system in the world.

Method: The proportion of surveys completed each year was calculated from 2011 to 2014. A logistic regression was used to assess factors associated with participation in all four years of surveys versus one to three years.

Results: There were 24 to 25 surveys per year available for completion. Of participants who completed a survey during the first four survey weeks of each year, 71%, 69%, 67% and 79% completed greater than 90% of available surveys in 2011, 2012, 2013 and 2014 respectively. Among 12,084 participants who completed a survey for themselves and other participants in 2014, age greater than 40 years (OR: 2.58, $P<0.01$) and working face to face with patients (OR: 1.39, $P<0.01$) increased the odds of participating in all four

years of the survey. Being female decreased the odds of participating in all four years (OR: 0.82, $P<0.01$). Vaccination against influenza and having children in the household were not associated with participating in all four years of the survey.

Conclusion: Age greater than 40 years and male gender were associated with sustained participation in this online weekly health survey.

Evaluation of the South Australian Surveillance System for Influenza based on notifications

Presenter: Salenna Elliott, Communicable Disease Control Branch, Department for Health and Ageing, South Australia

Authors: SR Elliott, C Graham, M Miller and J Raupach

Seasonal influenza became notifiable in South Australia (SA) in 2008. Major objectives are to monitor seasonal epidemics, identify outbreaks and support pandemic preparedness. This evaluation aimed to describe current operation, determine whether the system is meeting objectives and assess performance.

Analysis of notification data (2010–2013) investigated data completeness, representativeness and timeliness. Interviews with stakeholders from the public laboratory, Communicable Disease Control Branch (CDCB) and a small convenience sample of clinicians provided feedback about operation, usefulness and performance attributes including simplicity, acceptability, flexibility and stability.

The system generates timely and complete data that are broadly representative. It is simple, flexible and stable, but has several manually-dependent steps which may limit acceptability, timeliness and flexibility. Whilst laboratory and CDCB stakeholders found the system to be acceptable, clinicians questioned the need for dual notification and were uncertain how surveillance data were used. This could contribute to observed delays in submission of medical notifications, potentially delaying detection of outbreaks.

The surveillance system is achieving major objectives, but has some limitations. Recommendations include: 1) development of an electronic medical notification form, 2) establishment of regular direct reporting of influenza surveillance data to health service providers, and 3) education of institutions to directly report influenza outbreaks.

Short Oral Presentations 3
Chair: Martyn Kirk

Escherichia coli O157:H- outbreak associated with an agricultural show in Brisbane

Presenter: Bhakti Vasant, Public Health trainee, Queensland Health

Authors: B Vasant, R Stafford, S Vlack, P Titmus, AV Jennison, H Smith, J Barrett, CA Quagliotto, M Young, K Jarvinen, S Bennett and SB Lambert

Background: An *Escherichia coli* O157 H- outbreak associated with an annual agricultural show in Brisbane, 2013, was the largest outbreak of Shiga toxin-producing *Escherichia coli* (STEC) infection reported in Australia.

Methods: Our response included case questionnaires, a case-control study, and investigation and sampling of humans, animals, and the environment. A communication strategy was implemented, outbreak reports compiled, and formal debriefing conducted.

Results: Of 57 cases identified, 37 (65%) were children (median age 9 years; range 1–77 years). There were no cases of haemolytic uraemic syndrome, and no deaths. Epidemiological and microbiological investigations supported the hypothesis that STEC was transmitted from animals to humans. Daily outbreak management teleconferences co-ordinated investigation and management. Clinical, community, and media communications enhanced case finding and management. Prolonged duration of STEC carriage in some cases (median: 18 days, range: 2–52 days) presented significant challenges for public health follow-up. We were unable to identify changes in infection prevention at the show compared to previous years.

Conclusion: Revised *Infection Control Guidelines for Animal Contact* highlight the importance of hand-washing, risks with feeding animals, and cleaning animal enclosures. With further infection control measures implemented, surveillance did not detect any STEC cases associated with the 2014 show.

Outbreak of sporotrichosis in the Southwest of Western Australia

Presenter: Naru Pal, WA Country Health Service, Bunbury, Western Australia

Authors: N Pal, G Dowse, A Whittle, C Golledge and I Arthur

Background: An outbreak of cutaneous sporotrichosis, caused by the fungus *Sporothrix schenkii*, occurred in the Margaret River region of Western Australia between October 2012 to December 2013, with 24 cases identified.

Methods: We conducted a retrospective case review and used a questionnaire to determine case characteristics and risk factors.

Results: The median age of cases was 52 years (range, 6–86 years), the median duration of active lesions was 3.9 months (range, 2–12 months), and the median treatment duration was 4 months (range, 2–8 months). All cases eventually received treatment with oral itraconazole, although more than half (54%) received inappropriate antibiotics prior to laboratory confirmation. Ninety-two per cent of cases reported direct contact with straw mulch and hay products prior to development of lesions, with 80% linked to a particular supplier. 60% of cases used gloves and other protective measures when handling straw, but lesions occurred in areas not sufficiently protected. Case numbers declined after various public health measures were implemented.

Conclusion: This was the third and largest outbreak of sporotrichosis recorded in the Margaret River region, following clusters in 2000/01 and 2003/05. Outbreaks appear to be associated with moist conditions that lead to contamination of hay and straw products.

An emerging strain of *Salmonella* Typhimurium (STm) in south east Queensland

Presenter: Vicki Slinko, Metro South Public Health Unit, Brisbane, Queensland

Authors: VG Slinko, R Bell, J Bates, R Stafford, A Neill, G Pollard, L Hiley, G Micalizzi, T Graham, P Seal, J Barten, A Khan and KAJ Jarvinen

Salmonella notifications have increased significantly in South East Queensland recently. We investigated four outbreaks (OB) of *Salmonella* Typhimurium (STm) infection since mid-December 2014, with over 250 cases (115 laboratory confirmed). Two closely related MLVA strains were detected (STm-OB): 03-12-11-12-524 and 03-12-12-12-524. Most isolates were phage type U307 whereas other recent outbreak strains have been phagetype 135 or 135a.

Epidemiology implicated deep fried ice cream as the likely vehicle of transmission in Outbreak 1 (case series), 2 (RR 3.3; 95%CI: 2.3, 4.8) and 3 (RR undefined, $P<0.001$). STm-OB was detected in uncooked ice cream balls and restaurant environmental samples, with opportunities for cross contamination at each kitchen. All eggs came from the same producer. STm-OB was detected in drag swabs and used chicken feed from the farm. Outbreak 4 involved consumers of Kimbap sushi from various outlets with STm-OB detected in sampled whole sushi. No further investigations were possible as the supplier was not contactable.

MLVA genotyping has proved useful in identifying and linking outbreaks while epidemiology promptly implicated the vehicle of transmission. EH traceback and investigations revealed the likely source. Eggs need to be regarded as a contaminated product with appropriate food safety practised in food establishments.

Increasing incidence of *Salmonella* notifications in Australian States and Territories from 2000–2013

Presenter: Laura Ford, Australian National University, ACT

Authors: L Ford, K Glass, M Veitch, R Wardell, T Dobbins, B Polkinghorne and M Kirk

In Australia, salmonellosis is a major cause of bacterial gastroenteritis. National surveillance figures show that the number of *Salmonella* notifications has been increasing over time. We used negative binomial regression to estimate incidence rate ratios for sex, age, jurisdiction, and trend by jurisdiction for *Salmonella* Typhimurium and non-Typhimurium *Salmonella* serotypes separately. From 2000 to 2013, almost all states and territories had significantly increasing trends of infection for both *S.* Typhimurium and non-Typhimurium *Salmonella*, with significant state and territory incident rate ratios of annual increases ranging from 1.03 (95% CI 1.02–1.05) to 1.12 (95% CI 1.10–1.14) for *S.* Typhimurium and from 1.02 (95% CI 1.01–1.03) to 1.07 (95% CI 1.06–1.08) for non-Typhimurium *Salmonella*. *S.* Typhimurium rates were higher than non-Typhimurium rates in most age groups in the south eastern continental states, while non-Typhimurium *Salmonella* rates were higher in most age groups elsewhere. For all jurisdictions, the *S.* Typhimurium incidence rate peaked at 12–23 months of age and the non-Typhimurium *Salmonella* incidence rate peaked at 0–11 months of age, which may indicate different sources of transmission. Salmonellosis notifications are increasing in Australian states and territories. The trends and subtypes seen vary across the country and differ from trends reported internationally.

A case of foodborne botulism in Wellington, New Zealand: The public health experience

Presenter: Eamonn Deverall, Regional Public Health Wellington

Authors: E Deverall, S Jefferies, N Esson, M Gibson, A Nesdale, M Balm and I Rosemergy

Regional Public Health, responsible for the Greater Wellington region of New Zealand, received a notification of a suspected case of botulism on 19 December 2014. The patient's rapid deterioration, clinical presentation and EMG studies were compatible with the classic symptoms of botulism. This is only the 3rd case of botulism to be reported in New Zealand, and the first for 30 years.

This was an interesting and challenging case to investigate as the index case was unable to provide any information and had recently arrived from overseas. The likely source was identified as a wet risotto, produced in New Zealand. It had been kept beyond its best before date and stored inappropriately at room temperature for several months. Investigating the case highlighted the important potential risks posed by minimally heated, chilled foods and identified several areas for product safety improvement.

Informing the public health management of typhoid and paratyphoid in Australia

Presenter: Megan Young, Metro North Public Health Unit, MNHHS and Griffith University, Queensland

Authors: MK Young, V Slinko, J Smith, H Carroll, S Bennett, S Appleton and BJ McCall

Background: Queensland guidelines for the public health management of notified enteric fever cases changed in 2010. We aimed to determine the impact of this change on the likelihood of cases and contacts adhering to recommendations for faecal clearance/screening, and assess the duration of infectiousness of cases and extent of local transmission to contacts.

Methods: Data from notification records of typhoid and paratyphoid infection in southeast Queensland in 2008–2012 (inclusive) were extracted and analysed.

Results: Sixty-nine of 85 cases and 218 of 265 contacts submitted at least one faecal specimen. Cases were 2.7 (95%CI 1.2–6.0) and contacts were 4.4 (95%CI 3.0–6.4) times more likely to complete recommended faecal clearance/screening under previous compared to recent guidelines (requiring more specimens). In ten cases with positive post-treatment specimens, last recorded infectiousness was 19 days to six months after notification. The documented rate of local transmission of infection was 18/1000 contacts submitting at least one faecal specimen (95% CI 6–48/1,000).

Conclusion: Local transmission risk of enteric fever is low, although small numbers of cases may have prolonged bacilli excretion post-treatment. More complex clearance/screening regimens are associated with decreased compliance. Pursuing extensive faecal clearance/screening regimens is unlikely to be an effective means of preventing transmission in this setting.

Screening and monitoring travellers to detect and prevent the Ebola virus disease (EVD) in NSW

Presenter: Jocelyn Chan, Masters of Philosophy (Applied Epidemiology) scholar at Communicable Diseases Branch, Health Protection, NSW Health

Authors: J Chan, M Patel, S Tobin and V Sheppeard

Introduction: While entry screening and monitoring of arrivals from EVD affected countries has been adopted by US and Canada, some have asserted these measures are resource intensive and provide little additional risk mitigation. We report NSW's experience with screening and monitoring.

Methods: Data on arrivals from EVD affected countries were provided daily by immigration authorities between 1 October 2014 and 14 January 2015. Public health units performed exposure risk assessments and followed-up daily.

Results: Of the 67 arrivals, 66 were assessed and monitored. Returning aid workers were the largest category (n=23). 57 had no known exposures to EVD, 11 had low risk exposures and none had high risk exposures. 46 were followed up by phone, 13 by texting, and 7 by email; one developed fever and two others had transient vomiting and headache. None required assessment at a designated hospital or developed EVD.

Conclusion: As expected, the risk of disease in this cohort was low. Public health units supported arrivals in recognising and managing symptoms. While economic costs were not estimated, screening and monitoring five arrivals per week was feasible using existing public health infrastructure; this was considered essential given serious implications of missing even one case of EVD.

Western Australian policy for the management of pregnant women and neonates with Ebola virus disease

Presenter: Anna Beswick, WA Health

Authors: A Beswick, Terri-Lee Barrett, P Armstrong and A Robertson

Background: During preparedness efforts for Ebola virus disease (EVD) in Western Australia, the potential for cases in pregnant women or neonates, albeit small, was acknowledged and a lack of management policy identified.

Method: A working group comprising clinicians and public health officials was formed to review the ethical issues, examine the literature and current situation and develop a policy for clinicians

Result: There have been no reports of EVD in pregnant women or neonates in high income countries to date and limited reports of those managed in Africa. Key aspects of the policy include deployment of specialist resources to manage cases at quarantine hospitals and early recognition and treatment of haemorrhage and spontaneous abortion. Where invasive procedures for management of fetal distress may pose too high a risk of transmission to staff, fetal monitoring is not advised.

Conclusion: This policy filled a gap in the state policy regarding management of EVD and highlights the ethical difficulties in developing policy when there is little applicable evidence base. Nationally-agreed guidelines would be of benefit.

Investigation of a dramatic increase in community-associated MRSA in the Kimberley region of Western Australia

Presenter: Anna Beswick, Disaster Management, Regulation and Planning, WA Health

Authors: A Beswick, P Armstrong, G Coombs and S Tempone

Background: Infections caused by community-associated strains of methicillin resistant staphylococcus aureus (CA-MRSA) are an important cause of morbidity in the community and notifications in Western Australia have increased markedly in recent years, particularly in the Kimberley region of Western Australia.

Methods: Notification and laboratory data for the period 2005–2013 were analysed. Hospital inpatient data for MRSA-associated admissions, and distribution data for antibiotics used for MRSA infections, were examined. Semi-structured interviews were conducted with Kimberley-based clinicians experienced in the treatment of these infections.

Results: From 2005–2013, there was a 10-fold rise in the number of CA-MRSA notifications from the Kimberley. Four main CA-MRSA clones were responsible, three of which were positive for the Panton-Valentine leucocidin (PVL) gene. The number of hospital admissions and procedures for MRSA-associated conditions increased over this time period, as did the pattern of distribution of antibiotics active against MRSA, suggesting the increase is real. However, the propensity for testing for skin sepsis also seems to have occurred over the period, evidenced by increasing samples positive for Group A streptococci and reports from clinicians.

Conclusion: The rapid rise of CA-MRSA notifications in the Kimberley is probably due to a combination of increased incidence and increased testing for skin sepsis.

Short Oral Presentations 4
Chair: John Bates

Evaluation of hepatitis A surveillance in Australia: Enhancements for multi-jurisdictional outbreak detection

Presenter: Courtney R Lane, University of Melbourne, Victoria.

Authors: CR Lane, N Stephens and M Kirk

Background: Surveillance of hepatitis A in Australia may be insensitive to outbreaks due to insufficient viral strain typing. We evaluated hepatitis A surveillance to identify the perceived benefits of, and barriers to, enhancing current practices through the national collation of laboratory characterisation and case exposure data.

Methods: We followed established guidelines to evaluate attributes of current hepatitis A surveillance using literature review and semi-structured interviews with stakeholders. We used 2011–2013 hepatitis A notifications from two Australian jurisdictions and created a pilot database to assess data completeness and estimate resource requirements for enhanced surveillance.

Results: We found storage of laboratory and exposure data is fragmented and informally reported between stakeholders, which may decrease timeliness in outbreak investigations and hinder the detection of small temporally or geographically dispersed clusters. Four Australian jurisdictions do not routinely sequence locally acquired specimens and sequences are not routinely compared between laboratories. Sequencing of all locally acquired isolates and national collation of exposure data would require limited additional resources, approximated at $10,600 and 416 minutes annually. However, barriers to the comparison of laboratory and exposure data exist.

Conclusion: Enhanced national surveillance may improve cluster detection and decrease investigation time through national analysis of exposure information and comparison of sequence data.

Salmonella point source outbreaks in South Australia, investigation trends and findings, 2008 –2014

Presenter: Emma Denehy, Communicable Disease Control Branch, SA Health, South Australia

Authors: E Denehy, M Miller and A Koehler

A retrospective analysis of *Salmonella* point source outbreak investigations conducted at the Communicable Disease Control Branch of the South Australian Department for Health and Ageing was undertaken. The objectives were to characterise *Salmonella* point source outbreaks, describe investigation techniques and findings.

A descriptive analysis of *Salmonella* point source outbreaks recorded in the South Australian Department of Health Notifiable Infectious Disease Surveillance database and corresponding outbreak reports were reviewed from 2008 through 2014.

Among the forty outbreaks investigations, primary pathogens were *Salmonella* Typhimurium phage type 9 (55%), *Salmonella* Typhimurium phage type 135 (15%), and *Salmonella* Typhimurium phage type 44 (13%). The majority of outbreaks (70%) were investigated using descriptive case series analysis, with the remaining using analytical study tools. An implicated source was identified in 26 outbreaks, of these 22 (85%) were related to eggs, predominantly raw egg products, two to chicken and two to pork. The incidence of point source outbreaks has increased with time with the three year average outbreak investigations increasing from three (2008–2010) to nine (2012–2014).

The increasing number of investigations highlights the need for ongoing improvements in food safety, particularly for handling raw egg products. Efforts also need to be focused to intensify intervention strategies at the industry level.

Investigation of a possible outbreak of Barmah Forest virus infection

Presenter: Peter Markey, Disease Surveillance, Centre for Disease Control, Northern Territory Department of Health, Darwin, Northern Territory, Australia

Authors: N Kurucz, P Markey, A Draper, L Melville, R Weir, S Davis, A Warchot R Boyd and D Stokeld

Between October 2012 and October 2013, unprecedented high numbers of Barmah Forest virus (BFV) disease cases were reported in the Northern Territory (NT). An investigation was launched by the NT Department of Health in cooperation with the Department of Primary Industry and Fisheries and the Department of Land Resource Management. The investigation included mosquito virus isolations from mosquitoes collected in Darwin urban areas, BFV antibody testing in small peri-urban mammals and investigation of human cases reported in Darwin, nearby Palmerston and Alice Springs. No BFV was isolated from the 4641 mosquitoes tested, none of the mammals tested positive for BFV antibodies and the high BFV disease case numbers did not correlate with the relatively low mosquito vector numbers trapped in 2012/13. It was estimated that 26% of the 79 human cases investigated in the NT did not have an acute arboviral illness and therefore were false positives. Other jurisdictions in Australia also reported high numbers of BFV disease cases, and investigations elsewhere led to the withdrawal of the Alere PanBio BFV IgM ELISA test kit used in most laboratories. Current testing methods and case definitions need to be revised to reflect the true numbers of BFV disease cases occurring in Australia.

OzFoodNet: 15 years of enhanced foodborne disease surveillance in Australia

Presenter: Ben Polkinghorne, Australian Government Department of Health

Authors: B Polkinghorne, M Miller, A Draper, R Leader, K Knope and G Fitzsimmons

OzFoodNet is a national network of foodborne disease epidemiologists with a mission to investigate and describe the epidemiology of foodborne disease, and to devise methods to minimise foodborne illness in Australia. From 2001–2013, OzFoodNet investigated over 1,600 foodborne and suspected foodborne outbreaks affecting nearly 26,000 people with over 1,900 associated hospitalisations and 68 reported deaths. Overall, food prepared in restaurants was involved in 41% of outbreaks, Salmonella Typhimurium was the suspected or confirmed aetiological agent for 29% and dishes containing raw or under-cooked eggs were the suspected or confirmed food vehicle in 18%.

OzFoodNet has investigated more than 40 multi-jurisdictional outbreaks. Pathogens included hepatitis A, norovirus, typhoid, *Salmonella,* Shiga toxin-producing *Escherichia coli* and *Listeria monocytogenes.* Food vehicles identified include varied produce such as rockmelons, papaya, poultry, alfalfa sprouts, cheese, semi-dried tomatoes, almonds, oysters and eggs.

OzFoodNet has enhanced national foodborne investigation and research capacity and positively influenced national food safety policy. OzFoodNet needs to maintain vigilance as national and global food transportation make multi-jurisdictional outbreaks an ever-present risk and novel pathogens like hepatitis E emerge in Australia. OzFoodNet must advocate strongly to ensure the adoption of rapid microbial diagnostic techniques doesn't reduce our capacity to detect outbreaks or monitor trends.

Sources of salmonellosis in South Australia

Presenter: Kathryn Glass, Australian National University, ACT

Authors: E Fearnley, H Hocking, J Raupach, M Veitch, L Ford and MD Kirk

Salmonellosis is a significant cause of foodborne gastroenteritis in Australia, and rates of notified illness have increased over recent years. We adopt a Bayesian source attribution model to estimate the contribution of different foods at the animal reservoir level to illness due to *Salmonella* spp. in South Australia between 2000 and 2010, together with 95% Credible Intervals (CrI). We excluded known travel associated cases and those of rare subtypes (fewer than 20 human cases or fewer than 10 isolates from included sources over the 11 years). The remaining 76% (5591/6559) were classified as sporadic or outbreak associated. We attributed 35% (95% CrI: 20–49) of sporadic cases to chicken meat and 37% (95% CrI: 23–53) of sporadic cases to eggs. Of outbreak-related cases, 33% (95% CrI: 20–62) were attributed to chicken meat and 59% (95% CrI: 29–75) to eggs. Analysis of source-related parameters showed higher risk of illness from contaminated eggs than contaminated chicken meat, despite low *Salmonella* prevalence on eggs, suggesting that consumption and handling practices potentially play a bigger role in illness due to eggs than chicken meat. Our results strengthen the evidence that eggs and chicken meat are important sources and vehicles for salmonellosis in South Australia.

When is an increase in cases of acute rheumatic fever an outbreak?

Presenter: Kate Hardie, Centre for Disease Control, Northern Territory

Authors: J Francis, C Gargan, E Schimann, D Holt, B Remenyi, M Fittock and V Krause

Acute rheumatic fever (ARF) is endemic in Aboriginal communities in the Northern Territory (NT). In September 2014 doctors at a remote Aboriginal community-clinic in the NT reported a higher than expected number of cases of ARF and apparent clustering within households.

There were 13 cases of definite ARF during a 4-month period compared with an expected 2.2 for that period (6.6 cases per year). We report this series of ARF cases and describe our search for guidance on whether a public health response was required and if so, what form that should take.

Ultimately the public health response was informed by consultation with specialists in fields including pathology, cardiology, public health, paediatrics and infectious diseases and involved education targeting household contacts, throat and skin sore swabs from contacts to identify if a single dominant strain of Group A *Streptococcus* (GAS) was circulating, and administration of benzathine penicillin-G to contacts.

An increased incidence of ARF to unprecedented levels prompted a public health response designed to limit disease spread and improve understanding of GAS transmission and its impact on ARF within the community. We discuss whether there should be nationally consistent guidance on how to investigate and manage a possible ARF outbreak.

Salmonella Typhimurium phage type 44: A Victorian outbreak and review of MLVA patterns

Presenter: Zoe Cutcher, Australian National University, ACT, and Department of Health, Victoria

Authors: Z Cutcher, J Gregory, M Valcanis, K Mercoulia, M Kirk, N Stephens, and M Easton

Background: In December 2014, a *Salmonella* Typhimurium phage type 44 (STm44) outbreak occurred following a function in Victoria. We investigated the outbreak to determine a cause and compared multi locus variable-number tandem repeat analysis (MLVA) patterns to previous cases.

Methods: We conducted a cohort study using a menu based questionnaire and calculated relative risks for all food items. We compared MLVA patterns for the outbreak strain against other Victorian STm44 cases and reviewed outbreak investigations from 2009–2014 to examine potential sources.

Results: There were 10 cases among 29 guests interviewed. Risk of illness increased with consumption of the appetiser and frittata. Cross contamination from eggs was suspected. The outbreak strain was indistinguishable from 1.7% (7/ 392) of MLVA patterns since 2009. A predominant historical pattern accounted for 45% of all patterns; another 51% were closely related including the outbreak strain. There were 5 historical STm44 outbreaks (78 cases) and 1 cluster (102 cases); all were related to the predominant MLVA pattern. Previous investigations all implicated or suspected eggs as the source.

Conclusion: We were unable to identify a specific source for this outbreak, but cross contamination from eggs appears likely. MLVA provided limited differentiation between STm44 isolates.

Why HITnet kiosks didn't hit the mark for sexual health education of Western Australian Aboriginal youth

Presenter: Donna Mak, Communicable Disease Control Directorate, WA

Authors: D Vujcich, N Hadland, S Clews, B Sullivan and D Mak

Objective: To assess the use and appropriateness of sexual health modules installed on Heuristic Interactive Technology (HITnet) kiosks at Aboriginal Community Controlled Health Services (ACCHS), and aimed at Aboriginal teenagers visiting these sites.

Methods: Modules were assessed for cultural appropriateness using Yunkaporta's Aboriginal pedagogy framework. Data measuring kiosk use were obtained through kiosk activity reports. An online survey of ACCHS staff was used to qualitatively assess use and staff perceptions of HITnet kiosks.

Results: Modules were consistent with seven of the eight elements of Yunkaporta's framework. Generally, usage by teenagers (13–19 years) was low and the majority of users (56%) were either under 12 years or over 19 years of age. Key issues reported by ACCHS staff (n=11) included: lack of clarity regarding staff responsibility for overseeing kiosk functionality; kiosks attracting "inappropriate ages"; and "lack of privacy" based on kiosk location, screen visibility, and absence of headphones preventing discreet access.

Conclusions: While the modules were tailored to a young Aboriginal audience through technology thought to be appealing to this group, there were a number of practical barriers to their use. Information that is accessible via personal devices may be a better vehicle than public kiosks for conveying sensitive subject matter.

Trachoma prevalence trend in Australia

Presenter: Carleigh Cowling, University of New South Wales, New South Wales

Authors: C Cowling, M Kong, B Liu, T Snelling, D Wilson and J Kaldor

Background: Australia is the only high-income country where trachoma is endemic, occurring primarily in remote Aboriginal communities in the Northern Territory(NT), South Australia(SA) and Western

Australia(WA). The Australian Government funds trachoma surveillance, reporting and control programs which are largely based on the WHO SAFE strategy and is a signatory to the Global Elimination of Trachoma by 2020 initiative.

Methods: Data are collected annually in accordance with CDNA *Guidelines for the public health management of trachoma in Australia* from communities identified by jurisdictions as being at-risk or potentially at-risk of trachoma. We restricted comparisons over time to the 5–9 year age group, which is the target a group for the trachoma screening programs in all regions.

Results: Overall trachoma prevalence has declined from 15% in 2009 to 4% in 2012, and plateaued at 4% in 2013 at the National level across all communities screened, however this trend varies at a jurisdictional and regional level. Treatment coverage has increased from 65% in 2011 to 81% in 2013, and the doses of azithromycin distributed have also increased from 1738 in 2007 to 10,219 in 2013.

Conclusion: Trachoma prevalence in Australia has declined, however concerted effort is still required to eliminate trachoma at the regional level.

Plenary 3: Disease elimination and eradication in Australia and internationally
Chair: Associate Professor Martyn Kirk

In this panel session, expert presenters will discuss the eradication and elimination of diseases of public health importance including trachoma, measles and polio. In particular, we are approaching eradication for some of these diseases. Presenters will discuss some of the success stories, challenges and the future for these efforts in Australia and globally. The panel session will highlight the importance of collection of high-quality surveillance data in supporting and verifying disease control efforts.

Progress towards trachoma elimination in Australia and internationally

Professor John Kaldor, Professor of Epidemiology and NHMRC Senior Principal Research Fellow, Public Health Interventions Research Group, Kirby Institute, University of New South Wales, Sydney

Measles elimination in Australia: the path behind and road ahead

Associate Professor Stephen Lambert, Medical Epidemiologist, Queensland Centre for Children's Health Research, Brisbane

Polio eradication: is the elusive goal just around the corner?

Professor David Durrheim, Professor of Public Health Medicine, University of Newcastle and Director Health Protection, Hunter New England Area Health Service, NSW

Poster abstracts

Persistent STEC infection: Investigating delayed clearance of O128 in Queensland patients

Presenter: Rikki Graham, Molecular Epidemiology, Public Health Microbiology, Forensic and Scientific Services, Department of Health, Queensland, Australia

Authors: RM Graham, NX Fang, CJ Doyle and AV Jennison

Shiga-toxigenic *Escherichia coli* (STEC) is an important cause of human illness, and while focus has traditionally been on serogroup O157 STEC, non-O157 STEC are increasingly being recognised as being of public health significance. Our laboratory has found that serogroup O128 STEC has been associated with diarrhoeal disease in a number of cases but upon the resolution of symptoms, prolonged asymptomatic carriage occurs for extended periods of time.

According to Queensland Communicable Disease Control Guidelines, STEC positive patients are required to provide two negative clearance stool samples at least 24 hours apart. Cases from high risk

groups including children under 5 years, carers of children, healthcare workers and food handlers are typically excluded from high risk roles/attendance until clearance is recorded. For these reasons, failure to achieve clearance can cause social and emotional stress.

This study reports on the use of whole genome sequencing to investigate persistent O128 STEC isolates from Queensland patients and gain insight into genetic characteristics that may contribute to their ability to not only cause disease but to then persist asymptomatically in the host. Furthermore, the prolonged length of carriage (>14 months) of one case has provided a unique opportunity to study within-host genomic changes over time.

RAPID (Response and Analysis for Pacific Infectious Diseases): translating research into action

Presenter: Tony Merritt, Hunter Medical Research Institute, University of Newcastle, New South Wales

Authors: B Paterson, D Durrheim, T Merritt, K Eastwood and J Flint

Ideally, surveillance evaluation research should not only describe surveillance systems but provide evidence to improve public health practice. This presentation documents how knowledge gathered through a syndromic surveillance evaluation in Pacific Island Countries (PICs) with local health personnel was translated into action, in collaboration with global health partners. The evaluation identified a critical need to better equip local public health officials with the knowledge and skills to rapidly and appropriately respond to suspected infectious disease outbreaks across the Pacific. Principally funded by Australian aid and developed in partnership with the World Health Organization, the Secretariat of the Pacific Community and the Pacific Public Health Surveillance Network, *RAPID (Response and Analysis for Pacific Infectious Diseases):* is an example of a multi-organisational approach to swiftly address identified surveillance issues and strengthen regional surveillance capacity. Training, on-site capacity building, mentoring, peer-to-peer exchanges, outbreak support, and surveillance and response tool development were included in the project.

The *RAPID* project is a notable example of how evidence gathered through a surveillance evaluation can be used to improve public health surveillance practice. The project showcases how gains in surveillance capacity in lower and middle income countries can be achieved through cooperative partnerships and flexible approaches.

Ebola epidemic in West Africa: review of Australia's response

Presenter and author: Rosalie Schultz, Aspen Medical, NT

Ebola Virus Disease is a severe and frequently fatal illness caused by a zoonotic virus of the family Filoviridae whose likely main reservoir is bats. Human to human transmission of Ebola Virus occurs through contact with body fluids from infected patients. Patients are known not to be infectious until symptoms develop.

WHO declared the Ebola outbreak that began in 2013 in West Africa a Public Health Emergency of International Concern in August 2014. As part of its Ebola Response Roadmap, WHO recommended that all countries develop procedures to detect and respond to an Ebola exposure.

Australians have supported the global response to the outbreak through WHO and NGOs. The Australian government responded in November 2014, contracting an Australian company to manage an Ebola Treatment Centre in Freetown, Sierra Leone. The government also issued a ban on the issue of visas to people from countries affected by the outbreak. Australia's health protection services at national and state/territory levels have developed protocols to screen and manage all in-coming travellers from Ebola affected countries, including potentially exposed aid workers entering Australia.

While the Ebola affected countries in West Africa are remote geographically, the international concern of Ebola has demanded action from Australia.

Active TB case finding in the outer islands of Kiribati: A golden opportunity

Presenters: Onofre Edwin A. Merilles, Jr., Secretariat of the Pacific Community, Noumea New Caledonia and Takeieta Kienene, Ministry of Health and Medical Services, Republic of Kiribati

Authors: OE Merilles Jr., T Kienene, R Stapledon, R Lumb, R Brostrom and A Roth

Setting: Republic of Kiribati, ranks first in tuberculosis case notification rate in Western Pacific Region, 5-year annual average contact tracing yield of 14 cases (~3%), GeneXpert MTB/RIF in place, and Commonwealth of Australia funded 5-year project to accelerate scale-up

Objective: Expand TB case-finding efforts to high tuberculosis-burden outer islands through nurses doing island census

Design: Prioritisation of target islands; development of protocols to guide screening and diagnosis; training of nurses on symptom screening tool; training of clinic and laboratory staff on use of the GeneXpert MTB/RIF machine and implementation of algorithm; screening done from 23 February to 23 March 2015

Results: 3 outer islands targeted for screening with 13,373 population; Hawaii TB Symptom Screening tool customised for use in Kiribati; GeneXpert algorithms for diagnosis developed; village nurses trained on the screening protocol; 26 clinicians and 5 laboratory staffs trained on GeneXpert; 137 persons with suspicion of TB expected to be identified; yield of at least 24 bacteriologically confirmed pulmonary smear positive and negative TB cases

Conclusions: The TB programme envisions demonstrating that increasing the scope and scale of TB case-finding need not be expensive. Coordinating the initiative with other routine health activities and aligning screening and diagnostic work-up with new technologies are keys to success.

The Ebola 'end game' in Montserrado County, Liberia – some of the challenges

Presenter and author: Linda A Selvey, School of Public Health, Curtin University

I spent four weeks as WHO Field Coordinator for Montserrado County in January/February 2015. Montserrado county is the largest county in Liberia and includes the capital city, Monrovia. At that time, Montserrado was the only county in Liberia with onging Ebola transmission. Transmission was localised to two densely populated communities within the county in two transmission chains. While that might suggest that elimination of transmission was very close, three cases presented very late and between them exposed over 100 high-risk contacts, including in other communities and another county.

Community leadership conflicts, denial about Ebola, fear of the ETU and mistrust of contact-tracers impeded efforts to stop transmission. In this presentation, I will discuss these challenges, and initiatives that were put into place to address them.

Treatment delay among the tuberculosis patients of Bangladesh

Presenter: Mahfuza Rifat, University of Newcastle, Australia

Authors: J Hall, C Oldmeadow and A H Milton

Background: Bangladesh is one of the WHO ranked high burden country for tuberculosis. Delay in treatment may lead to progression of disease, poor outcome and increased risk of transmission. We conducted this study to identify the delays in treatment of tuberculosis and its association with other factors.

Method: Observational study including cross section of 646 drug sensitive tuberculosis patients. Information was collected through face-to-face interviews and record reviews. Unadjusted and multivariable regression were used.

Result: Median patient, health system and total delay were 2 weeks, 6 weeks and 9 weeks respectively. Compared to 'no education', patients with some education had less total delay (*P* 0.014); who consulted the informal providers (*P* 0.029) had more total delay compared to the designated centres of National

Tuberculosis programme. Health system delay was associated with private (*P* 0.007) and informal providers (0.004) and it was less with 'some level education' (0.004). Patient delay was associated with some occupations such as 'service' (*P* 0.042) and 'homemakers' (0.05).

Conclusion: Although the median delays have been much reduced compared to the previous studies conducted in Bangladesh, the health system delay showed that involvement of private and informal providers are still needed to improve the control programme.

Identification of measles vaccine virus by PCR in children more than 100 days following first dose of measles-containing vaccine

Presenter: Vicki Slinko, Metro South Public Health Unit, Brisbane, Queensland

Authors: VG Slinko, J McMahon, F Moore, CA Quagliotto, KAJ Jarvinen, BJ McCall, J Smith and SB Lambert

Fever, rash, and malaise due to measles-containing vaccines (MCVs) are well recognised adverse events, commonly occurring 7–10 days (range 5–12 days) after administration, and generally lasting 2–3 days. During these episodes, measles vaccine virus can be detected in clinical specimens, but detection long beyond this time frame, or person-to-person transmission of measles vaccine virus, has not been described.

Here we report a series of six children with detection of measles vaccine virus by polymerase chain reaction (PCR) testing during a clinical illness where measles testing was undertaken. In each child, vaccine virus was identified more than 100 days (up to 548 days) after administration of their most recent dose of MCV (first dose of measles-mumps-rubella vaccine in each case). Increasing clinical suspicion due to regular importations of measles into south-east Queensland over recent years, and the expanded use of PCR methods for diagnosis, has revealed these occurrences. Measles vaccine virus is identified by the Queensland public health virology laboratory following an initial positive measles PCR, with subsequent PCR and/or sequencing for vaccine strain. These findings alter the public health response required, with vaccine virus considered non-infectious, and also have implications in the longer term, for progress towards measles elimination.

Innovative uses of hepatitis A molecular testing in South East Queensland

Presenter: Vicki Slinko, Metro South Public Health Unit, Brisbane, Queensland

Authors: VG Slinko, J McMahon, KAJ Jarvinen, R Stafford, R Bell, K Heel, J Northill and BJ McCall

Diagnosis of hepatitis A infection has previously relied on demonstration of IgM antibodies against hepatitis A virus (HAV) in serum of acutely ill or recovering cases. Recently polymerase chain reaction methods (PCR) of HAV detection are being utilised in South East Queensland (SEQ) to confirm the diagnosis, identify further cases to detect outbreaks and even determine likely infectivity. Genotyping and/or sequencing of HAV have been used to link cases and by referencing an international database the possible origin may even be determined.

We present information on multiple cases and clusters of hepatitis A infection where this innovative use of HAV PCR has been used in SEQ. Serum or faecal PCR testing has identified both secondary cases as well as asymptomatic primary cases in young children thought to have infected older family members who developed symptomatic disease. Faecal PCR testing was used to assume clearance for school attendance in a young asymptomatic child. A large community outbreak was able to be linked through combination of genetic testing and epidemiological contact tracing to a child care centre at the epicentre of HAV infection. The outbreak ceased after attendees and staff members were vaccinated.

Understanding Q fever in Australia, 1991 to 2013

Presenter: Timothy Sloan-Gardner, Australian Government Department of Health, ACT

Authors: T Sloan-Gardner, P Massey, P Hutchinson, K Knope and E Fearnley

In Australia, abattoir workers and farmers, or those handling animal birthing products or slaughtering animals are at higher risk of Q fever.

We analysed data from the National Notifiable Diseases Surveillance System from 1991–2013, along with enhanced data from New South Wales and Queensland, to examine changes in the epidemiology of Q fever. Data were analysed using negative binomial regression using Stata 13.

There was a significant reduction (IRR 0.80 CI 0.68–0.95) in the Q fever notification rate after the end of the National Q Fever Management Program, however, the rate appears to increase in 2013.

The highest rates were in 40–59 year old males from Queensland and New South Wales. The age of Q fever cases and the proportion that were female both increased over time. The most frequently listed occupation for Q fever cases involved contact with livestock (15%), followed by no known risk occupations (10%).

We found that Q is no longer confined to abattoir workers and farmers. It is time to re-evaluate the at-risk groups recommended for vaccination. Additionally, more comparable and complete enhanced datasets, either at the jurisdictional or national level, would aid in the understanding of the epidemiology of Q fever in Australia.

Are we ready for rubella elimination? A review of rubella and congenital rubella syndrome (CRS) notification and hospitalisation data, Australia, 2008–2012

Presenter: Jocelyn Chan, National Centre for Immunisation Research and Surveillance (NCIRS), New South Wales

Authors: J Chan, A Dey, H Wang, N Martin and F Beard

Introduction: Following the successful elimination of rubella in the Americas, the Western Pacific Region of the World Health Organization proposed in 2014 a goal of regional rubella elimination, with a target date to be determined. In the context of this impending push towards elimination, we reviewed rubella epidemiology in Australia between 2008 and 2012.

Methods: Data were extracted from national notification (2008–2012) and hospitalisation (2008–2011) databases. Data were analysed by year, age, sex, state/territory, vaccination status, Indigenous status and place of acquisition.

Results: The average annual rubella notification rate between 2008 and 2012 was 0.18 per 100,000. The average annual hospitalisation rate between 2008 and 2011 was 0.03 per 100,000. One case of CRS was notified in 2012 and one hospitalisation with a principal diagnosis of CRS was recorded in 2008. Thirty seven per cent of notifications were acquired overseas and 89% of notifications were diagnosed by serology alone.

Conclusion: Rubella continues to be well-controlled in Australia and CRS is rare. The very low incidence and increasing proportion of imported cases suggest that elimination has been achieved. However, formal verification of rubella elimination in Australia will require the expansion of genotypic surveillance to demonstrate the absence of endemic strains.

Are health care workers aware of vaccination recommendations and do they support mandatory vaccinations?

Presenter: Helen Marshall, The University of Adelaide, South Australia

Authors: J Tuckerman, L Shrestha, J Collins and H Marshall

Background: Understanding HCWs knowledge and awareness of vaccination recommendations is important for determining strategies to improve uptake.

Methods: A quantitative survey (n=92) of HCWs and qualitative semi-structured one-to-one interviews with 22 HCWs examined their awareness of recommended vaccines, vaccine uptake and mandatory vaccination. Descriptive statistics, thematic analysis and coding were used to examine data.

Results: Besides the influenza vaccine, awareness of recommendations was low, as was recall of vaccinations received in the last five years. Despite HCWs being aware of the process required for patients to receive vaccinations, few knew how to access the appropriate service for themselves.

HCWs' opinions towards mandatory vaccinations were divided. Many felt that the decision to receive a vaccination was a personal choice and that making any vaccinations mandatory would remove autonomy. Others insisted that vaccinations for HCWs should be mandatory or if not, stricter workplace regulations should be enforced. Agreement towards mandatory vaccination for seasonal influenza was strongly aligned with HCWs' perception of risk, even in otherwise vaccine objectors. Conversely, there was also a strong sense of disapproval, at the prospect of mandatory seasonal influenza vaccination, even from vaccine acceptors.

Conclusion: Increasing HCWs' knowledge and awareness of vaccination recommendations and vaccine access is needed to improve uptake.

ADVALUE: Incorporating young people's views into priority setting for immunisation programs

Presenter: Helen Marshall, Women's and Children's Hospital and Robinson Research Institute, University of Adelaide

Authors: H Marshall, A Parrella, J Ratcliffe, R Tooher and A Braunack-Mayer

Adolescents' views and preferences are often over-looked when public health strategies that affect them are being considered for implementation. This study aimed to assess adolescent views and preferences for determining priorities for immunisation programs. A youth jury was held to deliberate on the question "What criteria should we use to decide which vaccines for young people in Australia should receive public funding?"

Jury members were selected using a stratified sampling technique and were recruited from the community through a market research company. The Jury was conducted in metropolitan Adelaide over two days. Fifteen youth aged 15–19 years participated in the jury. The jury's key priorities for determining publicly funded vaccines were:

Disease severity – whether the vaccine preventable disease (VPD) was life threatening and impacted on quality of life.

1. Transmissibility – VPDs with high/fast transmission and high prevalence.

2. Demonstration of cost-effectiveness, taking into account purchase price, program administration, economic and societal gain.

Youth jurors also indicated that there should be targeted programs for children at high risk of severe disease where a publicly funded program was unavailable and that social disadvantage 'socially at risk' should be a priority group in addition to 'medically at risk' children.

Characteristics associated with pertussis notification during pregnancy

Presenter: Lisa McCallum, University of Western Sydney, Hunter New England Health

Authors: L McCallum, B Liu, P McIntyre and L Jorm

Background: Mothers are one of the most likely sources of pertussis infection in infants; however studies reporting the epidemiology of maternal pertussis infection are few.

Methods: We constructed a cohort of mothers and their infants using routinely collected population-based datasets in New South Wales (NSW), Australia. All NSW resident mothers who gave birth to a singleton infant from 01 Jan 1994 – 31 Dec 2008 were included in the study.

Results: Among mothers, there were 3,887 pertussis notifications, with 386 notified pertussis infections occurring during pregnancy giving a rate of 30.2 (95% CI 21.1–43.1) per 100,000 pregnancies during the 15-year period. After adjusting for all other covariates, older mothers, delivering in an epidemic year, having had a previous pregnancy and living in a high socioeconomic area or giving birth in a private hospital were associated with increased risk of pertussis notification during pregnancy. Smoking during pregnancy, having a first antenatal visit later in the pregnancy and being born overseas were significantly associated with a lower risk of pertussis notification during pregnancy.

Conclusion: Diagnosis of pertussis in pregnant women is affected by socioeconomic status and access to health services. These factors should be taken into consideration when using disease notifications to understand the epidemiology of pertussis.

NetEpi as a tool for managing contact tracing during a measles outbreak

Presenter and author: Anthony Draper, Centre for Disease Control, Northern Territory and MAE Scholar, Australian National University

A single case of measles, let alone an outbreak can stretch the resources of a public health unit. An intensive contact tracing effort is essential to reduce secondary cases and the subsequent significant costs to the health care system and loss of productivity in the community. Contact tracing is labour intensive with public health unit staff involved in telephoning contacts, arranging follow up, undertaking enhanced surveillance and additional tasks that take them away from other duties in the public health unit. The time staff dedicate to a measles outbreak results in a considerable financial/resource cost to the unit.

In response to a large outbreak of measles in early 2014, the NT began to use the internet based NetEpi tool to manage contact tracing activities. Using NetEpi streamlined and simplified our subsequent measles responses.

Measles activity in Queensland since 2013

Presenter: Jamie McMahon, Public Health Virology, Queensland Forensic and Scientific Services

Authors: J McMahon, J Northill, J Cameron, G Hewitson, D Genge and F Moore

Measles cases in Queensland have been on the rise endangering Australia's measles free status. Public Health Virology provide a statewide molecular testing service that includes both PCR testing and genotyping of samples to assist in contact tracing. The rate of measles testing increased sharply in August 2013, which saw the start of the largest number of measles cases in Queensland since 1994. These cases were determined to be not one continuous outbreak but many clusters originating from imported cases from within the Asia/Pacific region. It has also been observed that with a heightened awareness of measles in the community there is an increase in detection rates of measles vaccine reactions.

The identification of a hepatitis A cluster linked to the consumption of imported frozen berries through phylogenetic analysis

Presenter: Jamie McMahon, Public Health Virology, Forensic and Scientific Services, Health Support Queensland, Department of Health

Authors: J McMahon, J Northill, G Hewitson, J Cameron, D Genge and F Moore

Hepatitis A infections from contaminated food sources has been an ongoing problem with the globalisation of the world food trade. In recent years there have been significant outbreaks of Hepatitis A in developed countries linked to contaminated food grown or processed in countries where sewage management and sanitisation is poor. These have included frozen berries, lettuce and salad products, raw vegetables, sun-dried tomatoes and shellfish originating from areas of South America, Asia, Africa and the Middle East.

We report on the recent cluster of Hepatitis A cases linked to frozen berries imported from China. Identification of this cluster in Queensland has been performed by PCR and the genotyping of samples has been able to confirm the cases are genetically identical. A phylogenetic analysis on interstate genetic sequences has confirmed the cases are linked to a nationwide cluster of Hepatitis A with an epidemiological link to frozen berries originating from China and Canada.

Immunisation by community pharmacists – more than just a 'flu shot'

Presenter: Nicole Flyod, The Pharmacy Guild of Australia

Author: K Gourlay

With vaccination recognised as within the scope of practice for a pharmacist by the Pharmacy Board of Australia, this presentation will discuss the benefits of utilising community pharmacies within the current immunisation process in Australia, how this could be enhanced to increase the coverage of immunisations against vaccine preventable disease.

This presentation will:

- Provide a brief overview of the international experience,
- Outline the activities in preparation for the practice of immunisation by community pharmacist immunisers, including competencies,
- Explore opportunities beyond just seasonal influenza immunisation, particularly measles and pertussis, particularly in regional and remote locations; and
- Provide an overview of pandemic preparedness in community pharmacy.

Community dispensing of s100 HIV medicines – what does this mean?

Presenter: Nicole Flyod, The Pharmacy Guild of Australia

Author: K Gourlay

From 1 July 2015 amendments will be made to the prescribing and dispensing arrangements for HIV antiretroviral agents that will allow these medicines to be dispensed through a pharmacy of the patient's choice regardless of where they were prescribed.

Previously these medicines, known as Section 100 Highly Specialised Drugs (s100 HSD) through the Pharmaceutical Benefits Scheme (PBS), were unable to be dispensed at a community pharmacy. Eligible community based prescribers and patients are required to be associated with a hospital to be able to access these medicines.

In addition, the restriction preventing the manufacture and sale of HIV in vitro diagnostic devices (home self-tests) has been removed via an amendment to the *Therapeutic Goods (Excluded Purposes) Specification*

2010. While there are currently no HIV self-tests included in the Australian Register of Therapeutic Goods, it is expected that when a listing is made, this will improve testing rates and lead to earlier diagnosis, intervention, treatment and better health outcomes.

This presentation will provide an overview of these changes, what this means for community pharmacy, and importantly, what it means for people living with HIV.

Chlamydia screening through community pharmacy – a missed opportunity

Presenter: Nicole Flyod, The Pharmacy Guild of Australia

Author: K Gourlay

Chlamydia is the most common Sexually Transmissible Infection in Australia. At 6 February 2015 there were already 6,501 notifications for 2015, with more than 81,000 notifications in 2014. Research reveals less than 10% of young people in the high-risk age group for chlamydia attending general practice are tested despite a high attendance rate (85.6% female and 64.4% male).

With the recommendation for annual testing for all sexually active men and women aged 15–25 in Australia, access to screening through Australia's most accessible health care destination, community pharmacy, is essential to reduce the incidence and long-term health consequences of chlamydia through early detection.

Further, more than 400,000 units of emergency hormonal contraception (EHC) are provided each year, predominantly at community pharmacy. There is a need to ensure all consumers receiving EHC have the opportunity to be tested for chlamydia given current prevalence and re-infection rates.

This presentation will:

- Provide a brief overview of community pharmacy involvement in the UK National Chlamydia Screening Programme;
- Provide an overview of Australian activities and research to-date; and
- Explore options, referral pathways and barriers and enablers to chlamydia screening through community pharmacy.

Improved chlamydia surveillance in New Zealand: interpreting the data

Presenter and Author: Jill Sherwood, Public Health Physician, Health Intelligence Team, Institute of Environmental Science and Research Ltd, Wellington

Background: Chlamydia is the most commonly reported sexually transmitted infection (STI) in New Zealand. Historically STI surveillance was a clinic based sentinel system with data also provided by a limited number of laboratories. Recent improvements in laboratory-based surveillance have enhanced the analysis of incidence trends, testing patterns and test positivity.

Methods: Data were collected from laboratories in all district health boards (DHBs). Repeat tests within a defined episode period were removed. Population-based rates, testing rates and test positivity were calculated by DHB, age group and sex.

Results: The national chlamydia rate for 2013 was 633 per 100 000 population (15–19 year age group: 3080 per 100 000 and 20–24 year age group: 2981 per 100 000), all decreases from 2012. Test positivity was 7.8% (8.6% in 2012); highest in females aged 15–19 years (15.4%) and 20–24 years (9.2%). Testing rates were highest in those aged 15–19 years (205 per 1000) and 20–24 years (298 per 1000).

Conclusions: The reduction in rates should be interpreted with caution. The improved rate calculation is not directly comparable with previous estimates. Testing rates in the 15–24 years age group were lower than the rate mathematical modelling suggests is required to decrease prevalence.

Human papillomavirus control: how are we going with vaccination coverage 7 years in?

Presenter: Julia Brotherton, National HPV Vaccination Program Register, VCS, Victoria

Authors: J Brotherton, G Chappell, J Brosi, K Winch, B Barbaro and M Saville

The National HPV Vaccination Program was launched in April 2007. The National HPV Vaccination Program Register monitors the population coverage achieved by the program using doses notified to the register as the numerator and ABS estimated resident population estimates as the denominator.

This presentation will discuss the latest HPV vaccination coverage estimates from the Register, including updated national and state based estimates for females and males, and analyses by area level measures of socioeconomic status and remoteness. Trends over time will be highlighted, including the marked improvements in coverage noted in some States and Territories since the initial launch of the program.

Acute rheumatic fever – a public health dilemma

Presenter: Keith Edwards, Centre for Disease Control, Royal Darwin Hospital, NT

Authors: K Edwards, M Fittock, C Chamberlain, N Davies, M Van Leeuwen, N Missen and D Williams

Acute rheumatic fever (ARF) is a disease of poverty, poor living conditions and disadvantage. Indigenous people living in tropical north Australia have the highest recorded incidence of ARF in the world (3%). A first episode of ARF usually occurs in childhood and causes joint pains and inflammation but recurrent episodes severely damage heart valves leading to the need for major cardiac surgery. Despite surgery, heart damage results in early death for many affected Indigenous people. The underlying cause is the social determinants of health and improvement in living conditions progresses slowly for Indigenous people. The only way to control the impact of this disease is to provide four weekly injections of LA Bicillin to those who have had a first episode so as to prevent recurrence. This injection prevents streptococcal infection which is the trigger for the disease. The Department of Health NT has put in place a Rheumatic Heart Disease Control Program since 1997 based in the Centre for Disease Control. This presentation will outline the development of the program, its difficulties and successes and the way forward to control and prevent the impact of this disease until it can be eradicated by improved living conditions for Indigenous people.

Challenges and opportunities in implementing routine bacterial genomics in public health microbiology.

Presenter: Takehiro Tomita, Microbiological Diagnostic Unit Public Health Laboratory, University of Melbourne at The Doherty Institute for Infection and Immunity, Melbourne, Australia

Authors: T Tomita, T Seemann, M Valcanis, K Stevens, D Bulach, J Kwong, J Coventry, K Mercoulia, T Stinear and B Howden

A range of typing methods have been employed for bacterial pathogen characterisation and epidemiological surveillance, such as serotyping, phage typing, and MLVA. Whole genome sequencing (WGS) has the potential to revolutionise the assessment of pathogen relationships in public health settings. However, it is not straightforward to implement WGS in a highly regulated microbiology environment.

At the MDU PHL, we have invested significantly to confront the challenges of WGS in a public health laboratory, with a vision to make WGS the primary typing method. We have found in-house sequencing capacity is critical, allowing rapid turn-around times in urgent cases. Close collaboration with microbial bioinformaticians has been vital for rapid, automated data analysis, including in silico MLST predictions, resistome information, and SNP based phylogeny. Pathogens analysed by WGS to date include: *Listeria monocytogenes,* VRE, *Yersinia enterocolitica,* Shiga toxin-producing *E. coli,* and KPC-producing *Klebsiella pneumoniae*. Reports to stakeholders contained a phylogeny including publicly available reference genomes, epidemiological data, metadata and interpretations useful for consideration in epidemiological investigations.

Key challenges include: defining and maintaining QC criteria in an environment where technology is continually changing; developing WGS workflows that optimise cost-effectiveness and timeliness; and reporting methodologies that satisfy consumers who have extensive experience interpreting traditional typing methods.

What's happening in rural NSW; what can enhanced gonorrhoea surveillance tell us?

Presenter: Priscilla Stanley, Western NSW/Far West Local Health District

Authors: PL Stanley, AM Parker and DA Belshaw

Background: As a notifiable condition, gonorrhoea continues to increase throughout NSW. Enhanced gonorrhoea surveillance (EGS) was implemented for 6 months in NSW from August 2013. Prior toEGS, limited information existed about gonorrhoea transmission in regional/remote western NSW.

Methods: An epidemiological scan from EGS undertaken for Far West and Western NSW Local Health District, specifically looked at treatment, sexual orientation, infection source and Aboriginality.

Results: During the period of EGS, 92 new cases of gonorrhoea were reported. 67% of these gonorrhoea notifications occurred in regional/rural locations and 33% in remote locations. 70% of treating clinicians returned EGS forms. An epidemiological scan indicates 66% of notifications compliant in recording Aboriginality. Geographic variations were evident in recording infection source and sexual exposure with homosexual transmission common in central NSW, reflecting inner Sydney patterns and Aboriginal/heterosexual transmission frequent in remote regions and mixing of infection sources between sub-populations occurring in these regions.

Conclusions: Ongoing epidemiological analysis of gonorrhoea cases will monitor trends amongst western NSW sub-populations. The Population Health Unit coordinates GP sexual health training focussing on service provision to priority sub-populations as identified in the 3rd National STI Strategy 2014–2017, along with specific messaging based on geographic trends promoting services and safe sex messaging.

Making the numbers speak – Creative presentations of notifications data

Presenter: Kari Jarvinen, Metro South Public Health Unit, Queensland

Authors: K Jarvinen, G Pollard, A Neill and D Seesaengnom

Background: The greater Brisbane Metro South Hospital and Health Service (Metro South Health, MSH) covers a population of 1,052,000 including significant cultural diversity. Over 13,500 notifiable conditions are reported annually.

Methods: We developed MS-Excel™ templates to automate detailed analysis of disease notifications and trends in MSH compared with rest of Queensland. Annual average notification rates were calculated for 2011 to 2013, and trends over the 2004 to 2013 period were compared. Tabular and diverse graphical representations are used to analyse and illustrate disease trends.

Results: There has been an increase in chlamydia notifications among 15–29 year olds, demonstrated poignantly by topographical and 3D-mapping. Gonorrhoea notifications have increased among a slightly older age group, while a broader increase has been evident for syphilis. Pertussis notifications showed distinct 'mountain peaks' of increased notifications among children and older age groups during the 2009–2012 epidemic years. A concerning increase was seen in the proportion of annual notifications in children. Varicella rates have increased among the elderly. Cyclic peaks are seen for diseases such as cryptosporidiosis.

Conclusion: Automated data analysis templates have proved useful in detailed analysis of notifications data, including through generation of multiple formats for presenting information.

Is the National Notifiable Diseases Surveillance System an effective surveillance system for flu?

Presenter: Anna-Jane Glynn-Robinson, Office of Health Protection, Australian Department of Health, Canberra, Australian Capital Territory.

Authors: AJ Glynn-Robinson, M Kirk, K Pennington and R Owen

Background: Influenza is an acute viral infection that spreads easily from person to person, and is a serious public health problem that can affect any age group. Robust, reliable and adaptable influenza surveillance systems are therefore required to provide national policy makers with data to guide appropriate public health responses. In Australia, the National Influenza Surveillance Scheme combines data from a variety of surveillance systems, including community, primary and tertiary healthcare settings and laboratories, to estimate the impact of seasonal influenza. We have evaluated the National Notifiable Diseases Surveillance System (NNDSS) captures of laboratory-confirmed influenza notifications against the six objectives of national influenza surveillance.

Methods: Stakeholder surveys and notification data from 2008–2013 were used to examine the systems attributes, including simplicity, acceptability, flexibility, sensitivity and data quality. To assess the demographic representativeness of the system, we compared NNDSS notifications with data from the Australian Sentinel Practice Research Network and the Influenza Complications Alert Network.

Conclusion: The study found four of the six national influenza surveillance objectives are currently being met by the NNDSS. As an acceptable, simple and usable system, that provides high quality data, the NNDSS serves an important function in the national surveillance of influenza in Australia.

Who is at risk of *Legionella* infection in Australia?

Presenter: Anna-Jane Glynn-Robinson, Office of Health Protection, Australian Department of Health, Canberra, Australian Capital Territory.

Authors: AJ Glynn-Robinson, M Kirk, T Dobbins and R Owen

Background: *Legionella* causes atypical pneumonia after susceptible persons inhale the bacteria in soil or water. Two main species cause infection in Australia – *L. pneumophila* and *L. longbeachae.* We describe the epidemiology of legionellosis in Australia from 2001–2012.

Methods: We analysed notification data on legionellosis from the National Notifiable Diseases Surveillance System by person, place and time using denominator data from ABS. We conducted negative-binomial regression examining notification rates for separate species using Stata 13.1.

Results: *L. longbeachae* was responsible for 50% of the 3,862 notifications, compared to 45% for

L. pneumophila and 5% other species. The median age for *L. longbeachae* cases was 63 years (13–99 years) and 60 % were male. Rates were highest in Spring (NRR 1.24, 95%CI 1.1–1.4) and in Western Australia (NRR 5.24, 95%CI 5.0–6.5). The median age for *L. pneumophila* cases was 60 years (1–97 years) and 69% were male. Rates were highest in Autumn, and in Victoria (NRR 1.64, 95%CI 1.44 –1.86).

Conclusion: Older males were at greatest risk of infection. *L. pneumophila* predominated in eastern states, while in contrast *L. longbeachae* was more common in the west. Prevention measures should target the main infecting species in each jurisdiction.

Can molecular epidemiology enhance the surveillance of HIV in Queensland?

Presenter: Craig Davis, School of Population Health, University of Queensland, Queensland

Authors: C Davis, K Hawke, S Lambert, C Lang, C Gilks, L Fitzgerald and S Reid

Molecular epidemiology (ME), which employs both molecular and epidemiological methods to better understand the genetic diversity, aetiology and distribution of diseases, has only recently begun to be applied to understanding the dynamics of HIV transmission HIV. We systematically applied ME methods

with two linked data sets with the aim of better understanding the dynamics of HIV transmission in Queensland: (1) results of routine drug resistance testing in Queensland from 2008–2013 period; and (2) notification surveillance records (including demographic, exposure, and clinical information).

Of 1,767 sequences, 88% (1,563) could be linked with notification records. Overall, 78.7% (1,390) of sequences were subtype B, with the remaining comprised of subtypes C (9.4%, 166), CRF-AE (6.5%, 115), and other mainly recombinant subtypes (5.4%, 96). Subtype B sequences significantly decreased over the period ($p < 0.01$) whereas CRF-AE significantly increased ($p < 0.05$). The majority of clusters identified were small in size (<5) although several large clusters were also identified of which the largest (> 50) was subtype B and comprised mainly of men who have sex with men. We discuss the potential uses of this information to inform prevention and control efforts, as well as discuss the challenges and limitations of using these methods for ongoing surveillance.

Spatiotemporal model of anaemia intensity and helminth co-infection in Burundi

Presenter: Mohamad Assoum, The University of Queensland

Authors: M Assoum, R Magalhaes, G Ortu, MG Basanez, C Lau, L Veerman and A Clements

Longitudinal data was collected from 40,553 children over the course of 5 years in 31 locations in Burundi. These locations acted as satellite collection points. Data that was collected included: egg counts for Ascaris, Trichuris, hookworm and mansoni, blood iron levels, age, sex, weight and height. Locational data also include the GPS x and y coordinates, average land surface temperature, NDVI, average precipitation and the distance to perennial water bodies. Univariate, multivariate, geospatial analysis and statistical temporal analysis of the combined data was conducted based on previously produced prevalence, intensity and co-infection models. The spatiotemporal model produced demonstrated the correlation between the aforementioned environmental conditions, intensity of infections and the prevalence of co-infection and a rise in the prevalence and intensity of anaemia in children throughout Burundi. It was see that in areas which are closer to perennial water bodies and as such Schistosomiasis mansoni rates are high as well as increased prevalence of co-infection led to a significant increase in anaemia prevalence and intensity in the first year. However, over the course of the five years, with the introduction of Schistosomiasis treatments, a gradual decline in the intensity of anaemia was found in the same areas, although the prevalence did not change very much. Thus this model established a direct correlation between the environmental factors, co-infection prevalence and infection intensity and the onset of anaemia and its varying intensities over a five year period. This paper demonstrated how to utilise spatiotemporal models to help track and predict incidences of disease, thus acting as a pertinent public health surveillance system.

Pandemic level notifications in 2014 highlights problems with messages about influenza

Presenter: Heath Kelly, Victorian Infectious Diseases Reference Laboratory, Melbourne

Authors: H Kelly, K Grant, M Chilver, L McCallum and C Dalton

Background Influenza notifications in Australia were higher in 2014 than in the pandemic of 2009 and public messages suggested the 'killer flu was sneaking in early'. However, even early in the season, it was apparent that 2014 was unlikely to be severe. We aimed to evaluate notifications as a method for assessing the relative intensity of influenza seasons.

Methods Influenza notifications were retrieved from the National Notifiable Diseases Surveillance System from 2005–14. Notifications from reference laboratories were noted. We compared all notifications with assessment of influenza activity using two national syndromic surveillance systems, the ASPREN network of general practitioners and Flutracking.net, an online survey of community members throughout the influenza season. A similar analysis was made using only data from Victoria.

Results Nationally there were 67,783 influenza notifications in 2014, compared with 59,028 in 2009. Notification numbers had been steadily increasing since 2009 but syndromic surveillance systems in Australia and Victoria indicated that 2014 was a mild to moderate season. Since 2009, increasing numbers of influenza notifications have been received from non-reference laboratories.

Conclusion It is clear that notifications no longer reflect influenza activity in the Australian or Victorian communities. Emphasising only notification numbers led to inappropriate messages about the influenza season in 2014. Balanced messaging requires an understanding of all data sources providing information on seasonal influenza activity.

Human astrovirus in South-East Queensland 2014

Presenter: Judith Northill, Public Health Virology, Queensland Health Forensic and Scientific Services.

Authors: J Northill, V Horton-Szar, G Hewitson, D Genge, J Cameron, J McMahon, S Schlebusch and F Moore

Mamastrovirus 1, also known as Human Astrovirus (HAstV) is a species of virus currently known to have 8 genotypes and belongs to the family *Astroviridae*, genus *Mamastrovirus*. HAstV is a common cause of gastrointestinal disease in humans worldwide.

From August to December 2014, faeces samples were screened for common pathogens using a commercial multiplex tandem nested PCR at Mater Pathology. Samples where Astrovirus was detected were forwarded to Forensic and Scientific Services for confirmation and further genotyping. In total 29 samples were detected and phylogenetic analysis performed to determine the current genotypes circulating in South-East Queensland.

Shiga toxin-producing *Escherichia coli* screening in the Australian Capital Territory

Presenter: Laura Ford, ACT Health, ACT

Authors: L Ford, C Moffatt and K Kennedy

Background: Shiga toxin-producing *Escherichia coli* (STEC) causes acute gastroenteritis and can result in severe illness and large outbreaks. Previously only 1 case of STEC has been detected in the Australian Capital Territory (ACT). This study aimed to enhance local laboratory capacity and undertake enhanced laboratory-based surveillance in the ACT.

Methods: Between October 2011 and December 2013, selective screening for *stx*1 and/or *stx*2 toxin genes using a nucleic acid based test was performed on 901 faecal samples. Interviews were conducted with patients with positive *stx* samples. Notification rates were adjusted to the age and sex structure of the ACT population.

Results: STEC was detected in 17 of 791 samples included in the final study. Of the 17 positive isolates, 14 were from 680 residents of the ACT, which equates to a prevalence of 2.1% and an annualised notification rate of 1.6 STEC cases per 100,000 (95% confidence interval 0.8–2.5 cases per 100,000) in the ACT.

Conclusion: This study demonstrates that STEC is a cause of gastroenteritis in the ACT. Local laboratory testing capability can help effectively and efficiently monitor STEC in a small jurisdiction.

Public health challenges in dealing with a Salmonella outbreak

Presenter: Satyamurthy Anuradha, QLD Health

Authors: S Anuradha, S Jurd, F Vosti, I Hunter, J Markey, D Finnigan, D Jurgeneit, L Mundy, A Regan, D Brook, V Dingjan, E Pullen and P Van Buynder

Background: Raw eggs have been linked to more than one-third of food-related salmonella infections. In January 2015, the Gold Coast Public Health Unit investigated an outbreak of *Salmonella* Typhimurium (MLVA type STm3-12-11-12-524) that was linked to raw eggs used in desserts supplied to a sushi franchise chain, with nine out of 11 outlets being implicated.

Methods: Initial notifications for Gold Coast were of sporadic cases that on questioning were linked to sushi outlets within the area. Further epidemiologic investigation pointed to the dessert from these outlets as the culprit rather than the sushi. We undertook a systematic environmental health assessment, including sampling and/or environmental swabs from the dessert manufacturer, sushi retail outlets and egg producers.

Results: Our investigations showed positive results for the same MLVA type in all three locations. Poor sanitation in the farm, ignorance of the risks associated with the handling of raw eggs in food products and time-temperature abuse at the outlets were all contributing factors.

Conclusion: A comprehensive through-food-chain approach to investigating outbreaks will allow prompt identification of the source. More timely and appropriate public health interventions are critical to protect the public and should be enacted at every level of the food industry.

Investigating an outbreak of *Staphylococcus* food-poisoning amongst travellers across two Australian states

Presenter: Stephanie Fletcher, Public Health Unit; South Western Sydney Local Health District

Authors: L Boonwaat, T Moore, R Chavada and S Conaty

Background: *Staphylococcus aureus* is a common cause of food poisoning in Australia. Outbreaks associated with commercial caterers have been reported. However, outbreaks often go undetected because laboratory testing for enterotoxin producing *S. aureus* is not routinely done for this self-limiting condition.

Methods: A retrospective cohort study was conducted among a group of tourists who were hospitalised in Sydney shortly after travelling from Queensland. The group had consumed food from a restaurant on the Gold Coast prior to transit. Laboratory analysis on stool specimen and environmental assessment of the implicated restaurant was conducted.

Results: Epidemiological investigations linked the outbreak to a restaurant in the Gold Coast where the suspected food was produced. Two stool samples from of hospitalised cases were confirmed to have enterotoxin producing *S. aureus* and several environmental samples were found to be contaminated with *S. aureus*. Investigations suggested that mishandling of food at the implicated restaurant was the likely cause of this outbreak.

Conclusion: Food poisoning due to toxin mediated *S. aureus* is frequently undetected and under reported. Public health units should consider laboratory diagnosis of toxin-producing pathogens such as *S. aureus* when investigating outbreaks with very acute and self-limiting presentations after consuming food.

A parsimonious model for estimating influenza vaccine effectiveness

Presenter: Courtney R Lane, Victorian Infectious Diseases Reference Laboratory, Melbourne, Victoria

Authors: CR Lane, K Carville and H Kelly

Background: Influenza vaccine effectiveness (VE) is increasingly estimated using the test negative study design. Cases have a symptom complex consistent with influenza and test positive for influenza, while non-cases have the symptom complex but test negative. We aimed to determine a parsimonious logistic regression model for this study design.

Methods: We constructed directed acyclic graphs (DAGs) using publicly available software to determine the minimum covariate set required. VE was estimated as 1-adjusted odds ratio. Changes in VE from addition of specified covariates were examined using surveillance data from 2007–13, excluding 2009. Using the parsimonious model VE was estimated for each year, for all years combined, and for influenza type and sub-type for all years combined.

Results: The DAG indicated that covariates specifying year (season), age group, co-morbidity, and week of onset were required. Restriction by time between onset and swab was also necessary. The inclusion of

comorbidity was not validated when testing inclusion of the variables using 7 years of data. VE for all years combined was estimated as 53% (38,64). For H3N2 VE was estimated as 42% (19,59), for H1N1pdm2009 75% (51,88) and for influenza B 63% (38,79).

Conclusion: With the exception of comorbidity, theoretical covariates specified by the DAG were validated when tested against surveillance data. A parsimonious model using the case test negative design allows regular estimates of VE.

Influenza outbreak management in residential care facilities

Presenter: Robin Gilmour, Communicable Disease Branch, NSW Ministry of Health

Authors: R Gilmour, S Tobin and V Sheppeard

Residential care facilities (RCF) are not required under the NSW Public Health Act, 2010 to report influenza outbreaks to public health units (PHU), although many still do. Across facilities management and recognition of an outbreak often varies. To understand this further NSW Health developed a survey to generate information on how RCFs prepare for and respond to influenza and influenza like illness outbreaks.

An email containing a weblink to an online survey was sent to 528 RCFs service providers requesting the survey be completed by the Director of Nursing or a senior nursing staff member. Responses were received from 236 (45%) facilities. Preliminary results indicate that overall 223 (95%) responding facilities were familiar with the National Guidelines, however only 50% (118 facilities) of all nursing staff at a facility would have read or have ready access to the guidelines. The majority of facilities (94%) were able to identify residents with signs and symptoms of influenza but only 47% of facilities answered correctly the number of residents with an influenza like illness required to declare an outbreak.

Overall the majority of RCFs reported to be adhering to the National Guidelines. Information obtained through this survey will assist NSW Health plan future resources needed to prevent and help manage influenza outbreaks in RCFs.

Influenza in South Australia – is 2014 our worst year on record?

Presenter: Caitlin Graham, Communicable Disease Control Brach, SA Health

Authors: C Graham, E Denehy, J Raupach and A Koehler

Influenza became notifiable in South Australia in May 2008, since then notifications have ranged approximately 4,000 to 11,000 laboratory confirmed cases each year.

A descriptive analysis including hospitalisation at the time of notification for influenza in South Australia across a five year period (2009–2014) was undertaken using the South Australian Department of Health Notifiable Infectious Disease Surveillance database. Death information was obtained using medical notifications and records maintained by the Department of Births, Deaths and Marriages.

An average of 6991 cases per year was notified for the 2009 to 2014 period**.** During 2014, 11,054 cases of influenza were reported in South Australia. The dominant influenza type was influenza A (92%), which was higher than the previous year (43%), and closely resembles the 2012 season (90%).

In 2014, 14% of notified influenza cases were hospitalised; equivalent to the average percentage of hospitalisations for the last five years. In 2014, 44 deaths were notified; a rate of 40 deaths per 10,000 notifications. Reported death rates between 2009 and 2013 have ranged from 7 and 27 deaths per 10,000 notifications.

Continued surveillance will help describe the changing nature of influenza and to guide public health control measures.

Hepatitis A virus antibodies in Australian blood donors

Presenter: Megan Young, School of Medicine and MHIQ, Griffith University, Queensland

Authors: MK Young, HM Faddy, J Fryk, GR Nimmo, AW Cripps

Background: A recent Australian outbreak of hepatitis A associated with consumption of contaminated berries highlighted the importance of post exposure prophylaxis as a means of control in low incidence countries. Passive immunisation is required for the most vulnerable populations in the event they are exposed. Trends in hepatitis A seroprevalence may impact on the production of effective immunoglobulin products for passive immunisation.

Methods: The seroprevalence of hepatitis A antibodies in Australian blood donors was measured and compared to published literature to gauge the likelihood of a decline in immunity. Hepatitis A antibodies were quantified in a random sample of those who were seropositive.

Results: An estimated 51% (95%CI 48–54%) of Australian blood donors were seropositive for hepatitis A. Rates varied across the country and increased with age. Comparison with published data supported an increase in seroprevalence in younger age groups. The geometric mean titre (GMT) of those who were seropositive was 1246.8 mIU/mL (Geometric Standard Deviation 11.8 mIU/ml) and increased with age.

Conclusion: A seeming increase in seroprevalence among donors is encouraging regarding Australia's ability to maintain immunoglobulin sufficiency. However, the overall GMT of hepatitis A antibodies in donations may be prone to decrease as current donor cohorts age.

Laboratory characterisation of invasive *Listeria monocytogenes* in New South Wales between 2011–2014: from molecular subtyping to the whole genome sequencing

Presenter: Qinning Wang, Centre for Infectious Disease and Microbiology, Public Health, ICPMR Westmead Hospital. NSW

Authors: Q Wang, N Holmes, P Howard, G Hill-Cawthorne and V Sintchenko

Listeriosis is a life-threatening foodborne disease caused by *Listeria monocytogenes* (Lm). In NSW, a binary typing (BT) and multi-locus variable number tandem repeat analysis (MLVA) have been used routinely to detect clusters and track the food sources of infection. Between 2011 and 2014, 114 clinical and 65 environmental and food isolates were characterised. The majority of them were obtained from the elderly or immunocompromised patients. Molecular typing identified 14 and 25 BT unique types for environmental and human cases, respectively. BT 255 was the commonest (21.1%) type among human isolates followed by BT 254 (19.3%) and BT 158 (14%). BT 82 was the predominate type (32.3%) for the environmental isolates followed by BT158 (18.5%) and BT 154 (12.3%). 44 MLVA types were identified in which the predominant MLVA type (04-17-16-05-03-11-14-00-16) was associated with 13.8% of the cases, followed by another two types linked to 12.5% and 10% of cases, respectively. These MLVA types were associated with a multi-jurisdictional outbreak and a hospital-acquired outbreak in NSW. Application of the whole genome sequencing of Lm significantly improved the resolution of subtyping and assisted in establishing potential links between production facilities and sources of environmental contamination leading to outbreaks of listeriosis.

Recent evolution of human *Salmonella enterica* serovar Typhimurium in New South Wales

Presenter: Cristina Sotomayor, Sydney Medical School, Westmead, The University of Sydney, and the NSW Enteric Reference Laboratory (ERL), ICPMR-Pathology West, Westmead, New South Wales

Authors: C Sotomayor, Q Wang, P Howard and V Sintchenko

Background: Multi-locus variable number tandem-repeat analysis (MLVA) typing has been implemented for *Salmonella enterica* serovar Typhimurium (STM) in New South Wales since 2006. It has significantly improved the resolution of public health surveillance of STM infections. The aim of this study was to examine the diversity and trends of STM MLVA patterns.

Methods: All STM isolates genotyped by MLVA at the NSW ERL between January 2009 and December 2014 were analysed.

Results: 11,209 isolates were included in the study. The yearly counts have significantly increased from 1636 (2009) to 2548 (2014). The most dominant MLVA pattern was 3-9-7-13-523 (phage type 170) and its complex (variants with single tandem repeat differences in the second, third and fourth loci) represented 27.1% of all STM. The diversity of the STM population showed seasonal variation but remained stable over the study period, with a McIntosh's index of diversity between 0.58 and 0.78. The proportion of novel MLVA patterns varied between 7.2 and 38.1% (17% on average).

Conclusions: Prospective genotyping of STM has documented the establishment of successful PT170 clones and the sustained level of STM diversity that accompanies the increasing incidence of human STM infections in NSW in the last 6 years.

An ongoing public health response to a syphilis outbreak in the North.

Presenter: Mark Russell, Centre of Disease Control, Alice Springs, Northern Territory

Authors: L Garton, TW Yip, M Gunathilake, JY Su, A Ishwar, J Creighton, R Sherry, A Hope, C Beatson, H Goodwin, N Ryder, M Thalanany, V Krause

In September 2013, a localised increase in syphilis notifications was detected in central Australia. A few further cases were detected, in this region, until numbers began to rise July 2014 with linkages back to 2013. The Centre for Disease Control (CDC) determined an outbreak was underway and formed an outbreak response team to plan and implement a public health response. Screening for syphilis in all sexually active individuals aged 30 years or less, was recommended. The outbreak case definition expanded to include Katherine and Barkly regions by end of 2014, due to enhanced monitoring of cases detected.

Using a point of care test (PoCT), community screens in population aged 12 to 30 years, were implemented, in addition to increasing opportunistic testing in remote clinics. Testing was performed by both CDC and local staff teams, with local clinical and community leaders integrally involved in conducting the activities.

As of 10/04/2015, 115 outbreak cases have been detected, with 57% of cases being aged 15–19 years.

CDC continues enhanced surveillance of the outbreak to rapidly identify and contain new cases. Further community screens are being conducted. CDC is in close communication with other jurisdictions, to monitor epidemiological links across borders.

The health professional behind the syringe: benefits of a pharmacist vaccination program

Presenter: Lisa M Nissen, Queensland University of Technology, Queensland

Authors: LM Nissen, ETL Lau, C Campbell, H Kastrissios, BD Glass, A Drovandi and M Rosenthal

Background: The Queensland Pharmacist Immunisation Pilot (QPIP) which ran in 2014 was Australia's first to allow pharmacists to administer vaccinations. An aim of QPIP was to investigate the benefits of trained pharmacists administering vaccinations in a community pharmacy setting.

Methods: Participant demographics and previous influenza vaccination experiences were recorded using *GuildCare* software. Participants also completed a 'post-vaccination satisfaction survey' following their influenza vaccination.

Results: A total of 10,889 participant records and 8,737 satisfaction surveys were analysed. Overall, 1.9% of the participants reported living with a chronic illness, and 22.5% were taking concomitant medications. As part of the consultation before receiving the vaccine, participants acknowledged the opportunity to discuss other aspects of their health with the pharmacist, including concerns about their general health, allergies, and other medications they were taking. It was worth noting that 17.5% of people would not have received an influenza vaccination if the QPIP service was unavailable. Additionally, approximately 10% of all participants were eligible to receive a free vaccination from the National Immunisation Program, but still opted to receive their vaccine from a pharmacist.

Conclusion: The findings from this pilot demonstrate the benefit of a pharmacist vaccination program in increasing vaccination rates, and have helped pave the way for expanding the scope of practice for pharmacists.

Harnessing the mobile phone market: SMS for salmonellosis public health follow-up

Presenter and author: Neil Franklin, Health Protection NSW

NSW has over 4000 salmonellosis notifications each year which makes public health follow up of most cases impractical. When follow-up does happen, it is often weeks later and the ability to prevent further cases has passed.

Between 24/1/2015–20/4/2015 all salmonellosis cases notified by electronic laboratory reporting (ELR) in NSW were sent an SMS asking them to reply with information on businesses they ate at prior to becoming ill. The SMS included a link to the fact sheet and a public health contact number.

1,449 salmonellosis cases were notified during the study period. 438 (30%) cases notified by ELR were sent the text asking for risk information. Preliminary results include 187 (42%) replies. 12% reported overseas travel and 7% reported only eating food at home. One outbreak was detected by this method with 2 cases reporting the same event. Six cases mentioned food venues that were already identified via direct complaint about the venue or later identified from cluster investigation

Texting proved to be a quick method to contact salmonellosis cases and gather specific risk information. This pilot extended to all cases may prove a useful way to collect important risk information and detect outbreaks quickly.

Challenges in the management of Ebola Virus Disease: Experience from Medecins sans Frontieres (Sierra Leone)

Presenter: Clair Mills, Medecins sans Frontieres

Authors: C Mills, R Kremer, G Caleo and K Lokuge

In response to the 2014 West Africa Ebola outbreak, Medecins Sans Frontieres (MSF) opened five Ebola treatment centres in Sierra Leone. Surveillance and contact tracing, health promotion and psychosocial support activities were also implemented. Treatment capacity was grossly inadequate from May to end November. With 450–600 reported cases per week, there were only three designated Ebola treatment centres in Sierra Leone functioning, two of them run by MSF.

As of 7 April 2015, MSF had admitted 2,405 patients, 1,572 who were confirmed with Ebola. 740 died (CFR 47%).

The nature of Ebola and the unprecedented scale of this outbreak, combined with the delayed national and international response, led to very challenging situations and ethical dilemmas for the MSF field teams. Staff were faced with overwhelming needs, a high case fatality rate and extremely limited treatment options – while working in physically and mentally gruelling conditions.

Balancing risks to staff versus benefits to patients; the limitations of clinical care for patients with Ebola in this context; prioritising resources for treatment compared with prevention and control; and the need for rapid implementation of new drug and vaccine trials versus pressing operational demands are some of the challenges that will be discussed.

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