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Immunisation in NSW

# Using operational research to ensure that immunisation benefits are enjoyed by all

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In Australia, having government-funded vaccines with established efficacy against infectious diseases is a necessary measure to ensure that everyone enjoys the protection these vaccines offer. In addition mechanisms for effectively delivering these vaccines to hard-to-reach groups and those at greatest risk must also be identified, tested, refined and expanded appropriately. Locally generated evidence can inform immunisation strategies, both local and global, to ensure that children who need to be immunised will get vaccinated, and vaccinated on time.[1]

The operational research model – the systematic search for knowledge on interventions, tools or strategies that enhance program effectiveness – is increasingly recognised as the most appropriate method for addressing perplexing questions within public health programs.[2] Even though the focus of operational research is usually a particular setting (e.g. closely assessing the quality of and access to local services, identifying ways in which they can be improved and evaluating the feasibility of local approaches or interventions), the findings can be of global relevance, depending on the quality of research design and methods, and the generalisability of the findings.[3]

The operational research agenda may explore innovative approaches to program delivery or it may introduce and test relatively small refinements to improve the quality of existing service provision. For 'operational research' the need for review by an ethics committee should always be considered and if in any doubt, full ethics review should occur.

Strengths of the operational research approach include: high local relevance, ability to convince local decision-makers, relatively short lag times before findings are implemented, and cost-effectiveness.[4] This is patent in the three local operational research papers published in this edition of the *Bulletin*. It is encouraging to have contributions from three of the eight former area health services addressing priority immunisation issues that have important application beyond their local area. The timely identification and immunisation of Aboriginal infants; improved coverage with all antigens, including varicella, before school entry; and understanding the serotype replacement consequences of pneumococcal vaccination locally, are contributions that demonstrate the inherent value of the operational research approach. These three papers also demonstrate what can be achieved through investing in building the capacity for health program staff to apply appropriate research methods.

The remaining two papers in this special issue of the *Bulletin* can also be viewed broadly as operational research. The paper from the state's three largest paediatric centres describing the children's hospitals influenza vaccine initiative by Wood and Cashman demonstrates the value of effective collaboration to enable the timely investigation into an unexpected adverse event. Ongoing pertussis outbreaks pose questions about the appropriateness of current pertussis-vaccine containing schedules. The paper by Quinn and co-workers introduces the potential of pertussis serosurveillance as a relatively new tool to better understand perplexing pertussis epidemiology in Australia. This understanding will assist in decisions about optimal timing of vaccine doses.

The full benefits of operational immunisation research accrue when research findings are integrated into the immunisation program. Global public health bodies are strongly encouraging this 'follow-through' phase.[5] This phase is

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completed when dissemination of results has led to documented policy and/or guideline changes that are being monitored. The operational research philosophy is fully embraced when program staff continually consider ways of improving their program and test these ideas through further operational research.

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# Invasive pneumococcal disease in western Sydney, 2002–2010

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In January 2005 the 7-valent pneumococcal conjugate vaccine (7vPCV) was funded on the National Immunisation Program for all children as a three-dose regimen given at 2, 4 and 6 months of age with a catch-up program for children up to 3 years of age. In the same year, the 23-valent pneumococcal polysaccharide vaccine (23vPPV) was funded for all Australians aged 65 years and over. A 23vPPV nationally funded program for Indigenous Australians aged 50 years and over, as well as those aged 15–50 years with specified underlying medical risk factors, has been in place since 1999.[1]

We review the burden of invasive pneumococcal disease (IPD) in the former Sydney West Area Health Service (SWAHS) during the period 2002–2010 with particular attention to the proportion of IPD due to serotypes covered or not covered by the vaccines (Box 1).

## Methods

IPD (defined by the isolation of *Streptococcus pneumoniae* from a normally sterile site such as blood or cerebrospinal fluid) has been a notifiable condition under the NSW *Public Health Act 1991* since January 2001. All New South Wales (NSW) laboratories are required to report positive culture results to their local public health unit (PHU). All serotyping was performed at the Institute for Clinical Pathology and Medical Research, Westmead, one of three reference laboratories for this purpose in Australia. PHU staff enter this information, including serotyping results if available, into a statewide database.[2]

The population investigated was that of the former SWAHS (estimated 2010 population of 1 168 076). To demonstrate vaccine impact on disease burden the population was divided into three age categories for analysis: 0–4 years, 5–64 years and 65 years and over. Non-Aboriginal people aged 5–64 years are not routinely vaccinated. Annual age-specific rates and Poisson confidence intervals were calculated from January 2002 to December 2010 in Microsoft Excel® using notification data and Australian Bureau of Statistics population estimates from a population health database held by the NSW Department of Health (Health Outcomes and Information Statistical Toolkit). The average annual age-adjusted rates for each of the three age categories for the period 2002–2004 (baseline) were compared to the 2006–2010 post-vaccination implementation period. The serotype incidence percentage was calculated by summing the total cases caused by that serotype divided by the total notified cases for that time period.

## Results

### *Changing incidence of IPD over time by age category*

All three age categories showed a reduction in the age-specific IPD incidence over time with the greatest reduction in the 0–4-year age group (Figure 1).

Comparison of average annual IPD notification rates for the two time periods also shows that the greatest reduction in IPD incidence has been in the 0–4-year age group (72.8% reduction) (Table 1). The 5–64-year age group had a 39.4% decrease which is greater than that seen in the

**Table 1. Average annual age-specific invasive pneumococcal disease counts and incidence, Sydney West Area Health Service, 2006–2010 compared with 2002–2004**

| Age group (years) | Average annual count | | Average annual rate/100 000 | | Relative reduction (%) (95% CI) |
|---|---|---|---|---|---|
| | 2002–2004 | 2006–2010 | 2002–2004 | 2006–2010 | |
| 0–4 | 54 | 16 | 69 | 19 | 72.8 (66.1–78.5) |
| 5–64 | 56 | 36 | 6 | 4 | 39.4 (29.9–47.9) |
| 65 and over | 36 | 27 | 35 | 24 | 31.4 (18.9–42.4) |

Sources: NSW Notifiable Conditions Information Management System; Health Outcomes and Information Statistical Toolkit, NSW Department of Health.

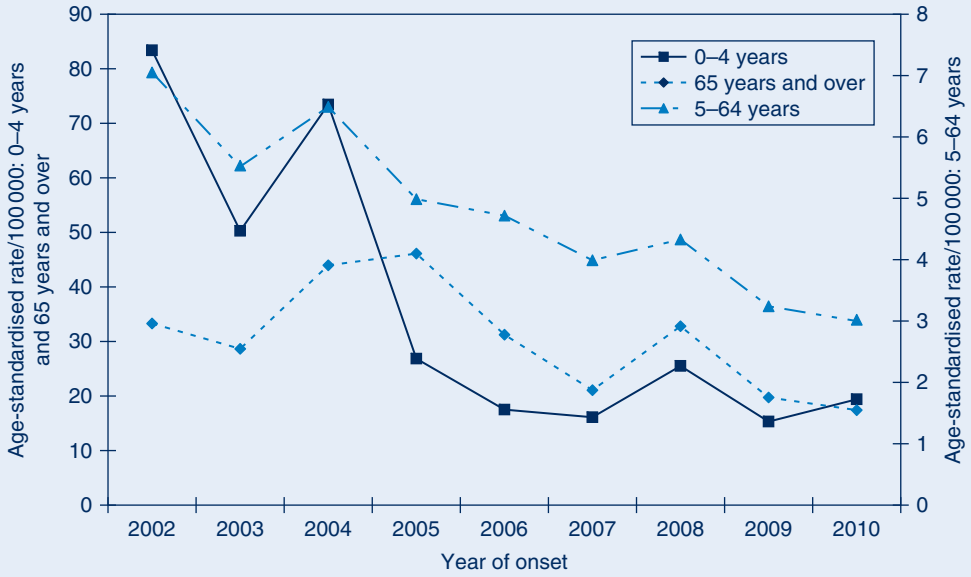


**Figure 1. Age-specific incidence rates, invasive pneumococcal disease, Sydney West Area Health Service, 2002–2010**
**Source: NSW Notifiable Conditions Information Management System.**

65 years and over age group (31.4%), despite this population being targeted for 23vPPV.

### *Comparison of serotype incidence*

The proportion of IPD cases due to serotypes contained in the 7vPCV was reduced in 2006–2010 compared with 2002–2004. Serotype 19A demonstrated the biggest increase, followed by serotype 3. Twelve of the 15 non-vaccine serotypes found in this population were relatively more common during 2006–2010. Non-vaccine serotypes caused 6% of all notified cases in 2002–2004 and 16% of all notified cases in 2006–2010.

## Discussion

A significant reduction in the overall incidence of IPD occurred in the SWAHS population since the National Immunisation Program pneumococcal vaccines were introduced. The greatest reduction was in children aged less than 5 years, although significant reductions were noted across all age groups. These results are similar to those from other settings where unvaccinated cohorts enjoyed a reduced incidence of IPD due to a herd immunity effect.[3–5]

Serotype epidemiology has also changed since the pre-vaccination period. All 7vPCV-containing serotypes have reduced in relative frequency across the entire population. Serotype 19A, a component of the 23vPPV, is now the dominant serotype. Factors in the emergence of 19A may include: poor 23vPPV coverage rates in the 65 years and over age group (54%);[6] inferior efficacy of the polysaccharide vaccine in elderly persons compared to that of 7vPCV in infants; and pressure from antibiotic use, as 19A is frequently resistant to penicillin and erythromycin.[7–10]

**Box 1. Serotypes covered by the three pneumococcal vaccines provided in NSW, 2004–2011**

7-valent pneumococcal conjugate vaccine (7vPCV): 4, 6B, 9V, 14, 18C, 19F, 23F

13-valent pneumococcal conjugate vaccine (13vPCV): all 7vPCV serotypes plus 1, 3, 5, 6A, 7F, 19A

23-valent pneumococcal conjugate vaccine (23vPPV): all 7vPCV serotypes plus 1, 2, 3, 5, 7F, 8, 9N, 10A, 11A, 12F, 15B, 17F, 19A, 20, 22F, 33F

## Conclusion

The introduction of the 7vPCV into the National Immunisation Program has significantly reduced the incidence of IPD across all ages but particularly in 0–4-year olds. Non-7vPCV serotypes now predominate, particularly serotype 19A. A 13-valent pneumococcal conjugate vaccine 13vPCV (which includes serotypes 19A and 3) will replace 7vPCV from July 2011 (Box 1). This is expected to reduce the incidence of IPD in the 0–4-year age group and potentially the remainder of the population through a herd immunity effect.

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# Aboriginal identification in Hunter New England infants

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The delayed immunisation of Indigenous children in the first year of life is an important issue in Australia.[1] The proportion of Aboriginal infants not fully immunised at 12 months of age (15%) is over double that of non-Aboriginal children (7%) in the Hunter New England Local Health District (HNELHD).

In the past when Hunter New England Population Health received Australian Childhood Immunisation Register lists of children overdue for vaccination, Aboriginal children were identified in partnership with local Aboriginal health workers and followed up in an attempt to facilitate more timely future doses of vaccine. However, this meant that children were already overdue at an age when they were most vulnerable to many vaccine-preventable diseases. This 'lesson from the field' describes a new approach to improve both Aboriginal immunisation rates and the recording by health staff of mothers' identification of their baby's Aboriginal status.

The Hunter New England Aboriginal Health Partnership requested that action be taken to close the gap in Aboriginal infant immunisation. The Partnership is an executive steering group with membership consisting of the Chief Executive Officer of the HNELHD and the chairperson or elected representative of each of the nine Aboriginal Community Controlled Health Services in the district. The Partnership aims to improve the health of Aboriginal people in the Hunter New England region by providing leadership, ongoing advice on general health policy, strategic planning, service issues and equity of allocation of resources. The Partnership provides a forum and a process for sharing information and is committed to the practical application of the principles of Aboriginal peoples' self-determination, a partnership approach and intersectoral collaboration. To facilitate immunisation, the Partnership supported the use of newborn data from all routine health service records for the purposes of contacting the child's parents.

Through a new approach to improve the timeliness of Aboriginal infant vaccination the parents of newborn Aboriginal infants are contacted soon after birth by an Aboriginal immunisation officer in the Population Health Unit. The officer facilitates the early linking of mothers with providers of immunisation. The approach also emphasises the importance of the accurate recording by health staff of mothers' identification of their baby's Aboriginal status.

As the program aims to contact the family of Aboriginal infants prior to their first scheduled immunisation, its success depends on the accuracy and completeness of Aboriginal identification recording in newborn datasets. However, the Aboriginal immunisation officer employed to contact the mothers of Aboriginal infants noted that the

records held by the Community Health Information Management Enterprise (CHIME), the principal inpatient database, were often inaccurate and did not reflect community knowledge. Consequently, this prompted the systematic comparison of recorded Aboriginal identification in two datasets, CHIME and *ObstetriX*.[2]

## Methods

The recorded identification of Aboriginal infants in two health service datasets was compared over a 3-month period, August–October 2010. Data from the NSW Health *ObstetriX* database were compared to the birth notification data available from the CHIME.

The *ObstetriX* database is completed in HNELHD maternity units. During the post-natal interview, midwives ask all mothers to nominate whether their baby will identify as Aboriginal and this information is then recorded in the *ObstetriX* database. Aboriginal births recorded in this database are supplied monthly to the Population Health Unit by the 15 maternity midwifery unit managers in HNELHD to permit follow up by the Aboriginal immunisation officer of these babies' mothers.

CHIME is the principal inpatient database in HNELHD and contains detailed patient demographic information collected during any presentation within the HNELHD. The CHIME data are automated and are available to the Population Health Unit within a few days of birth. However, Aboriginal identification of infants is not verified and defaults to the mother's recorded identity. This system populates all the HNELHD clinical records.

## Results

Less than half (46%; 72/158) of newborns were recorded as Aboriginal in both data sets. Fifty-three percent of newborn Aboriginal children (84/158) were only recorded in *ObstetriX* and 1% (2/158) only in CHIME.

## Discussion

Accurate recording by health staff of mothers' identification of their baby's Aboriginal status in medical information systems is essential to the success of the initiative linking Aboriginal infants and immunisation service providers. Strategies which allow Aboriginal people to identify themselves assist in the provision of services that can close the gap in health experience.[3]

The discordance between the *ObstetriX* and CHIME datasets identified by this study resulted in the HNELHD embarking on a program to encourage staff to support more complete identification by Aboriginal clients of the service. A training package for clerical staff who record demographic data was developed. Database managers now routinely compare Aboriginal identification data across databases, a quality measure initiated by this study.

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# Why are children on the NSW North Coast not being vaccinated against chickenpox?


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In November 2005, varicella (chickenpox) vaccination administered at 18 months of age was included in the government funded National Immunisation Program for all children born after 1 May 2004.[1] Each month the former North Coast Public Health Unit received a report on children recorded as overdue to receive the vaccination according to the Australian Childhood Immunisation Register (ACIR). It appeared that a disproportionate number of children aged 20–60 months were recorded as overdue for varicella vaccine.

This study explored why 907 children living in northern New South Wales (NSW) and aged 20–60 months as at April 2010 had received, according to the ACIR, all their other due vaccinations but not varicella vaccination.

**Box 1. Questions asked of parents as part of the study to determine why children aged 20–60 months living on the NSW North Coast were not vaccinated against varicella**

- Has your child been vaccinated against chickenpox?
  - If yes, give details of provider, date and batch number (from Baby Health Record)
  - If no, why not?
- Has your child had chickenpox (the disease)?
  - If yes, at what age did they have chickenpox?

## Methods

The parents of children aged less than 5 years and identified as being vaccinated with all other scheduled vaccines except varicella were sent: a copy of their child's vaccination record; a chickenpox factsheet; a questionnaire; and a letter explaining that their child was overdue for varicella vaccination and highlighting the importance of the vaccination. As this study formed part of routine follow-up of children identified by the ACIR as being overdue for vaccination it did not require ethics approval. This is in accordance with NSW Health Policy Directive PD2005_098.[2]

The questionnaire included questions asking whether their child had been vaccinated and whether the child had also had chickenpox. See Box 1 for questions.

The ACIR records for all children were checked 12 months after the initial contact.

## Results

A total of 406 questionnaires (45%) were returned to the Public Health Unit. More than a quarter of respondents ($n = 111$, 27.3%) indicated that their child had been vaccinated. This was verified by contacting their providers and updating the ACIR. Fifty respondents (12.3%) indicated that their child had experienced varicella infection and was therefore not vaccinated. Twenty-six of these children were reported to have had the infection before the age of 18 months.

The letter prompted 155 respondents (38.2%) to seek vaccination from their immunisation provider. Three percent of parents ($n = 12$) indicated that they had not been offered the vaccine by their vaccine provider, while approximately 6% ($n = 26$) indicated that they would rather their child got "natural disease". Other reasons for not vaccinating included wanting to wait until the child was older ($n = 2$), wanting to wait until the vaccine had been around for longer ($n = 2$) and medical contraindications (not registered with the ACIR) ($n = 4$). Some parents said that they had forgotten ($n = 7$).

Twelve months after the intervention, according to the ACIR 501 children (55%) remained unvaccinated and 42 parents (4.6%) had completed a conscientious objector form indicating they did not wish their child to receive the vaccine.

## Discussion

Based on the returned questionnaires and verification with the immunisation provider, many children who had no varicella vaccine recorded on the ACIR had been vaccinated or had experienced self-reported varicella disease. The simple intervention of a letter indicating their child's status, describing the potential complications of chickenpox and encouraging vaccination, prompted almost 40% of the respondents to have their overdue children vaccinated against chickenpox.

Globally, many families and countries cannot afford to protect their children against varicella and it is not a public health priority in settings where vaccine-preventable pneumonia (pneumococcus), diarrhoea (rotavirus) and measles are common and must remain the focus of immunisation programs.[3] However, in Australia, where the vaccine is available free to children, greater effort should be made to encourage parents and providers to optimally protect their children.

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# The seroepidemiology of pertussis in NSW: fluctuating immunity profiles related to changes in vaccination schedules

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**Abstract:** The pertussis epidemic experienced in NSW in 2008–2009 was likely to be in part due to changes in diagnostic practice since 2007, which amplified disease notifications. We used population-based seroepidemiology as a less biased means of interpreting age-specific pertussis infection patterns in NSW from three serosurveys undertaken in 1997–98 (during an epidemic), 2002 (post-epidemic) and 2007 (inter-epidemic), using a standardised pertussis toxin IgG enzyme-linked immunosorbent assay (ELISA). There was a decrease in the proportion of high anti-pertussis toxin IgG titres (>62.5 ELISA Units/mL) across all age groups in the 2007 serosurvey compared to the previous two serosurveys. In the 2007 serosurvey, the proportion of undetectable (<5 ELISA Units/mL) anti-pertussis toxin IgG titres increased in many age groups. The seroepidemiological profiles of the three serosurveys demonstrate fluctuating immunity profiles related to changes in vaccination schedules.

Despite longstanding immunisation programs in developed countries such as Australia and the United States, periodic epidemics of pertussis continue at intervals of 3–4 years on a background of endemic circulation. In 2008–2009, New South Wales (NSW) experienced a sustained pertussis epidemic which was unusually large in magnitude.[1] However, interpretation of notification data from the current pertussis epidemic is confounded by changes in diagnostic practice since 2007. The shift to widespread use of polymerase chain reaction for pertussis diagnosis in all age groups is likely to have amplified the number of cases notified during the epidemic, particularly in children.

Population-based, cross-sectional seroepidemiology offers a less biased means of comparison of age-specific patterns of pertussis compared to other disease surveillance methods, but requires acceptable standardisation and reproducibility of serologic tests and the ability to extrapolate these results to estimates of symptomatic cases. The European Sero-Epidemiology Network (ESEN) standardised the use of a serologic criterion for recent infection, measured in IgG-pertussis toxin (PT) using enzyme-linked immunosorbent assay (ELISA) Units[2] (EU) between participating laboratories using different serologic methods. This development allowed meaningful comparison of *Bordetella pertussis* seroepidemiology in six European countries.[3,4] This standardised methodology has previously been applied to Australian sera collected in 1997–98 and 2002 to describe national pertussis trends by age and over time.[5,6] Since then, several important changes in pertussis vaccine schedules have occurred in all states and territories, including NSW. First, the 18-month booster dose of diphtheria-tetanus-acellular pertussis vaccine (DTPa) was removed from the National Immunisation Program schedule in 2003. Second, an adult formulation acellular pertussis-containing vaccine (dTpa) was added to the schedule for adolescents, with school-based vaccination commencing in May 2004.[7] Earlier schedule changes are outlined in Table 1.

This study compares NSW data from three cross-sectional serosurveys, undertaken in 1997–98 (during an epidemic), 2002 (post-epidemic) and 2007 (inter-epidemic). The aim was to evaluate age-specific patterns of presumptive recent pertussis infection in the context of changes to the vaccine schedule and pertussis notifications.

## Methods

### *Population and study design*

The 2007 sera used in this study were selected from a bank of approximately 7200 sera collected opportunistically


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**Table 1. Significant pertussis vaccine schedule changes in NSW, 1975–2003**

| Year | Vaccine type | Event |
|---|---|---|
| 1975 | DTPw | Diphtheria-tetanus-whole cell pertussis (DTPw) national vaccination schedule recommended for infants aged 3, 4 and 5 months |
| | DTPw | Booster dose for infants aged 15–18 months introduced |
| 1978 | DTPw | Booster dose removed from schedule |
| 1982 | DTPw | National vaccination schedule changed to a primary series at 2, 4 and 6 months of age |
| 1985 | DTPw | Booster dose re-introduced at 18 months of age due to an increase in pertussis incidence in children aged 4–5 years |
| 1994 | DTPw | Booster dose at 4–5 years of age added to the recommend vaccination schedule |
| 1997 | DTPa | Diphtheria-tetanus-acellular pertussis (DTPa) recommended for the booster doses of vaccination |
| 1999 | DTPa | All five scheduled doses of DTPw replaced with DTPa |
| 2000 | DTPa | Second booster dose recommended at 4 years instead of 4–5 years |
| 2003 | DTPa | Booster dose at 18 months of age removed from schedule |
| | dTpa | Adult formulated diphtheria-tetanus-acellular pertussis (dTpa) recommended as a booster dose at 15–17 years of age |

from a geographically representative group of 29 diagnostic laboratories receiving samples from hospitalised and ambulant persons throughout Australia as part of a national serosurveillance program,[8] but was restricted to NSW laboratories for this study. The sera in the opportunistic sample were residual from specimens submitted for diagnostic testing and would otherwise have been discarded. Residual sera were from subjects who: were immunosuppressed; had received multiple or recent (within 3 months) blood transfusions; or were known to have HIV infection, were excluded by staff at the diagnostic laboratory. Sera were identified by a medical record number, sex, age, state/territory of origin and a unique identifier, to ensure that only one sample from any subject was tested. Approval for the serosurvey was obtained from the Western Sydney Area Health Service Human Research Ethics Committee.

In all relevant age groups the required sample size was calculated to achieve a 95% probability of precision ±7% around a point estimate based on the expected level of seroprevalence. There were equal numbers of males and females within each age group.

The population and study design for the 1997–98 and 2002 serosurveys have been described previously.[5,6] Across the three serosurveys the proportion of samples from outer regional and remote locations remained similar. In contrast, the proportion of samples from a metropolitan area decreased (85% in 1997–98 to 70% in 2007) and the proportion from an inner regional area increased (10% in 1997–98 to 25% in 2007).

### *Testing and serologic criteria for recent pertussis infection*

For the 2007 serosurvey a total of 1152 sera randomly selected from those available in each age group were tested using an established ELISA method adapted from Giammanco et al[9] and previously described in full.[5,6] The 1997–98 serosurvey was conducted at the ESEN reference laboratory at the University of Palermo, Italy and both the 2002 and 2007 analyses were performed at the Centre for Infectious Diseases and Microbiology, Sydney, Australia. The method used in our laboratory was validated against a panel of sera from the ESEN reference laboratory. In 2007 the assay was again validated, using a panel of samples from the 2002 serosurvey.

The minimum level of detection of the assay, defined as the minimum amount of antibody that must be present for the serum to have at least one optical density (OD) value within the linear range of the reference serum response curve, was estimated to be 2 EU/mL. Anti-PT IgG levels were divided into four categories, previously described by the ESEN study group as suggestive of pertussis exposure within certain time periods:[4] $<5$ EU/mL (undetectable), $5 - <62.5$ EU/mL (exposure more than 12 months previously), $62.5 - <125$ EU/mL (exposure within 12 months) and $\geq 125$ EU/mL (exposure within 6 months). Exposure implies 'significant' exposure, with antibody response and is inclusive of infection and immunisation. These categories were originally derived by de Melker and colleagues from a longitudinal cohort study of Dutch patients with clinically confirmed pertussis infection, and demonstrated that high anti-PT IgG levels can be a sensitive and specific indicator of recent infection. These levels fell below the nominated threshold levels in almost all patients within 12 months of infection.[2]

### *Statistical analysis*

Proportions and 95% confidence intervals were calculated for sera in each of the categories described above, by age group. A chi-square test was used to compare proportions by age group and serosurvey, with $p$ values $<0.05$

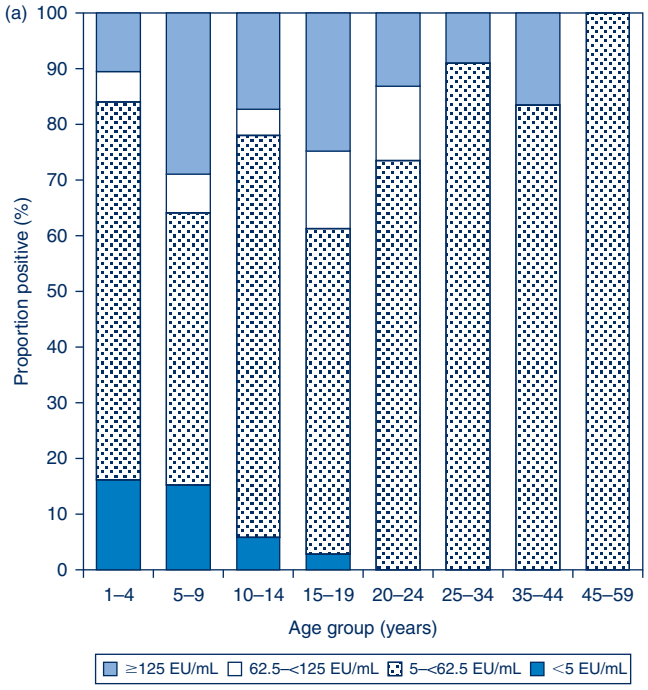


**Figure 1a. Cross-sectional distribution of anti-pertussis toxin IgG levels in the NSW population in eight age groups (1–4 through to 45–59 years) in 1997–98.**

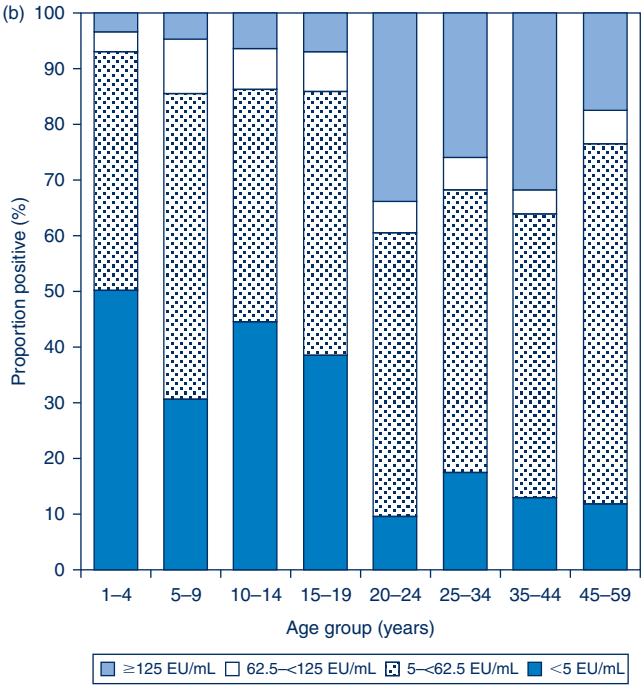


**Figure 1b. Cross-sectional distribution of anti-pertussis toxin IgG levels in the NSW population in eight age groups (1–4 through to 45–59 years) in 2002.**

considered significant. Analyses were performed using Microsoft Excel and SAS (version 9.2, SAS Institute, Cary, NC, USA).

## Results

The distribution of titres in each of the three serosurveys is provided in Figure 1(a,b,c). In 1997–98, the highest proportion of anti-PT IgG levels ≥62.5 EU/mL was seen in the 5–9 and 15–19-year age groups. In contrast, in 2002 the highest proportion of anti-PT IgG levels ≥62.5 EU/mL was seen in the 20–24, 25–34 and 35–44-year age groups. The proportion with anti-PT IgG levels ≥62.5 EU/mL was reduced across the population in 2007 compared to the previous collections.

To further highlight these differences, the proportion of sera with anti-PT IgG levels ≥62.5 EU/mL across the three collections are presented in Figure 2. When comparing the 2002 serosurvey to the 1997–98 serosurvey, significant decreases were seen in the 5–9 and 15–19-year age groups. When comparing the 2007 serosurvey to the 2002 serosurvey, significant decreases were seen in the 5–9 and 10–14-year age groups and in all age groups for those aged 20 years and over.

If the population proportions with anti-PT IgG levels <5 EU/mL (undetectable) over the period of the three collections are compared, many age groups experienced a significant increase in undetectable levels (Figure 3).

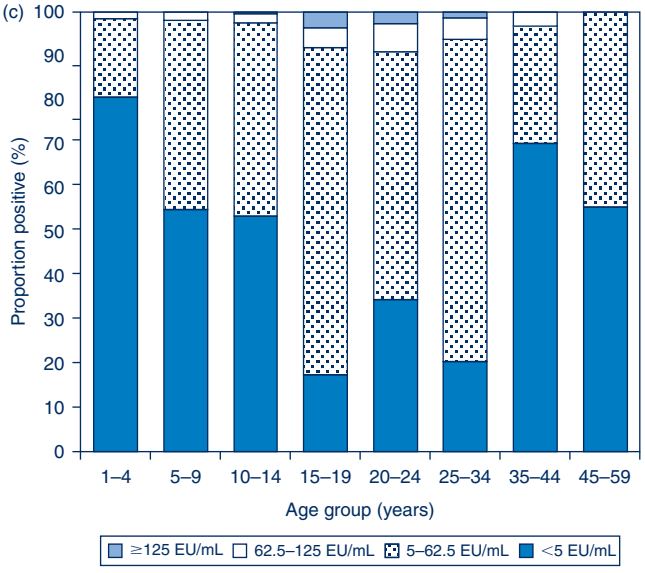


**Figure 1c. Cross-sectional distribution of anti-pertussis toxin IgG levels in the NSW population in eight age groups (1–4 through to 45–59 years) in 2007.**
**Sources: 1997–98, 2002 and 2007 serosurveys.**

In particular, the proportion of 1–4-year olds increased from 16% in 1997–98 to 81% by 2007 and the proportion of 35–44-year olds increased from 0% in 1997–98 to 70% by 2007. The only age group for which a significant decrease was observed between the 2002 and 2007 collections was 15–19-year olds.

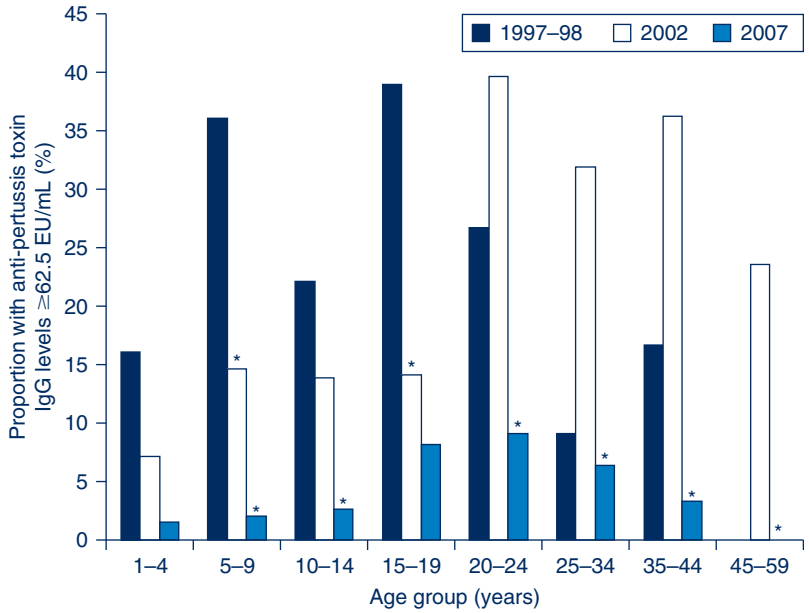


**Figure 2. Cross-sectional proportion of anti-pertussis toxin IgG levels (≥62.5 EU/mL) in the NSW population in eight age groups (1–4 years through to 45–59 years) in 1997–98, 2002 and 2007.**

*Significant decreases compared to the previous collection.

**Sources: 1997–98, 2002 and 2007 serosurveys.**

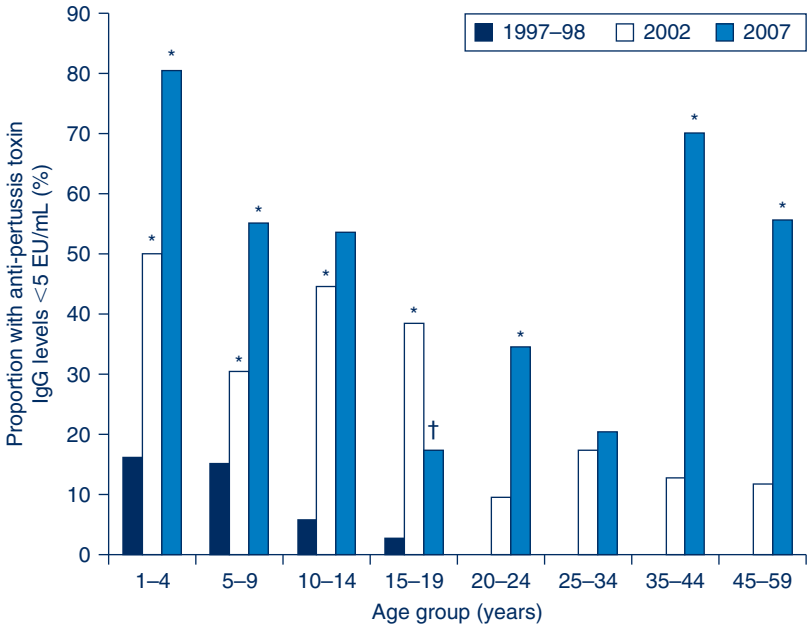


**Figure 3. Cross-sectional proportion of undetectable anti-pertussis toxin IgG levels (<5 EU/mL) in the NSW population in eight age groups (1–4 years through to 45–59 years) in 1997–98, 2002 and 2007.**

†Significant decreases compared to the previous collection. *Significant increases compared to the previous collection.

**Sources: 1997–98, 2002 and 2007 serosurveys.**

## Discussion

This study compares patterns of anti-PT IgG by age, between three time periods. The use of seroepidemiological data for this purpose avoids problems associated with traditional disease surveillance data, such as changes in diagnostic testing practices and physician awareness.[10,11] Overall, in the 2007 serosurvey there was a decrease in the proportion of high anti-PT IgG levels (≥62.5 EU/mL) across all age groups, compared with earlier serosurveys, which was significant for many age groups. This was in keeping with the relatively low levels of pertussis notifications in this year, which immediately preceded epidemic

notifications in NSW.[1] Another unusual finding was the increase in the proportion of undetectable anti-PT IgG levels (<5 EU/mL), which again was significant in several age groups.

Of particular note is the significant decrease in high anti-PT IgG levels in the 1–4-year age group, from 16% in 1997–98 to 2% in 2007, despite this age group being eligible for recent vaccination, which is thought to be a contributor to high anti-PT IgG levels in serological surveys.[2] The lower proportion of high anti-PT IgG levels in the 2007 serosurvey in sera from children aged less than 10 years is also reflected in notification data showing low level reporting for this age group in the period 2002–2007.[12,13] The 1–4-year age group in the 2007 serosurvey were not eligible for an 18-month booster dose of DTPa after it was removed from the schedule in September 2003.[7] This may have contributed to the significant increase in the proportion of undetectable anti-PT IgG levels in this age group, and is consistent with significant waning of immunity after the primary series of DTPa in the absence of a later booster.

In the 1997–98 serosurvey, adolescents aged 10–14 years (not eligible for a pre-school booster) and 15–19 years (eligible for three-dose primary DTPw vaccination only) had high proportions of high anti-PT IgG levels, which again reflected high notification rates in the 10–19-year age group at this time.[14] In the 2002 serosurvey, high anti-PT IgG levels continued to be observed for the cohort eligible for a primary series of DTPw only, who were now young adults aged 20–24 years. The relatively high rate of notifications in the 10–19-year age group also continued, reaching a peak in 2001 of 132 notifications per 100 000 population.[14] This rate fell to 62 per 100 000 population in 2002, and mirrored the 2002 serosurvey finding of a significantly lower proportion of high anti-PT IgG levels for both the 10–14 and 15–19-year age groups compared to the 1997–98 serosurvey. The decline in anti-PT IgG levels in the 10–14-year age group may be attributable to children who had received the scheduled pre-school booster dose moving into this cohort.[15]

Despite the availability of booster doses during childhood for the now adolescent cohort, coverage was poor, in part due to concerns about the safety of DTPw.[10,16] When an adult formulated acellular pertussis-containing vaccine (dTpa) became available, it was added to the schedule as a booster dose for adolescents aged 15–17 years.[7] The dose would target the cohort with sub-optimal childhood booster coverage, who continued to be conspicuous in pertussis notifications[17] and it was hoped that in the future would prevent an increase in cases due to waning of childhood vaccinations. In 2004, NSW Health delivered this vaccine through a school-based program to those aged 12–19 years and followed this with delivery to Year 7 students (aged 12–13 years) in 2005. The majority of those targeted in this program were aged 15–19 years at the time of the 2007 serosurvey. This was one of the few age groups to not show a significant decrease in the proportion of anti-PT IgG levels between the 2002 and 2007 serosurveys (Figure 2). Also, in contrast to all other age groups, the 15–19-year age group had a significant decrease in the proportion of undetectable anti-PT IgG levels in 2007, compared to the 2002 serosurvey (Figure 3). This may largely be attributed to the vaccination program, as disease activity between 2002 and 2007 was low.[12,13]

The large increase in undetectable anti-PT IgG levels in the 35–44-year age group is of concern, as this may indicate an increased susceptibility to infection at an age when people are likely to be parents of young infants. Young infants are most at risk from severe disease and death due to pertussis,[13,18] and parents are an important source of infection.[19,20] At the present time a 'cocooning strategy' (vaccinating close contacts of infants) is the recommended method of protecting infants too young to be immunised for pertussis.[21] In March 2009, in response to the epidemic, NSW Health commenced funding of a cocooning dose of dTpa for parents, grandparents and other close contacts of infants less than 12 months of age.[22] The success of this strategy is currently being investigated.

There were several limitations to this study. The sera used in this study were opportunistically collected rather than randomly sampled. We tried to minimise any biases by obtaining sera that were submitted for a range of diagnostic tests (excluding HIV) to major laboratories throughout NSW that serviced mainly ambulatory populations. We cannot exclude the possibility of selection bias toward a more sick population. No information regarding the clinical status of individuals whose sera were used here was collected and we did not exclude sera that were submitted for the diagnosis of pertussis. However, the method used here has been validated for measles against a prospective, cluster-sampling method and was found to be representative.[23] The immunisation status of subjects was unknown. While it is possible to estimate immunisation coverage for the childhood population in this study, it is largely unknown for the older age groups.

## Conclusion

The seroepidemiological profiles of the three serosurveys demonstrate fluctuating immunity profiles related to disease patterns and changes in vaccination schedules. The unique seroepidemiological profile from the 2007 serosurvey is interesting and requires further analysis. The small proportion of high anti-PT IgG levels indicative of recent infection, coupled with an increased proportion of very low anti-PT IgG levels, is likely to have set the stage for a large, sustained epidemic. Testing and analysis of serological profiles using a collection from the time of the epidemic would be a valuable means to elucidate the role of population immunity in pertussis epidemic cycles.

## Acknowledgments

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# Influenza immunisation program at three tertiary paediatric hospitals in NSW in 2010

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**Abstract:** This is a report of an innovative influenza immunisation program in three tertiary paediatric hospitals in NSW. A targeted once-off program of influenza immunisation funded by NSW Health was offered during 2010 at the Children's Hospital at Westmead, Sydney Children's Hospital and the John Hunter Children's Hospital. Authorised immunisers offered influenza immunisation to paediatric patients, outpatients and relatives of children with chronic illnesses. Influenza immunisation was administered to 3458 people, 1251 (36%) of whom were children with chronic conditions. In 2009 before the program, 420 influenza vaccines were prescribed for children in two of these hospitals. This number increased to 949 in 2010, the year of the program. Dedicated vaccination clinics at tertiary paediatric hospitals provide additional opportunities to ensure that children at high risk of severe influenza disease and its complications are vaccinated. The information obtained from the hospital vaccination program contributed to the national investigation of febrile convulsions following influenza vaccines in children in 2010.

Young children and infants have a high incidence of influenza infection, and hospitalisation for the treatment of influenza and its complications is most common in children less than 2 years of age.[1–3] The Australian Immunisation Handbook recommends influenza immunisation for children over 6 months of age, and strongly recommends influenza immunisation for children over 6 months of age with medical conditions predisposing to severe influenza.[4]

Many children with moderate-to-severe chronic medical conditions attend outpatient care at one of the three major paediatric hospitals in New South Wales (NSW), the Children's Hospital at Westmead, Sydney Children's Hospital and the John Hunter Children's Hospital. Over 400 000 children are seen each year in the outpatient departments at the three hospitals; approximately 290 000 children are treated at the Children's Hospital at Westmead, 100 000 at Sydney Children's Hospital and 20 000 at the John Hunter Children's Hospital.[5] The Sydney Children's Hospitals Network, including Sydney Children's Hospital and the Children's Hospital at Westmead, has over 44 500 inpatient admissions annually, making it the largest Australian and New Zealand children's health service.

In 2010 NSW Health provided funding to conduct influenza vaccination at these three hospitals through a targeted once-off program. The program funding was for a 6-month period and was principally used to employ nurse immunisers with the aim of offering influenza immunisation to protect paediatric inpatients and outpatients and decrease the numbers of preventable influenza hospitalisations.

Siblings and parents of children with medical conditions attending outpatient clinics were also offered influenza vaccination to reduce opportunities of influenza exposure thereby cocooning the medically at-risk children.

This paper describes the program delivery of influenza vaccine to children with chronic medical conditions and their families at the three major paediatric hospitals in NSW in 2010.

## Methods

The clinics at each hospital varied in location, timing and staffing. Individual details including unique hospital identifier, age, sex, presence of chronic medical condition, influenza vaccine received and contact details were recorded at the time of vaccination. All clinics commenced offering the monovalent H1N1 2009 vaccine, Panvax® (CSL Biotherapies, Australia), in February 2010 and changed to seasonal trivalent inactivated influenza virus (TIV) vaccine in March 2010 when it became available. The John Hunter Children's Hospital used only Influvac® (Solvay Biologicals, 0.5 mL) as the seasonal influenza vaccine, as it was the only vaccine obtained by the hospital, while Sydney Children's Hospital and The Children's Hospital at Westmead used either Fluvax®



10.1071/NB11039

**Table 1. Numbers of influenza vaccines administered at three NSW paediatric hospitals to children with chronic medical conditions and their families, 2010**

| | Monovalent H1N1 (Panvax) *n* | Children with chronic medical conditions given monovalent H1N1 (Panvax) *n* (%) | Seasonal Influenza vaccine *n* | Children with chronic medical conditions given seasonal influenza vaccine *n* (%) | Total administered vaccine N |
|---|---|---|---|---|---|
| CHW | 543 | 174 (32) | 1427 | 413 (29) | **1970** |
| SCH | 232 | 108 (47) | 297 | 194 (65) | **529** |
| JHCH | 391 | 166 (42) | 568 | 196 (35) | **959** |
| **Total** | **1166** | **448 (38)** | **2292** | **803 (35)** | **3458** |

CHW: The Children's Hospital at Westmead.
SCH: Sydney Children's Hospital.
JHCH: John Hunter Children's Hospital.

(CSL Biotherapies, 0.5 mL), Fluvax® junior (CSL Biotherapies, 0.25 mL) or Influvac® (Solvay Biologicals, 0.5 mL).

The clinics were advertised by posters around the hospital, through presentations at medical team meetings, during grand rounds and by individual approaches from the nurse immuniser to parents of patients attending the hospital. At the Children's Hospital at Westmead, nurse immunisers from the Sydney West Area Health Service (SWAHS) public health unit were seconded for the duration of the influenza vaccine program.

As this was a new approach in NSW, participating hospitals and their associated public health unit met monthly by teleconference to implement, coordinate and manage the program. These groups have continued to meet during the 2011 influenza season to coordinate and promote influenza immunisation and monitor vaccine safety.

We evaluated clinic outcomes using the following indicators: the number of children and members of their families immunised; a comparison of current vaccine administration against previous attempts to increase influenza vaccine coverage at the paediatric hospitals.

### *National adverse events signal*

On Friday 23 April 2010, following the advice of the Chief Medical Officer of Australia, seasonal influenza vaccine administration to children aged 5 years and under was suspended due to an adverse events signal of possible increased fever reactions and febrile convulsions following influenza immunisation in young children. This significantly affected the influenza vaccine program in the three paediatric tertiary hospitals but clinic records provided a unique cohort of children to rapidly provide additional data to inform the national investigation.

From 27 April 2010, research nurses and clinic staff from the Children's Hospital at Westmead and the National Centre for Immunisation Research and Surveillance (NCIRS) used a scripted telephone interview to ask parents of children 6 months to 5 years old, who had received influenza vaccines in the three hospitals since the initiation of the program, about reactions following the receipt of the influenza vaccine. Parental reports were collated for each child who was vaccinated, and analysed by type of influenza vaccine received, age of the child, and the presence of chronic co-existing medical conditions (including gastroenterological, respiratory, oncological, cardiac and neurological conditions).

We therefore also evaluated the utility of the clinic in responding to and understanding a national adverse events signal.

## Results

The combined number of influenza-containing vaccines (monovalent H1N1 or seasonal trivalent) given to children presenting with medical conditions and their families at the three hospitals was 3458 vaccines (Table 1). Children with chronic medical conditions accounted for 36% ($n = 1251$) of vaccines administered.

The immunisation nurses reported that the offer to immunise family members to assist in the protection of their unwell child against influenza was popular and over 2000 family members were immunised.

The 2010 influenza program at the paediatric tertiary hospitals administered more influenza vaccinations to outpatients than in previous years (as measured by the influenza vaccines dispensed from the two hospital pharmacies that had provided ad hoc vaccination to paediatric outpatients previously) (Figure 1).

### *National adverse events signal*

Commencing on 27 April 2010, parents of children 6 months to 5 years old ($n = 333$), who had received influenza vaccines in the three hospitals since the initiation of the program, were contacted. Overall nearly 20% ($n = 66$) of parents reported that their child had a fever within 48 hours of vaccination. There was a statistically

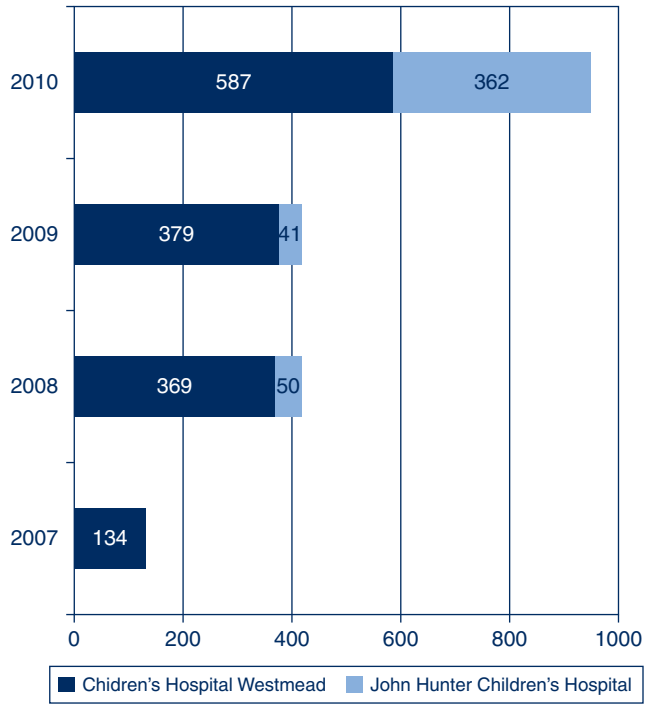


**Figure 1. Influenza vaccines dispensed at two NSW paediatric hospitals: comparison of program year 2010 with previous years, 2007–2009.**
**Source: Hospital pharmacy records at The Children's Hospital at Westmead and the John Hunter Children's Hospital.**

significant higher parental fever report for CSL Fluvax® compared with Influvac® (RR 6.5, 95% CI 3.1 to13.9, $p < 0.0001$), or CSL Panvax® (RR 2.9, 95% CI 1.8 to 4.3, $p \leq 0.0001$).[6] There were no reports of febrile convulsions in any children.

## Discussion

The dedicated paediatric hospital influenza vaccine program in 2010 immunised higher numbers of high medical at-risk children compared to previous years. Cocooning protection was offered through vaccination of family members. Having a database of the cohort of vaccinees allowed the hospitals to rapidly investigate an important safety alert and contribute to the national investigation of febrile convulsions following influenza vaccines in children in 2010.

The Royal Children's Hospital, Melbourne, Victoria has had a dedicated immunisation service for many years, delivering vaccinations including annual influenza vaccine, providing immunisation advice and managing adverse events. Parents and siblings of patients are able to purchase influenza vaccine from the hospital pharmacy. This service delivered approximately 2000 doses of influenza vaccine to patients, parents and siblings in 2009.[7] Additional benefits of these hospital-based programs include the opportunity for specialists to seek advice regarding immunisation for their patients with complex medical conditions, the development of common vaccination protocols for use in complex medical cases, the capacity to investigate safety concerns quickly, and a training opportunity for hospital nursing staff with vaccines which are usually administered in primary care settings.

As the Australian Immunisation Handbook notes: 'it is vital that healthcare professionals take every available opportunity to vaccinate children and adults'.[4] It is particularly important that children with chronic medical conditions who are most at risk of severe vaccine-preventable diseases are vaccinated in a timely way.

## Conclusion

Dedicated vaccination clinics at tertiary paediatric hospitals provide a valuable method of ensuring that children at high risk of severe influenza disease and its complications are vaccinated. The clinics can provide these vulnerable children with additional protection by vaccinating their family members (cocooning), while at the same time facilitate important clinical, surveillance and education activities.

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# Pertussis in NSW and its prevention in infants and children

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Pertussis is an acute respiratory tract illness caused by infection with the bacterium *Bordetella pertussis*. While pertussis affects all age groups, unimmunised and partially immunised babies are at greatest risk of severe disease and death.[1] Notification of cases of pertussis to the New South Wales (NSW) Department of Health is mandated under the NSW *Public Health Act 1991*.

## Epidemiology

In countries with universal childhood vaccination, epidemics of pertussis occur periodically on a background of endemic circulation, although levels of disease activity are much lower in comparison to those in unimmunised populations. In NSW and nationally, a large epidemic unfolded from 2008. In 2008–2009, the pertussis notification rate in NSW was about three times higher than the previous 5-year average. Disease activity has continued at high levels during 2010. The relatively higher rate is likely explained by both a real epidemic as well as improved diagnoses related to the wider use of molecular-based testing (polymerase chain reaction (PCR)), which is more sensitive than other available methods.[2]

Preventing the spread of *B. pertussis* is challenging due to a number of factors including: the organism is highly infectious; immunity following vaccination and/or previous infection wanes after a few years; and adolescents and adults commonly have mild to moderate illness following infection (which can often go undiagnosed) and can unknowingly transmit the pathogen to others including susceptible infants and children.[1,3]

## Vaccination schedule and coverage

It is recommended that children in Australia receive primary doses of a pertussis-containing vaccine (given in NSW in a hexavalent combination of DTPa-HepB-Hib-IPV) at 2, 4 and 6 months of age and that a booster dose (DTPa) is administered at 4 years of age. A second booster of adolescent/adult vaccine (dTpa) is recommended between the ages of 12 and 15 years, and is offered via NSW Health's School-Based Vaccination Program. The adolescent/adult dTpa vaccine is also recommended for adults who live and/or work with young children, including health professionals, new parents and those planning to have children. Pertussis vaccine coverage in NSW children under the age of 12 months is high (92.7%), although coverage and timeliness could be improved, particularly among Aboriginal children.[4]

## Prevention strategies: early infant pertussis

Pertussis infection during the first 6 months of life can be life threatening as these infants are too young to have received a full primary course of vaccine. There is therefore a need for public health action to focus on preventing pertussis in early infancy. Potential vaccine strategies currently being explored that may provide direct protection include neonatal immunisation (providing the first dose of vaccine straight after birth) and immunisation during pregnancy; studies are underway investigating the effectiveness and safety of these approaches. Indirect protection may be achieved through universal, population level pertussis immunisation programs and/or targeted vaccination of those likely to be in contact with newborns such as parents and grandparents (often termed 'cocooning').[3]

In 2009 the NSW Department of Health initiated a program to minimise the spread of the organism and protect susceptible infants and children. The program comprises a number of strategies, including: a large social marketing campaign promoting vaccination and other risk mitigation behaviours; promotion of the first vaccine dose at 6 weeks of age (rather than 8 weeks) and the first booster dose at 3½ years of age (rather than 4 years); and provision of a free booster dose of pertussis vaccine for new parents (including those planning pregnancy), grandparents and carers of babies under 12 months of age.

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 10.1071/NB11032

# Tickborne diseases

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Tickborne diseases are a worldwide public health problem. While Australia is free from many of the serious tickborne diseases endemic in North America and Europe, ticks are still a hazard to human health in Australia and measures to prevent tick bites should be undertaken.

## Tick ecology

There are over 800 species of tick worldwide; Australia is home to approximately 75 of these. Of Australian species, approximately 15 attack humans, the most important of these being *Ixodes holocyclus*, otherwise known as the 'paralysis tick'. This species is active year-round in Australia. The paralysis tick is particularly vulnerable to desiccation, thus it tends to be most active during periods of high humidity, such as after rainfall.

## Ticks as a vector for disease

Various tick species are competent vectors of human disease-causing pathogens including a variety of bacteria, viruses, rickettsia and protozoa. Ticks are capable of vertically transmitting several pathogens to their larval stage offspring which can then be passed to the nymph and thence to the adult. Thus an infective state can be maintained across multiple generations without contact with a vertebrate reservoir.[1] The vectorial capacity of ticks varies among species and specific pathogens are often transmitted only by a limited range of ticks.

## Australian tickborne diseases

There are three tickborne diseases in Australia: two spotted fever group rickettsiae (Queensland tick typhus and Flinders Island spotted fever) and Q fever. Although Q fever has been isolated from ticks, transmission to humans has not been definitively proven. A number of other micro-organisms (including the flavivirus Saumarez Reef virus) have been isolated from ticks in Australia but to date are not known to have caused any cases of human disease.

Queensland tick typhus, caused by *Rickettsia australis*, is found along Australia's eastern seaboard as far south as Victoria. The vector is the paralysis tick. Flinders Island spotted fever, caused by *R. honei*, is found in south-eastern Australia, including Tasmania, and is transmitted by the reptilian tick, *Bothriocroton hydrosauri*. Both infections cause mostly mild illness which is characterised predominantly by fever, rash, myalgia, headache and lymphadenopathy. Despite this, several cases of severe infection with Queensland tick typhus requiring admission to intensive care have been reported and death may occur if left untreated.[2] Both infections respond well to antibiotic therapy.

In addition to infectious diseases, tick bites can cause a range of adverse reactions, from localised pain and swelling to systemic allergic reactions including anaphylaxis or tick paralysis. Tick paralysis, caused by a toxin found in the saliva of *I. holocyclus*, is a frequent cause of paralysis and death in animals and although rare today, had been fatal in humans before the development of an antivenene.

## Prevention and control

Personal precautions can include avoiding tick infested areas, using repellents, wearing light coloured clothing and regularly checking for ticks. Environmental strategies such as fencing to exclude wildlife from areas inhabited by humans, modification of the habitat to decrease humidity through clearing of vegetation and mowing lawns and chemical control of ticks in the environment may also be used.[3]

## Conclusion

The global burden of tickborne disease is significant. In Australia however, tickborne diseases do not contribute greatly to the overall communicable disease burden, due to both low overall incidence and the relative mildness of locally endemic tickborne diseases. However, exposure to ticks does carry a risk and changes in the distribution of ticks and the epidemiology of tickborne diseases have been witnessed internationally. Changes in climate and human lifestyle will influence the presentation of tickborne disease.

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10.1071/NB11025

# Communicable Diseases Report, NSW, September and October 2011

***Communicable Diseases Branch***
***NSW Department of Health***

For updated information, including data and facts on specific diseases, visit www.health.nsw.gov.au and click on **Public Health** and then **Infectious Diseases.** The communicable diseases site is available at: http://www.health.nsw.gov.au/publichealth/infectious/index.asp.

Figure 1 and Tables 1 and 2 show notifications of communicable diseases received in September and October 2011 in New South Wales (NSW).

## Enteric infections

### *Outbreaks of foodborne disease*

Four outbreaks of gastrointestinal disease thought to be due to consumption of contaminated food were reported in September and October 2011. Three outbreaks were identified through complaints to the NSW Food Authority (NSWFA) and one outbreak was identified through reports to a public health unit. For one of these outbreaks, the causative organism was identified as *Campylobacter*; a pathogen was not identified in the other three outbreaks.

In one outbreak, identified through a complaint about a restaurant to the NSW Food Authority, six people from a group of 20 work colleagues developed diarrhoea 12 hours after eating lunch. The people who became ill ate a chicken curry dish with rice. NSW Food Authority investigated the premises and found that chicken curries were cooked daily, usually stored in a cool room and reheated later. The chicken curry served to this group was likely made two days previously. Bacterial toxins, such as *Clostridium perfringens* or *Bacillus cereus* can multiply during slow cooling processes. The restaurant was advised to immediately shorten the cooling step to reduce the opportunity for bacterial growth.

The second outbreak was identified through reports of gastrointestinal illness to a public health unit. Eighty-seven of approximately 500 people who attended a function developed vomiting and diarrhoea 24 hours after eating dinner. The function was catered by a commercial catering business. Just over half of the people (59%) who attended the dinner were surveyed. A poached prawn salad was found to be associated with illness (Odds Ratio = 6.3; Confidence Interval 3.2–13.1). The salad was assembled from pre-prepared products and involved no further cooking steps. As there was no food available to be sampled after the function, it was not possible to identify a specific ingredient as the source of illness, or the point of contamination. Good hygiene was reported by food handlers and the catering manager. No stool samples were submitted by any of the case-patients so the pathogen could not be confirmed in this outbreak, although the clinical picture suggests a viral pathogen.

In the third outbreak, a complaint was made to the NSW Food Authority after two friends became ill with diarrhoea, abdominal pain, nausea and headache, 9 to 10 hours after sharing a meal of Chinese food. Symptoms lasted for approximately 5 days. One case-patient submitted a stool sample that was positive for *Campylobacter*. The only other possible exposure was another shared breakfast meal, consumed 5 days before illness onset. NSW Food Authority reviewed the database for other complaints about both restaurants, but due to multiple shared exposures and lack of high-risk foods for *Campylobacter*, site visits were not conducted. The source and vehicle remain unknown, although an incubation period of 5 days and the reported symptoms are consistent with the campylobacteriosis diagnosis.

The fourth outbreak was identified through a complaint about a bakery made to the NSW Food Authority. A group of three people developed abdominal cramps, vomiting and diarrhoea 10 hours after eating Vietnamese pork rolls. This was the only common meal consumed by the three people. The NSW Food Authority inspected the premises and found issues such as the use of raw egg batter, the potential for cross contamination from raw chicken stored in the fridge, and inadequate cleaning and sanitising of food equipment and contact surfaces. Food samples and environmental swabs were taken but no pathogen was identified. However, it was considered that the poor adherence to food safety standards could have led to contamination from raw foods, causing illness. The Food Authority issued an improvement notice the next day in respect to all the defects. Re-inspection of the business premises confirmed that all cleaning, hygiene and food safety defects had been corrected in accordance with the directions of the improvement notice.

 10.1071/NB11049

### *Outbreaks of gastroenteritis in institutional settings*

In September and October 2011, 65 outbreaks of gastroenteritis in institutions were reported, affecting 898 people. Twenty-nine outbreaks occurred in aged-care facilities, 17 in child-care centres, 17 in hospitals and 2 in other facilities. All these outbreaks appear to have been caused by person-to-person spread of a viral illness. In 42 (65%) outbreaks, one or more stool specimens were collected. In 18 (43%) of these, norovirus was detected. Rotavirus was detected in six (14%) outbreaks (norovirus was also identified in one of these outbreaks). *Clostridium difficile* was detected in three outbreaks along with norovirus; this finding was thought to be coincidental during a viral gastroenteritis outbreak. In 13 outbreaks, all stool specimens were negative for pathogens. Results for six outbreaks are still outstanding. Stool specimens for laboratory testing were not available for the remaining 23 (35%) outbreaks.

Viral gastroenteritis increases in winter months. Public health units encourage institutions to submit stool specimens from cases for testing during an outbreak to help determine the cause of the outbreak (for further information see *Control guidelines for gastrointestinal outbreaks in institutions*).

## Bloodborne virus

### *HIV infection*

The 2010 annual report on HIV infections in NSW has been published on the NSW Health website (http://www.health.nsw.gov.au/resources/publichealth/infectious/diseases/pdf/new_hiv_2010_summary.pdf). There were 307 people newly diagnosed with HIV infections notified in NSW, which was similar to the number notified in 2009 ($n = 329$). In 2010, 357 HIV-positive tests were reported from reference laboratories for people that had not been previously diagnosed with HIV infection in NSW. Of these, 50 (14%) were found to have been previously diagnosed with HIV infection elsewhere, or they were not NSW residents. The remaining 307 were NSW residents newly diagnosed with HIV. A total of 282 (92%) were male and the median age at diagnosis was 38 years. Most cases were reported to be homosexually acquired (234; 76%), and the remaining acquisition was identified as heterosexual (50; 16%), injecting drug use (9; 3%), mother-to-child transmission (1; 0.3%) and other or unknown sources (13; 4%). Promoting safer sex practices among males with homosexual exposures and regular testing in this group remain important.

## Respiratory infections

### *Influenza*

Influenza activity in NSW, as measured by the number of people who presented to 56 select Emergency Departments with influenza-like illness, and the number of patients who tested positive for influenza at diagnostic laboratories was moderate during September and October 2011. The rate of laboratory-confirmed influenza activity has been declining steadily since activity peaked in mid July 2011.

There were 204 presentations (rate 1.4 per 1000 presentations) for September, and 122 presentations (rate 0.8 per 1000 presentations) for October to select Emergency Departments with influenza-like illness. There were 332 (7%) cases of laboratory-confirmed influenza reported in September; including 143 (43%) influenza A (negative for H1N1 2009) presumed to be influenza A (H3), 150 (45%) influenza B, and the remaining 38 (12%) influenza A H1N1 2009. There were 90 (3%) cases of laboratory-confirmed influenza reported in October, including 46 (51%) influenza A (negative for H1N1 2009) presumed to be influenza A (H3)), 26 (29%) influenza B, and the remaining 18 (20%) influenza A H1N1 2009.

For a more detailed report on respiratory activity in NSW see: http://www.health.nsw.gov.au/PublicHealth/Infectious/influenza_reports.asp.

## Vaccine-preventable diseases

### *Meningococcal disease*

Fourteen confirmed cases of meningococcal disease were notified in September and October 2011. Of these, nine cases were due to serogroup B, one case was serogroup C, one case was serogroup Y and the serogroup was unknown for the remaining three cases.

A free vaccine for serogroup C meningococcal disease is available for infants at 12 months of age.[1] Consequently, serogroup C disease is now seen mainly in adults and in unimmunised children. In NSW this year, 81% of cases of meningococcal disease where the serogroup was known were caused by serogroup B, for which there is no vaccine. One case of serogroup C disease in an adult was notified this year.

### *Measles*

Seven cases of measles were notified in NSW in September and October 2011. Two case-patients were aged between 0–4 years and the remainder were young adults aged between 15–39 years. One case-patient was reported to have been vaccinated.

Many Australian-born people who are now aged between 20 and 40 years may not have received any, let alone two, doses of measles vaccination required for best protection, and do not have immunity from past infection. Measles mumps and rubella (MMR) vaccine is now routinely given at 12 months of age and again at 4 years (although it can be given from 3½ years of age); two MMR vaccines give long-lasting immunity.

An accurate immunisation history is often difficult to determine in adults who may be unsure of the exact details of the vaccines they received in childhood and do not have written records. There is no whole-of-life immunisation register that can be used to verify the vaccines adults have been given. General practitioners (GPs) are able to provide free MMR vaccine to anyone born after 1966 who has not received two doses of vaccine or who is unsure of his or her vaccination history.

### *Pertussis (whooping cough)*

In September and October 2011, 2400 cases of pertussis were notified in NSW compared to 2443 cases for the same period in 2010. In total, 11 033 cases of pertussis have been reported up to 31 October 2011 compared to 5845 for the same period in 2010. To date in 2011, the highest number of notifications was reported in children aged between 5–9 years (3365 cases), 0–4 years (2030 cases) and young people aged between 10–14 years (1900 cases).

A free vaccine is recommended for infants at 2, 4 and 6 months although the first dose can be given as early as 6 weeks of age. A booster dose is recommended at 4 years but this can be given as early as 3½ years of age.[2]

Immunisation reduces the risk of infection, however the vaccine does not provide lifelong protection, and re-infection can occur.[1] Because pertussis immunity wanes over time, many older children and adults are susceptible to infection and can be the source of new infections in infants.[3] For a limited time, NSW Health is providing free pertussis (dTpa) vaccine through GPs to all new parents, grandparents and any other adults who will regularly care for infants less than 12 months of age. Free vaccine boosters are also provided to Year 7 and Year 10 students as part of NSW Health's School-based Vaccination Program.

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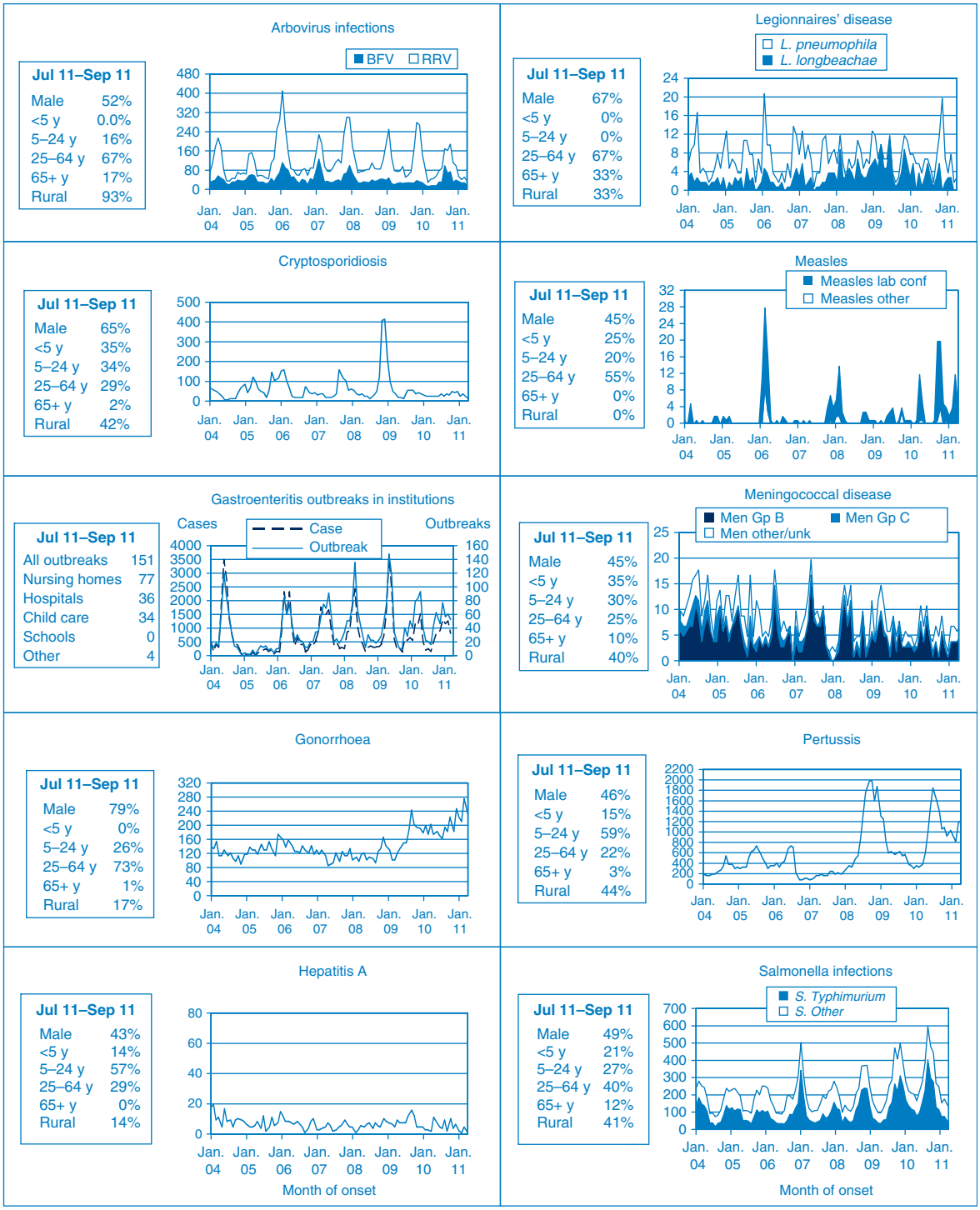
**Figure 1. Notifications of selected communicable diseases, NSW, January 2004 to October 2011, by month of onset.**

Preliminary data: case counts in recent months may increase because of reporting delays.
Laboratory-confirmed cases only, except for measles, meningococcal disease and pertussis.
BFV, Barmah Forest virus infections; RRV, Ross River virus infections; lab conf, laboratory confirmed.
Men Gp C and Gp B, meningococcal disease due to serogroup C and serogroup B infection; other/unk, other or unknown serogroups.
NB: Multiple series in graphs are stacked, except gastroenteritis outbreaks.
NB: Outbreaks are more likely to be reported by nursing homes & hospitals than by other institutions.

| NSW population | |
|---|---|
| Male | 50% |
| <5 y | 7% |
| 5–24 y | 27% |
| 25–64 y | 53% |
| 65+ y | 13% |
| Rural | 46% |



## Table 1. Notifications of scheduled medical conditions received in September 2011 by Local Health District in NSW

| Condition | Local Health District (2011) | | | | | | | | | | | | | | | | Total | |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| | Murrumbidgee | Southern NSW | Western NSW | Far West | Hunter New England | Northern NSW | Mid North Coast | Central Coast | Northern Sydney | South Eastern Sydney | Illawarra Shoalhaven | Sydney | South Western Sydney | Western Sydney | Nepean Blue Mountains | Justice Health | For Sep[b] | Year to date[b] |
| **Bloodborne and sexually transmitted** | | | | | | | | | | | | | | | | | | |
| Chancroid[a] | – | – | – | – | – | – | – | – | – | – | – | – | – | – | – | – | – | – |
| Chlamydia (genital)[a] | 51 | 41 | 52 | 11 | 273 | 60 | 44 | 78 | 129 | 323 | 89 | 196 | 164 | 139 | 70 | 6 | 1730 | 15 382 |
| Gonorrhoea[a] | 2 | 5 | 5 | – | 23 | 5 | 4 | 2 | 15 | 71 | 3 | 52 | 24 | 26 | 9 | 1 | 248 | 1985 |
| Hepatitis B – acute viral[a] | – | – | – | – | 1 | – | – | – | – | – | – | 1 | – | – | – | – | 2 | 24 |
| Hepatitis B – other[a] | 2 | – | 9 | 2 | 3 | 2 | 1 | – | 26 | 29 | 1 | 38 | 33 | 51 | 5 | 2 | 204 | 1972 |
| Hepatitis C – acute viral[a] | – | – | – | 1 | 2 | 2 | – | – | – | – | – | – | – | – | – | – | 5 | 37 |
| Hepatitis C – other[a] | 19 | 8 | 19 | 2 | 29 | 16 | 15 | 9 | 12 | 18 | 30 | 24 | 36 | 26 | 10 | 4 | 278 | 2543 |
| Hepatitis D – unspecified[a] | – | – | – | – | – | – | – | – | – | – | – | – | 1 | – | – | – | 1 | 5 |
| Lymphogranuloma vanereum | – | – | – | – | – | – | – | – | 2 | – | – | – | – | – | 1 | – | 3 | 32 |
| Syphilis | 1 | 2 | 5 | – | – | – | 1 | 1 | 3 | 16 | 2 | 12 | 8 | 7 | 2 | – | 60 | 535 |
| **Vectorborne** | | | | | | | | | | | | | | | | | | |
| Barmah Forest virus[a] | 2 | 1 | – | – | 7 | 7 | 4 | – | – | – | 1 | – | – | – | – | – | 22 | 407 |
| Ross River virus[a] | 2 | – | 3 | – | 7 | 4 | 3 | – | – | – | 1 | – | – | – | – | – | 20 | 516 |
| Arboviral infection (other)[a] | 1 | – | – | – | 1 | – | – | 1 | 1 | – | 2 | 1 | – | 1 | – | – | 8 | 95 |
| Malaria[a] | 1 | – | – | – | – | – | – | – | – | – | – | 1 | 1 | 3 | – | – | 6 | 61 |
| **Zoonoses** | | | | | | | | | | | | | | | | | | |
| Anthrax[a] | – | – | – | – | – | – | – | – | – | – | – | – | – | – | – | – | – | – |
| Brucellosis[a] | – | – | – | – | – | – | – | – | – | – | – | – | – | – | – | – | – | 4 |
| Leptospirosis[a] | 1 | – | – | – | – | – | – | – | – | – | – | – | – | – | – | – | 1 | 31 |
| Lyssavirus[a] | – | – | – | – | – | – | – | – | – | – | – | – | – | – | – | – | – | – |
| Psittacosis[a] | – | – | – | – | – | – | – | – | – | – | – | – | – | – | 1 | – | 1 | 12 |
| Q fever[a] | – | – | – | – | 2 | 1 | 1 | – | 1 | – | – | 1 | 2 | – | – | – | 8 | 82 |
| **Respiratory and other** | | | | | | | | | | | | | | | | | | |
| Blood lead level[a] | 7 | – | 8 | 1 | 2 | – | – | – | – | 2 | – | 2 | – | 1 | 2 | – | 25 | 223 |
| Influenza[a] | 41 | 16 | 40 | 5 | 138 | 54 | 16 | 21 | 70 | 94 | 25 | 41 | 63 | 147 | 62 | – | 833 | 5343 |
| Invasive pneumococcal infection[a] | 4 | 5 | 6 | – | 10 | 1 | 1 | 3 | 8 | 8 | 5 | 4 | 5 | 9 | 3 | – | 72 | 415 |
| *Legionella longbeachae* infection[a] | – | – | – | – | – | – | – | – | – | – | – | – | – | – | – | – | – | 22 |
| *Legionella pneumophila* infection[a] | – | – | – | – | – | – | – | – | – | – | – | – | – | 1 | – | – | 1 | 46 |
| Legionnaires' disease (other)[a] | – | – | – | – | – | – | – | – | – | – | – | – | – | – | – | – | – | 7 |
| Leprosy | – | – | – | – | – | – | – | – | – | – | – | – | – | – | – | – | – | 1 |
| Meningococcal infection (invasive)[a] | – | – | 1 | – | 1 | – | – | 1 | – | 1 | 1 | – | – | – | – | – | 5 | 52 |
| Tuberculosis | 1 | 1 | 1 | – | 2 | – | – | 2 | 2 | 9 | 2 | 1 | 1 | 5 | – | – | 27 | 257 |
| **Vaccine–preventable** | | | | | | | | | | | | | | | | | | |
| Adverse event after immunisation | 1 | 2 | – | – | 1 | – | – | – | 3 | – | 1 | – | – | 3 | – | – | 11 | 166 |
| H. *Influenzae b* infection (invasive)[a] | – | – | – | – | – | – | – | – | – | – | – | – | – | – | – | – | – | 4 |
| Measles | – | – | – | – | – | – | – | – | – | 1 | – | 2 | 3 | – | – | – | 6 | 71 |
| Mumps[a] | – | – | – | – | – | 2 | – | – | – | 1 | – | – | 1 | 1 | – | – | 5 | 39 |
| Pertussis | 148 | 29 | 70 | 3 | 64 | 51 | 62 | 49 | 138 | 120 | 81 | 42 | 110 | 202 | 132 | – | 1303 | 9936 |
| Rubella[a] | – | – | – | – | – | – | – | – | – | 1 | – | – | – | – | – | – | 1 | 15 |
| Tetanus | – | – | – | – | – | – | – | – | – | – | – | – | – | – | – | – | – | 1 |
| **Enteric** | | | | | | | | | | | | | | | | | | |
| Botulism | – | – | – | – | – | – | – | – | – | – | – | – | – | – | – | – | – | – |
| Cholera[a] | – | – | – | – | – | – | – | – | – | – | – | – | – | – | – | – | – | – |
| Cryptosporidiosis[a] | 1 | 1 | – | – | – | 1 | 1 | – | 1 | 2 | 3 | 1 | – | 2 | – | – | 13 | 280 |
| Giardiasis[a] | 2 | 4 | 8 | – | 20 | – | 2 | 5 | 22 | 33 | 10 | 11 | 9 | 16 | 4 | – | 146 | 1898 |
| Haemolytic uraemic syndrome | – | – | – | – | – | – | – | – | – | – | – | – | – | – | – | – | – | 4 |
| Hepatitis A[a] | 1 | – | – | – | – | – | – | – | – | 1 | – | 1 | – | – | – | – | 3 | 44 |
| Hepatitis E[a] | – | – | – | – | – | – | – | – | – | – | – | – | – | – | – | – | – | 16 |
| Listeriosis[a] | – | – | – | – | – | – | – | – | – | 1 | – | – | – | – | – | – | 1 | 15 |
| Rotavirus[a] | – | 1 | 2 | – | 6 | 4 | 1 | 2 | 36 | 21 | 2 | 9 | 6 | 15 | 4 | – | 109 | 623 |
| Salmnellosis[a] | 8 | 3 | 7 | 1 | 16 | 6 | 6 | 9 | 20 | 12 | 4 | 16 | 13 | 10 | 6 | – | 137 | 2855 |
| Shigellosis[a] | – | – | – | – | – | – | 1 | – | – | – | 1 | 2 | – | 1 | 1 | – | 6 | 92 |
| Typhoid[a] | – | – | – | – | – | – | – | – | – | – | – | 1 | – | – | – | – | 1 | 43 |
| Verotoxin producing *E. coli*[a] | – | – | – | – | – | 1 | – | – | – | – | – | – | 1 | – | – | – | 2 | 6 |
| **Miscellaneous** | | | | | | | | | | | | | | | | | | |
| Creutzfeldt–Jakob disease | – | – | – | – | – | – | – | – | – | – | 1 | – | – | – | – | – | 1 | 7 |
| Meningococcal conjunctivitis | – | – | – | – | – | – | – | – | – | – | – | – | – | – | – | – | – | 1 |

[a]Laboratory-confirmed cases only. [b]Includes cases with unknown postcode.
NB: Data are current and accurate as at the preparation date. The number of cases reported is, however, subject to change, as cases may be entered at a later date or retracted upon further investigation. Historical data configurations are included for continuity/comparison purposes and to highlight regional differences.
NB: HIV and AIDS data are reported separately in the Public Health Bulletin quarterly.
Data are reported as of public health unit office.

## Table 2. Notifications of scheduled medical conditions received in October 2011 by Local Health District in NSW

| Condition | Local Health District (2011) | | | | | | | | | | | | | | | | Total | |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| | Murrumbidgee | Southern NSW | Western NSW | Far West | Hunter New England | Northern NSW | Mid North Coast | Central Coast | Northern Sydney | South Eastern Sydney | Illawarra Shoalhaven | Sydney | South Western Sydney | Western Sydney | Nepean Blue Mountains | Justice Health | For Oct[b] | Year to date[b] |
| **Bloodborne and sexually transmitted** | | | | | | | | | | | | | | | | | | |
| Chancroid[a] | – | – | – | – | – | – | – | – | – | – | – | – | – | – | – | – | – | – |
| Chlamydia (genital)[a] | 67 | 31 | 63 | 11 | 214 | 92 | 37 | 70 | 134 | 195 | 91 | 157 | 134 | 139 | 49 | 8 | 1498 | 16 880 |
| Gonorrhoea[a] | 1 | 1 | – | 2 | 17 | 8 | 2 | 3 | 17 | 68 | 4 | 58 | 28 | 31 | 8 | – | 249 | 2234 |
| Hepatitis B – acute viral[a] | 1 | – | – | – | 1 | – | – | – | – | – | – | – | – | – | – | 1 | 3 | 27 |
| Hepatitis B – other[a] | 3 | 1 | 3 | – | 6 | 1 | 2 | 5 | 23 | 27 | 3 | 45 | 47 | 35 | 4 | – | 205 | 2177 |
| Hepatitis C – acute viral[a] | – | – | 1 | 1 | – | – | – | – | – | – | – | – | – | – | – | – | 2 | 39 |
| Hepatitis C – other[a] | 15 | 11 | 11 | 4 | 24 | 22 | 4 | 26 | 11 | 25 | 5 | 27 | 33 | 23 | 6 | – | 247 | 2790 |
| Hepatitis D – unspecified[a] | – | – | – | – | – | – | – | – | – | – | – | – | – | – | – | – | – | 5 |
| Lymphogranuloma vanereum | – | – | – | – | – | – | – | – | – | – | – | – | – | – | – | – | – | 32 |
| Syphilis | – | – | 1 | – | 2 | – | – | 2 | 2 | 3 | 1 | 3 | – | – | – | – | 14 | 549 |
| **Vectorborne** | | | | | | | | | | | | | | | | | | |
| Barmah Forest virus[a] | 1 | 1 | – | – | 3 | 8 | 2 | 3 | 1 | – | – | – | – | – | – | – | 19 | 426 |
| Ross River virus[a] | 3 | – | 3 | – | 10 | 2 | 2 | – | – | – | – | 1 | – | – | – | – | 21 | 537 |
| Arboviral infection (other)[a] | – | – | – | – | – | 2 | – | – | 2 | – | – | – | 1 | 2 | – | – | 7 | 102 |
| Malaria[a] | – | – | – | – | 1 | – | 1 | 1 | – | – | – | – | 1 | 1 | 1 | – | 6 | 67 |
| **Zoonoses** | | | | | | | | | | | | | | | | | | |
| Anthrax[a] | – | – | – | – | – | – | – | – | – | – | – | – | – | – | – | – | – | – |
| Brucellosis[a] | – | – | – | – | – | – | – | – | – | – | – | – | – | – | – | – | – | 4 |
| Leptospirosis[a] | – | – | 1 | – | – | – | – | – | – | 1 | – | – | – | – | – | – | 2 | 33 |
| Lyssavirus[a] | – | – | – | – | – | – | – | – | – | – | – | – | – | – | – | – | – | – |
| Psittacosis[a] | – | – | – | – | 1 | – | – | – | – | – | – | – | – | – | 1 | – | 2 | 14 |
| Q fever[a] | 1 | 2 | – | – | 3 | 3 | 2 | – | – | – | – | – | – | – | – | – | 11 | 93 |
| **Respiratory and other** | | | | | | | | | | | | | | | | | | |
| Blood lead level[a] | 1 | – | 3 | – | – | – | – | – | – | – | – | – | – | – | 1 | – | 5 | 228 |
| Influenza[a] | 24 | 9 | 28 | 2 | 38 | 12 | 15 | 6 | 33 | 48 | 7 | 10 | 19 | 54 | 25 | – | 330 | 5673 |
| Invasive pneumococcal infection[a] | 2 | 3 | 4 | – | 10 | 1 | 1 | 1 | 3 | 2 | 1 | 6 | 5 | 2 | 2 | – | 43 | 458 |
| *Legionella longbeachae* infection[a] | – | – | 1 | – | – | – | – | – | – | – | – | – | – | – | – | – | 1 | 23 |
| *Legionella pneumophila* infection[a] | – | – | 1 | – | – | – | – | – | – | – | – | – | – | 2 | – | – | 3 | 49 |
| Legionnaires' disease (other)[a] | – | – | – | – | – | – | – | – | – | – | – | – | – | 1 | – | – | 1 | 8 |
| Leprosy | – | – | – | – | – | – | – | – | – | – | – | – | – | – | – | – | – | 1 |
| Meningococcal infection (invasive)[a] | – | – | – | 1 | 3 | – | – | – | – | – | 2 | 1 | – | 2 | – | – | 9 | 61 |
| Tuberculosis | 3 | 2 | – | – | 1 | – | – | – | 1 | 7 | 1 | 1 | 1 | – | – | – | 17 | 274 |
| **Vaccine–preventable** | | | | | | | | | | | | | | | | | | |
| Adverse event after immunisation | 1 | 2 | – | – | 1 | – | – | – | 5 | – | 1 | – | – | 1 | – | – | 11 | 177 |
| H. *Influenzae b* infection (invasive)[a] | – | – | – | – | – | – | – | – | – | – | – | – | – | – | – | – | – | 4 |
| Measles | – | – | – | – | – | – | – | – | – | – | – | – | 1 | – | – | – | 1 | 72 |
| Mumps[a] | – | – | – | – | – | – | – | – | 1 | 2 | – | – | – | – | – | – | 3 | 42 |
| Pertussis | 101 | 37 | 84 | 1 | 46 | 52 | 30 | 36 | 134 | 114 | 53 | 32 | 117 | 170 | 89 | – | 1097 | 11 033 |
| Rubella[a] | – | – | – | – | – | – | – | – | – | – | – | – | – | – | – | – | – | 15 |
| Tetanus | – | – | – | – | – | – | – | – | – | – | – | – | – | – | – | – | – | 1 |
| **Enteric** | | | | | | | | | | | | | | | | | | |
| Botulism | – | – | 1 | – | – | – | – | – | – | – | – | – | 1 | – | – | – | 2 | 2 |
| Cholera[a] | – | – | – | – | – | – | – | – | – | – | – | – | – | – | – | – | – | – |
| Cryptosporidiosis[a] | 1 | 1 | 2 | – | 2 | – | 1 | – | 1 | 4 | – | 3 | 2 | 4 | – | – | 21 | 301 |
| Giardiasis[a] | 6 | 1 | 8 | 1 | 16 | 1 | 3 | 5 | 26 | 34 | 8 | 23 | 8 | 5 | 7 | 1 | 153 | 2051 |
| Haemolytic uraemic syndrome | – | – | – | – | – | – | – | – | – | – | – | – | – | – | – | – | – | 4 |
| Hepatitis A[a] | – | – | – | – | – | – | – | – | – | 1 | – | – | 1 | – | 1 | – | 3 | 47 |
| Hepatitis E[a] | – | – | – | – | – | – | – | – | – | – | – | – | 1 | 1 | – | – | 2 | 18 |
| Listeriosis[a] | – | – | – | – | – | – | – | – | – | – | – | – | – | – | 1 | – | 1 | 16 |
| Rotavirus[a] | 3 | 18 | 34 | 1 | 17 | 6 | 2 | 6 | 42 | 16 | 3 | 12 | 9 | 111 | 55 | – | 335 | 958 |
| Salmnellosis[a] | 8 | 2 | 5 | – | 22 | 14 | 9 | 3 | 25 | 25 | 7 | 16 | 17 | 14 | 4 | – | 171 | 3026 |
| Shigellosis[a] | – | – | – | – | 1 | – | – | – | – | 5 | – | – | 1 | 1 | – | – | 8 | 100 |
| Typhoid[a] | – | – | – | – | – | – | – | – | – | – | – | – | – | – | – | – | – | 43 |
| Verotoxin producing *E. coli*[a] | 1 | – | – | – | – | – | – | – | – | – | 1 | – | – | – | – | – | 2 | 8 |
| **Miscellaneous** | | | | | | | | | | | | | | | | | | |
| Creutzfeldt–Jakob disease | – | – | 1 | – | – | – | – | – | – | – | – | – | 1 | – | – | – | 2 | 9 |
| Meningococcal conjunctivitis | – | – | – | – | – | – | – | – | – | – | – | – | – | – | – | – | – | 1 |

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NSW GOVERNMENT Health

# Contents

## Immunisation in NSW



# NSW PUBLIC HEALTH BULLETIN


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