Australian Government
Department of Health

Communicable Diseases Intelligence

Volume 37 | Supplement | December 2013

Vaccine preventable diseases and vaccination coverage in Aboriginal and Torres Strait Islander people, Australia 2006–2010

National Centre for Immunisation Research and Surveillance of Vaccine Preventable Diseases

ISSN 1445-4866 Online

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http://www.health.gov.au/cdi

Subscriptions and contacts

Communicable Diseases Intelligence is produced every quarter by:
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This journal is indexed by *Index Medicus* and Medline

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Vaccine preventable diseases and vaccination coverage in Aboriginal and Torres Strait Islander people, Australia 2006–2010

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Communicable Diseases Intelligence
Australian Government Department of Health

NCIRS is a collaborating unit of the
Australian Institute of Health and Welfare

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Acknowledgements

NCIRS wishes to acknowledge the following organisations and individuals for their assistance and contribution to this report.

Provision of data

The Vaccine Preventable Disease Surveillance Section, Office of Health Protection, Australian Government Department of Health for data from the National Notifiable Diseases Surveillance System (NNDSS).

The Australian Institute of Health and Welfare (AIHW), Hospitals Unit, Economics and Health Services Group, for data from the National Hospital Morbidity Database.

The Department of Human Services, for vaccination coverage data from the Australian Childhood Immunisation Register.

Australian Bureau of Statistics (ABS) for population data and mortality data.

Department of Health for analysed data on pandemic influenza 2009.

Ms Polly Wallace for providing her MPhil thesis chapter on pandemic influenza.

External reviewers of disease chapters

Associate Professor Ross Andrews (Menzies School of Health Research)

Dr Liz Moore (National Aboriginal Community Controlled Health Organisation)

Manuscript editing and librarianship support

Ms Donna Armstrong (NCIRS)

Ms Catherine King (NCIRS)

Mr Edward Jacyna (NCIRS)

NCIRS is supported by the Australian Government Department of Health, the NSW Department of Health, The Children's Hospital at Westmead, and the University of Sydney.

Executive summary

This report outlines the major positive impacts of vaccines on the health of Aboriginal and Torres Strait Islander people from 2007 to 2010, as well as highlighting areas that require further attention.

Hepatitis A disease is now less common in Aboriginal and Torres Strait Islander children than in their non-Indigenous counterparts. Hepatitis A vaccination for Aboriginal and Torres Strait Islander children was introduced in 2005 in the high incidence jurisdictions of the Northern Territory, Queensland, South Australia and Western Australia. In 2002–2005, there were 20 hospitalisations for hepatitis A in Aboriginal and Torres Strait Islander children aged <5 years – over 100 times more common than in other children – compared to none in 2006/07–2009/10.

With respect to invasive pneumococcal disease (IPD), there has been a reduction of 87% in notifications of IPD caused by serotypes contained in 7-valent pneumococcal conjugate vaccine (7vPCV) since the introduction of the childhood 7vPCV program among Aboriginal and Torres Strait Islander children. However, due to a lower proportion of IPD caused by 7vPCV types prior to vaccine introduction, the decline in total IPD notifications has been less marked in Aboriginal and Torres Strait Islander children than in other children. Higher valency vaccines (10vPCV and 13vPCV) which replaced 7vPCV from 2011 are likely to result in a greater impact on IPD and potentially also non-invasive disease, although disease caused by non-vaccine serotypes appears likely to be an ongoing problem. Among Aboriginal and Torres Strait Islander people aged ≥50 years, there have been recent increases in IPD, which appear related to low vaccination coverage and highlight the need for improved coverage in this high-risk target group.

Since routine meningococcal C vaccination for infants and the high-school catch-up program were implemented in 2003, there has been a significant decrease in cases caused by serogroup C. However, the predominant serogroup responsible for disease remains serogroup B, and Aboriginal and Torres Strait Islander children have significantly higher incidence of serogroup B disease than other children. A vaccine against meningococcus type B has now been licensed in Australia.

The decline in severe rotavirus disease after vaccine introduction in 2007 was less marked in Aboriginal and Torres Strait Islander children than in other children. By far the highest hospitalisation rates continue to occur among Aboriginal and Torres Strait Islander children in the Northern Territory. Consideration of the role of age cut-offs and 2-dose versus 3-dose schedules may be necessary. Genotype surveillance is critically important to allow detection of any possible emergence of genotypes for which there is lower vaccine-derived immunity.

Although *Haemophilus influenzae* type b disease rates have decreased significantly since the introduction of vaccines in 1993, the plateauing of rates in Aboriginal and Torres Strait Islander children, and increasing disparity with other children, are concerning. While it is possible that higher disease rates in young infants could be associated with the later age of protection from the newer 4-dose schedule, it is also possible that higher vaccine immunogenicity will result in reduced carriage. Close monitoring is important to detect any re-emergence of Hib disease as soon as possible.

Pandemic and seasonal influenza and pneumonia are other diseases with comparatively higher rates in Aboriginal and Torres Strait Islander people. For Aboriginal and Torres Strait Islander people aged ≥50 years, it is unclear whether or not there has been a decline in influenza hospitalisations since the start of the National Indigenous Pneumococcal and Influenza Immunisation Program in 1999, but hospitalisation rates are still higher in Aboriginal and Torres Strait Islander people. Achieving high coverage in those aged ≥15 years should now be a priority.

A prolonged mumps outbreak occurred in 2007/2008 predominantly affecting Aboriginal and Torres Strait Islander adolescents and young adults in north-western Australia. A potential contributor to this mumps outbreak was greater waning of immunity after receipt of the first dose of mumps-containing vaccine at 9, rather than 12, months of age in the Northern Territory in the 1980s and 1990s. However, outbreaks in Australia and overseas have subsided without additional boosters being routinely implemented.

Pertussis epidemics continue to occur in Australia and affect both Aboriginal and Torres Strait Islander and other people. Parents are now encouraged to have their infant's first vaccination given at 6 weeks of age, instead of the usual 2 months, and this is being successfully implemented for Aboriginal and Torres Strait Islander and other infants. Timely provision of the 4- and 6-month doses remains very important.

High coverage for standard vaccines, poor timeliness of vaccination and lower coverage for 'Indigenous only' vaccines are continuing features of vaccination programs for Aboriginal and Torres Strait Islander people. There have been some improvements in vaccination timeliness in recent years for all children, but disparities remain between Aboriginal and Torres Strait Islander and other children. Poor timeliness reduces the potential benefits of vaccination, most importantly for pneumococcal, Hib and rotavirus vaccines in infants. The age cut-offs for rotavirus vaccines present a particular challenge for timely vaccination, limiting the capacity for catching up on late vaccination and resulting in lower overall coverage. This is more pronounced for the 3-dose than for the 2-dose rotavirus schedule.

Coverage for vaccines recommended only for Aboriginal and Torres Strait Islander children continues to remain substantially lower than that for universal vaccines. This underlines the importance of immunisation providers establishing the Indigenous status of their clients, so that additional vaccines are offered as appropriate.

The absence of any coverage data for Aboriginal and Torres Strait Islander adolescents, or for adults since 2004/2005, is a substantial obstacle to implementing and improving programs in these age groups.

1. Introduction

This is the third report on vaccine preventable diseases and vaccination coverage in Aboriginal and Torres Strait Islander people. The first (1999–2002) was published in 2004, and the second (2003–2006) was published in 2008.

Documented improvements in the quality of data on Indigenous status noted in previous publications and further documented in this report have enabled this report to be substantially more comprehensive than previous reports. This report includes notifiable disease data from all eight jurisdictions, up from five in the previous report. Hospitalisation data is presented for six jurisdictions and death data for five jurisdictions. National vaccination coverage data was obtained from the Australian Childhood Immunisation Register.

This report is modelled on two other regularly published national reports – *Vaccine preventable diseases and vaccination coverage in Australia* (produced by the National Centre for Immunisation Research and Surveillance (NCIRS)) and The health and welfare of Australia's Aboriginal and Torres Strait Islander peoples (produced by the Australian Institute of Health and Welfare (AIHW) and the Australian Bureau of Statistics (ABS)). It provides a comparison of disease burden and vaccination coverage in Aboriginal and Torres Strait Islander and non-Indigenous people not available in the Vaccine preventable diseases and vaccination coverage reports, and provides detailed data on vaccine preventable diseases and vaccination coverage not available in The health and welfare of Australia's Aboriginal and Torres Strait Islander peoples reports.

Individual chapters are provided for those diseases responsible for a substantial burden of illness in Aboriginal and Torres Strait Islander people, and rare diseases are combined in a single chapter. Data are provided for all diseases and vaccines included in the National Immunisation Program (NIP) for the period of analysis with the exception of human papillomavirus (HPV); data on the impact of HPV are not available from the sources used in this report for other vaccine preventable diseases. The impact of the HPV program has been reported elsewhere,[1] but so far there are no data available on vaccination coverage or vaccine impact in Aboriginal and Torres Strait Islander people. Tuberculosis has not been included in this or other reports in this series, as the control of this disease in Australia is based on diagnosis and treatment rather than vaccination. Data on tuberculosis, including in Aboriginal and Torres Strait Islander people, are published elsewhere.[2]

The aim of this report is to present recent data from routinely collected sources, along with informed commentary, to facilitate service delivery, policy development and further research on the prevention of vaccine preventable diseases in Aboriginal and Torres Strait Islander people. The primary audience of this report is health professionals.

2. Vaccine preventable diseases

2.1 *Haemophilus influenzae* type b disease

Haemophilus influenzae is a Gram-negative coccobacillus. Serotype b of Hib is a leading cause of serious disease, particularly in childhood. It can cause diseases of the respiratory tract including otitis media, sinusitis and bronchitis. Serious manifestations of Hib disease include meningitis, bacteraemia, epiglottitis, septic arthritis, pericarditis, osteomyelitis, soft tissue abscesses and cellulitis, and death. Other diseases, such as epididymitis, endocarditis, peritonitis and tracheitis, have also been associated with Hib.[3]

Relevant vaccine history

1993

- Hib vaccine recommended in childhood vaccination schedule for all children. PRP-OMP*-containing vaccines, providing protection at an earlier age than other vaccines, used for Aboriginal and Torres Strait Islander children.

2005

- PRP-OMP* used in the Northern Territory, Queensland, South Australia and Western Australia for Indigenous children.
- Combined DTPa-hepB-IPV-Hib (PRP-T)† or PRP-OMP* used for non-Indigenous children in these states and all children in other jurisdictions.

2009

- Combined DTPa-hepB-IPV-Hib (PRP-T) vaccine at 2, 4, 6 and 12 months of age used in all jurisdictions.

Key points

Rates of Hib disease are now much lower than in the pre-vaccine era, for both Aboriginal and Torres Strait Islander and other children. However, throughout the post-vaccine period, higher rates have been noted in Aboriginal and Torres Strait Islander children than in other children, with the disparity increasing over time. Notification rates are now almost 13 times higher in Aboriginal and Torres Strait Islander people than in other people. Persisting Hib carriage in Aboriginal and Torres Strait Islander people highlights the need for ongoing vigilance.

* PRP-OMP: *Haemophilus influenzae* type b polysaccharide conjugated to the outer membrane protein of *Neisseria meningitidis*

† PRP-T: *Haemophilus influenzae* type b polysaccharide conjugated to tetanus toxoid

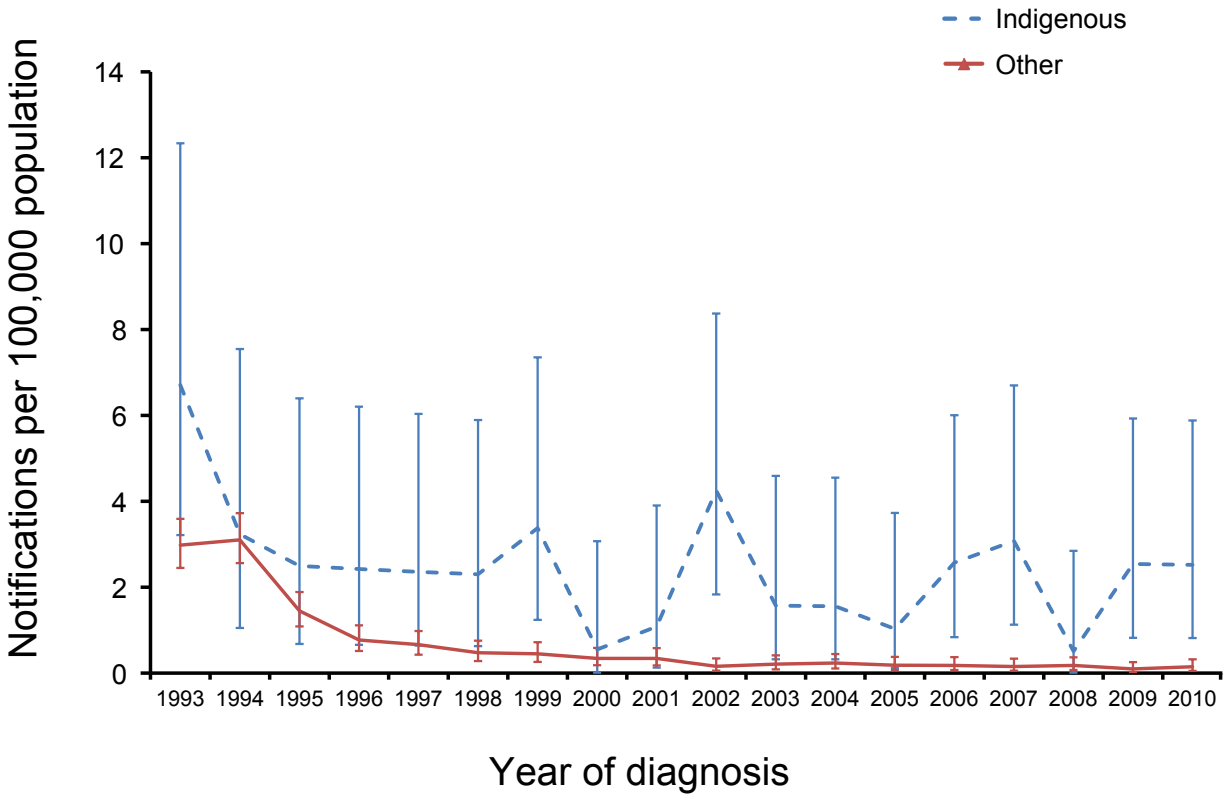
Disease trends

Enhanced Hib disease notification data* for all jurisdictions for the period 1993–2010 has been presented here for those <15 years of age, the age group in whom the disease burden is primarily seen (Figure 2.1.1). When compared to the disease rates prior to 1993, notification rates in the post-Hib vaccine era were significantly lower but, between 2007 and 2010, rates among Aboriginal and Torres Strait Islander children remained higher than in other children, in whom rates remained low and stable.

Notification data are presented for all jurisdictions for the period 2007–2010. No hospitalisation data are presented for invasive Hib disease because no type-specific ICD-10 code exists.

* A follow-up questionnaire is sent for each notification, collecting additional data.

Figure 2.1.1: Hib notification rates and 95% confidence intervals, all Australian states, 1993 to 2010,* <15 years of age, by Indigenous status



* Notifications where the date of diagnosis was between 1 January 1993 and 31 December 2010.

Table 2.1.1: Hib notifications, all Australian states, 2007 to 2010, by age group and Indigenous status

Age group (years)	Indigenous status	Notifications* (2007–2010)		
		n	Rate†	Rate ratio
0–4	Indigenous	15	5.6	15.7§
	Other	19	0.4	
5–14	Indigenous	2	0.4	8.1
	Other	5	0.0	
15–24	Indigenous	2	0.5	13.4§
	Other	4	0.0	
25–49	Indigenous	4	0.6	14.7§
	Other	12	0.0	
≥50	Indigenous	2	0.7	9.9§
	Other	20	0.1	
All ages‡	Indigenous	25	0.9	12.9§
	Other	60	0.1	

* Notifications where the date of diagnosis was between 1 January 2007 and 31 December 2010.

† Average annual age-specific rate per 100,000 population.

‡ Includes cases with age unknown. Rates for all ages combined are age-standardised to the Australian Bureau of Statistics Australian population estimates for 2006.

§ Indicates statistically significant, 95% confidence intervals do not overlap 1.0

A total of 85 notifications of invasive Hib disease were recorded during this reporting period, of which 25 (29.4%) were reported in Aboriginal and Torres Strait Islander people. The notification rate was higher in Aboriginal and Torres Strait Islander people across all age groups, with the highest rate of 5.6 per 100,000 in the 0–4 years age group (Table 2.1.1).

The highest notification rate ratio (15.7:1) was also seen in the youngest age group, followed closely by the 25–49 years and 15–24 years age groups (Table 2.1.1). The overall Indigenous to non-Indigenous rate ratio increased from 8.8:1 in the previous reporting period (2003–2006) to 12.9:1 in 2007–2010.

The ICD-10-AM/ICD-10 code G00.0 (*Haemophilus meningitis*) was used as the code most likely to include deaths due to *Haemophilus influenzae* type b. There were no deaths reported from the five reporting jurisdictions (New South Wales, the Northern Territory, Queensland, South Australia and Western Australia) for the period 2006–2010 with *Haemophilus meningitis* as either the underlying or a contributing cause.

Comment

Hib notification rates have decreased significantly since the introduction of vaccines in 1993. National data on Aboriginal and Torres Strait Islander people are not available for the pre-vaccine period, but studies in the Northern Territory reported an incidence of between 270 and 530 per 100,000 in children aged <5 years.[4,5] In contrast, the national rate for Aboriginal and Torres Strait Islander children in this age group in this reporting period was 5.6 per 100,000. However, while rates in other children have continued to decline in recent years, those in Aboriginal and Torres Strait Islander children have not, resulting in a widening disparity.

Data from most Indigenous populations worldwide have shown dramatic decreases in invasive Hib disease, but the rates remain higher than in corresponding non-Indigenous populations.[6] Environmental and social deprivation, including household crowding and high smoking rates, are the most likely causes for this disparity and are the most commonly shared characteristics between Indigenous populations in developed countries. Studies have shown that Hib nasopharyngeal carriage continues in Aboriginal and Torres Strait Islander children in some areas despite high levels of vaccination.[7] It has also been suggested that some Indigenous populations have an increased susceptibility to Hib disease as well as poor immune responses to immunisation. Genetic factors have been implicated in some settings,[6] but in Aboriginal and Torres Strait Islander children, weaker immune responses occurred only after the first year of life, suggesting environmental causes were responsible for the poorer immune response.[8]

PRP-OMP vaccine has been preferred for Aboriginal and Torres Strait Islander children in Australia and American Indian children in the United States. It provides protection at an earlier age than other vaccines, and Aboriginal and Torres Strait Islander children are affected at an earlier age than other children.[9] A resurgence of Hib disease was observed in Alaskan Native children following the replacement of a PRP-OMP vaccine with HbOC (Hib oligosaccharide CRM197), which then prompted a return to PRP-OMP vaccines in Alaska.[10] The progressive withdrawal of PRP-OMP vaccines (3 doses) in Australia from 2005 to 2009, replaced by 4 doses of PRP-T, was due to an international shortage of PRP-OMP vaccine. While it is possible that higher disease rates in young infants could be a consequence of the later age of protection from PRP-T vaccine, it is also possible that higher immunogenicity of PRP-T vaccine will result in reduced carriage. Resurgence of Hib disease following use of PRP-T vaccine has not been reported from Canada[11] or New Zealand,[12] where this vaccine has been used for both Indigenous and non-Indigenous children. Vigilant surveillance and high vaccination coverage are particularly important in this setting, but substantial socioeconomic improvements remain imperative.

2.2 Hepatitis A

Hepatitis A is caused by hepatitis A virus (HAV) which is a picornavirus.[3] Infection may be inapparent, asymptomatic or symptomatic. Symptoms include fatigue, malaise, abdominal pain, nausea and vomiting. Features typical of hepatitis A include jaundice, dark urine and pale-coloured stools.[3] Fulminant hepatitis A is the most severe form of the disease and the host factors associated with an increased risk of this complication include older age and chronic liver disease, with mortality as high as 60%. Infections acquired before the age of 5 years are asymptomatic in 50% to 90% of cases, so under-reporting may be an issue, whereas 70% to 90% of infected adults will have symptoms.[3]

Relevant vaccine history

1999

- Hepatitis A vaccine commenced for Aboriginal and Torres Strait Islander children 18 months to 6 years of age living in North Queensland.

2005

- Hepatitis A vaccination (2 doses) recommended and funded for Aboriginal and Torres Strait Islander children 12–24 months of age residing in the Northern Territory, Queensland, South Australia and Western Australia.

Key points

Since 2005 there has been a substantial reduction in both notifications and hospitalisations of hepatitis A in Aboriginal and Torres Strait Islander people. A recent increase in notifications has been noted in other people, due to a large foodborne outbreak.

Disease trends

Notification rates for hepatitis A have dropped significantly over the period from 2000 to 2010, especially for Aboriginal and Torres Strait Islander people. The decline has been more marked since 2006/2007 which reflects the impact of the expanded hepatitis A vaccination program introduced in 2005 in areas of high incidence (the Northern Territory, Queensland, South Australia and Western Australia). There were no cases notified among Aboriginal and Torres Strait Islander people in 2007 or 2010 and only small numbers in 2008 and 2009. The small increase in 2009 (Figure 2.2.1) corresponds to the Australia-wide outbreak of hepatitis A which occurred mainly in non-Indigenous people.

Notification data are presented for all jurisdictions for the period 2007–2010. Hospitalisation data are presented for six jurisdictions (all except Tasmania and the Australian Capital Territory) by financial year for the period July 2006 to June 2010. This includes data from jurisdictions that were not included in the vaccination program because their disease rates were already low. The 2005/2006 year has been excluded from the hospitalisation data for hepatitis A to restrict the data to the post-vaccination period.

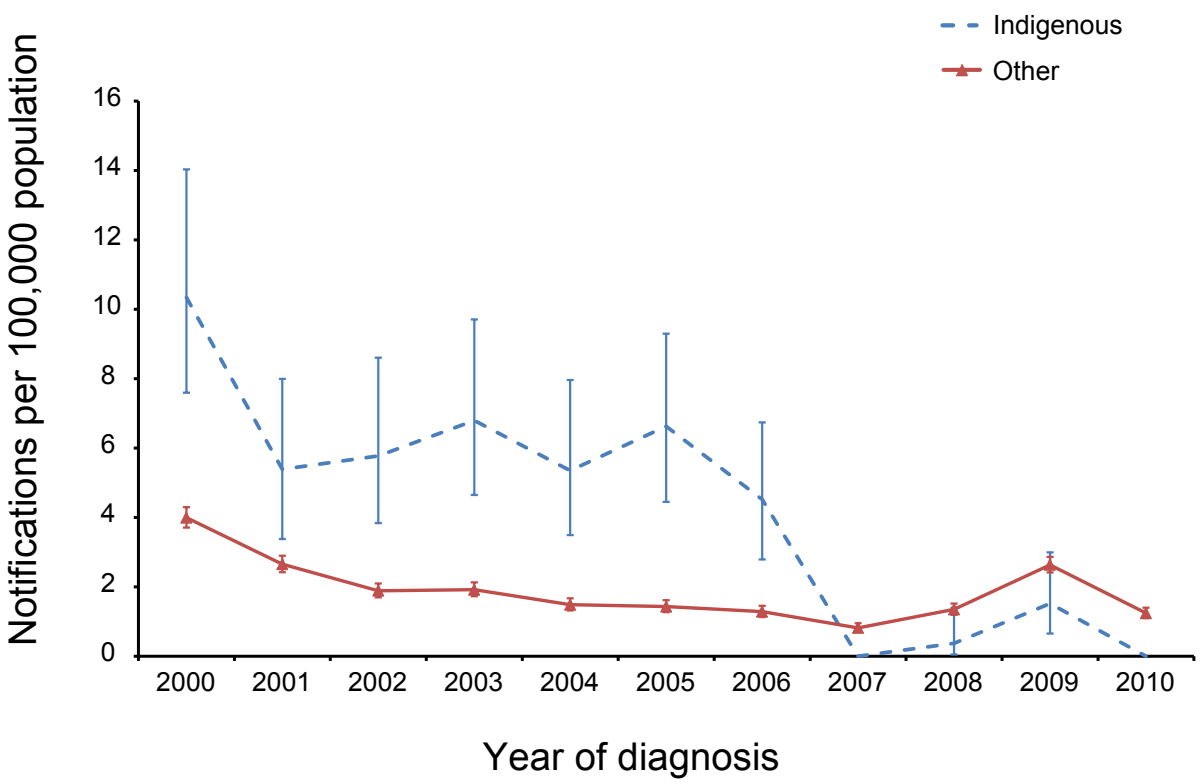
A total of 1,272 notifications and 869 hospitalisations for hepatitis A were recorded during their respective reporting periods; of these, 11 (0.9%) notifications and 19 (2.2%) hospitalisations were reported in Aboriginal and Torres Strait Islander people (Table 2.2.1 and Table 2.2.2). Both these numbers are much lower than in the previous reporting periods: 162 (14%) notifications (2003–2006) and 66 (11%) hospitalisations (2002/2003–2004/2005).

Notification rate point estimates in Aboriginal and Torres Strait Islander people were lower than in other people across all age groups. The differences were statistically significant for age groups from 15 years and above. The highest rate in Aboriginal and Torres Strait Islander people was 1 per 100,000, in the 5–14 years age group, while the highest rate in others was 2.2 per 100,000, in the 15–24 years age group (Table 2.2.1). A similar pattern was seen in hospitalisation rates, with point estimates lower in Aboriginal and Torres Strait Islander people than in others across all age groups except 25–49 years (Table 2.2.2). The overall Indigenous to non-Indigenous rate ratios were 0.3:1 for notifications and 0.9:1 for hospitalisations.

No notifications or hospitalisations were recorded for Aboriginal and Torres Strait Islander people over 50 years of age during this reporting period. Among Aboriginal and Torres Strait Islander children aged 0–4 years, there were no notifications during 2008–2010 and no hospitalisations during 2006/2007–2009/2010 for the first time since 2000 (data not shown).

There were 6 deaths reported from the five reporting jurisdictions (New South Wales, the Northern Territory, Queensland, South Australia and Western Australia) for the period 2006–2010 with hepatitis A as the underlying cause and 12 deaths with hepatitis A as either the underlying or a contributing cause. None of these deaths were reported in Aboriginal or Torres Strait Islander people.

Figure 2.2.1: Hepatitis A notification rates and 95% confidence intervals, selected Australian states,* 2000 to 2010,† by Indigenous status



* Jurisdictions with satisfactory data quality over the whole time period; see Appendix A (New South Wales, Northern Territory, Queensland, South Australia, Victoria, Western Australia).

† Notifications where the date of diagnosis was between 1 January 2000 and 31 December 2010. Rates are age-standardised to the Australian Bureau of Statistics Australian population estimates for 2006.

Table 2.2.1: Hepatitis A notifications, all Australian states, 2007 to 2010, by age group and Indigenous status

Age group (years)	Indigenous status	Notifications* (2007–2010)		
		n	Rate†	Rate ratio
0–4	Indigenous	1	0.4	0.3
	Other	70	1.3	
5–14	Indigenous	5	1.0	0.5
	Other	194	1.8	
15–24	Indigenous	3	0.7	0.3§
	Other	258	2.2	
25–49	Indigenous	2	0.3	0.2§
	Other	500	1.7	
≥50	Indigenous	0	0.0	0.0§
	Other	239	0.9	
All ages‡	Indigenous	11	0.5	0.3§
	Other	1,261	1.5	

* Notifications where the date of diagnosis was between 1 January 2007 and 31 December 2010.

† Average annual age-specific rate per 100,000 population.

‡ Includes cases with age unknown.

§ Indicates statistically significant, 95% confidence intervals do not overlap 1.0

Table 2.2.2: Hepatitis A hospitalisations, selected Australian states, 2006 to 2010, by age group and Indigenous status

Age group (years)	Indigenous status	Hospitalisations* (2006–2010)		
		n	Rate†	Rate ratio
0–4	Indigenous	0	0.0	0.0
	Other	13	0.3	
5–14	Indigenous	2	0.4	0.8
	Other	48	0.5	
15–24	Indigenous	3	0.7	0.6
	Other	129	1.2	
25–49	Indigenous	14	2.2	1.8§
	Other	342	1.2	
≥50	Indigenous	0	0.0	0.0§
	Other	323	1.3	
All ages‡	Indigenous	19	0.9	0.9
	Other	850	1.1	

* Hospitalisations (New South Wales, Northern Territory, Queensland, South Australia, Victoria, Western Australia only) where the date of separation was between 1 July 2006 and 30 June 2010.

† Average annual age-specific rate per 100,000 population.

‡ Includes cases with age unknown. Rates for all ages combined are age-standardised to the Australian Bureau of Statistics Australian population estimates for 2006.

§ Indicates statistically significant, 95% confidence intervals do not overlap 1.0

Comment

In the immediate pre-vaccine period, notification and hospitalisation rates were significantly higher in Aboriginal and Torres Strait Islander people than in others in almost all age groups and in all ages combined.[13] In Aboriginal and Torres Strait Islander children <5 years of age, notification rates were 24 times higher and hospitalisation rates were 150 times higher than rates in other infants. In the post-vaccination data presented here, point estimates were lower for Aboriginal and Torres Strait Islander than for other people within almost every age group. There were no significant differences in notification or hospitalisation rates for all ages combined between Aboriginal and Torres Strait Islander people and others. This, together with the trends shown in Figure 2.2.1, makes a compelling case for the success of the expanded hepatitis A vaccination program.

Since the introduction of the targeted vaccination program for Aboriginal and Torres Strait Islander children in high incidence jurisdictions, it appears that hepatitis A has almost disappeared from these areas and is now predominantly acquired outside the Northern Territory in the non-Indigenous population. This trend will have further implications in identifying and targeting non-Indigenous at-risk groups.

Prior to vaccination, in many remote Aboriginal and Torres Strait Islander communities, the probability of exposure to the hepatitis A virus was high, thus most infants became infected and immune at an early age. The Northern Territory and the Kimberley region had significantly higher rates of hepatitis A than the rest of Australia. It was thought that the disease was hyperendemic in Aboriginal and Torres Strait Islander communities in the Northern Territory.[14] In the non-Indigenous population the probability of exposure was low, leaving the vast majority of the population non-immune and susceptible to more serious infection with increasing age.[14] The difference in the epidemiology in the two population groups reflected different transmission dynamics arising from inequalities in housing, sanitation infrastructure, hygiene and education, and also illustrates the interaction between risk of exposure, population immunity and disease incidence.[14]

In recent years, however, most cases of hepatitis A in Australia have been imported via overseas travellers returning from countries where hepatitis A is endemic.[15,16] Outbreaks due to contaminated food or water have also been reported.[15,16] In 2009, an Australia-wide outbreak with 415, predominantly non-Indigenous, cases was associated with imported semi-dried tomatoes.[17]

Internationally, Indigenous populations like American Indian and Alaskan Native people have also suffered disproportionately from infectious diseases compared with the general population in the United States. Previously in the United States, hepatitis A recommendations targeted at-risk individuals and children living in states and communities with high hepatitis A rates, which included all Native American people. By 2004, hepatitis A disease had declined to such an extent in these populations that an epidemiological shift was noted, with approximately two-thirds of reported hepatitis A cases occurring in states with historically lower incidence and without childhood hepatitis A vaccination recommendations. Recent recommendations now call for routine hepatitis A vaccination of all children.[18]

Hepatitis A infection in Aboriginal and Torres Strait Islander people is now rare, and in fact possibly even less common than in non-Indigenous people, following the introduction of a targeted vaccination program. Continued surveillance and evaluation of the current vaccination program may help to identify if there is a need for future changes to the program. There is also a need to promote vaccination of travellers to prevent outbreaks and to further reduce the burden of disease in Australia.

2.3 Hepatitis B

Hepatitis B infection is caused by hepatitis B virus (HBV) which replicates in the liver and causes hepatic dysfunction. Infection with HBV causes a broad spectrum of liver disease, including subclinical infection, acute self-limited hepatitis and fulminant hepatitis, with some of those infected developing chronic infection leading to chronic liver disease and death from cirrhosis or hepatocellular carcinoma. The main burden of disease is related to chronic HBV infection. The risk of developing chronic HBV infection varies with age; approximately 90% of infected infants, 30% of infected children 1–4 years of age and <5% of those infected as adults will develop chronic disease.[3] The analysis in this report is restricted to acute hepatitis B.

Relevant vaccine history

1980s

- Hepatitis B vaccination funded for high-risk infants, including Aboriginal and Torres Strait Islander infants, in some jurisdictions then nationally in 1988.

1990

- Neonatal hepatitis B vaccination funded for all infants in the Northern Territory (3-dose schedule: birth, 1 month and 6 months).

1997

- Hepatitis B vaccination recommended and funded for all adolescents aged 11–12 years (initially 3-dose schedule using the paediatric formulation; changed to 2 doses of adult formulation at various times since 2001 in different jurisdictions).

2000

- Universal infant vaccination included in childhood schedule with a birth dose of monovalent paediatric hepatitis B vaccine, followed by 3 doses of hepatitis B-containing combination vaccine.

Key points

Since the introduction of hepatitis B vaccination there has been a substantial reduction in HBV infection rates in Australia, although the rates are still higher in Aboriginal and Torres Strait Islander people than in other Australians. Consideration of further booster or catch-up vaccination may be necessary.

Disease trends

Notification data (only for cases recorded as acute) are presented for all jurisdictions for the period 2007–2010. Hospitalisation data (principle cause of admission only) are presented for six jurisdictions (all except Tasmania and the Australian Capital Territory) by financial year for the period July 2005 to June 2010.

A total of 1,023 notifications and 711 hospitalisations for acute hepatitis B were recorded during these reporting periods; of these, 72 (7%) notifications and 31 (4.4%) hospitalisations were reported in Aboriginal and Torres Strait Islander people (Table 2.3.1 and Table 2.3.2).

No notifications or hospitalisations were recorded during this reporting period for Aboriginal and Torres Strait Islander children aged 0–4 years. For all other age groups, higher rates were recorded among Aboriginal and Torres Strait Islander people than among other people. Both notification and hospitalisation rates increased with age, peaking in the 25–49 years age group for both Aboriginal and Torres Strait Islander and other people.

The overall Indigenous to non-Indigenous rate ratios were 3.1:1 for notifications and 2.2:1 for hospitalisations, both of which were statistically significantly different to 1:1. The biggest difference in notification rates was in the 15–24 years age group (rate ratio 4.6:1), while for hospitalisations the greatest difference was in the 5–14 years age group (rate ratio 6.8:1, not statistically significantly different to 1:1).

There were 32 deaths reported from the five jurisdictions (New South Wales, the Northern Territory, Queensland, South Australia and Western Australia) for the period 2006–2010 with hepatitis B as the underlying cause, of which 6 to 9 were reported in Aboriginal and Torres Strait Islander people (the ABS provides ranges when absolute numbers of deaths are low). None of the deaths in Aboriginal and Torres Strait Islander people were in children <5 years of age. There were 179 deaths reported with hepatitis B as either the underlying or a contributing cause, of which 25 (14%) were reported in Aboriginal and Torres Strait Islander people; none of these deaths were in children <5 years of age.

Table 2.3.1: Hepatitis B* notifications, all Australian states, 2007 to 2010, by age group and Indigenous status

Age group (years)	Indigenous status	Notifications† (2007–2010)		
		n	Rate‡	Rate ratio
0–4	Indigenous	0	0.0	0.0
	Other	9	0.2	
5–14	Indigenous	2	0.4	4.5
	Other	9	0.1	
15–24	Indigenous	24	5.4	4.6ll
	Other	138	1.2	
25–49	Indigenous	42	6.1	3.0ll
	Other	625	2.1	
≥50	Indigenous	4	1.5	2.3
	Other	170	0.6	
All ages§	Indigenous	72	3.5	3.1ll
	Other	951	1.1	

* Recorded as acute only.

† Notifications where the date of diagnosis was between 1 January 2007 and 31 December 2010.

‡ Average annual age-specific rate per 100,000 population.

§ Includes cases with age unknown. Rates for all ages combined are age-standardised to the Australian Bureau of Statistics Australian population estimates for 2006.

ll Indicates statistically significant, 95% confidence intervals do not overlap 1.0

Table 2.3.2: Hepatitis B* hospitalisations, selected Australian states, 2005 to 2010, by age group and Indigenous status

Age group (years)	Indigenous status	Hospitalisations† (2005–2010)		
		n	Rate‡	Rate ratio
0–4	Indigenous	0	0.0	0.0
	Other	1	0.0	
5–14	Indigenous	1	0.2	6.8
	Other	3	0.0	
15-24	Indigenous	6	1.2	2.2
	Other	77	0.6	
25-49	Indigenous	19	2.4	1.9ll
	Other	447	1.3	
≥50	Indigenous	5	1.7	3.4ll
	Other	152	0.5	
All ages§	Indigenous	31	1.6	2.2ll
	Other	680	0.7	

* Principal cause of admission only.

† Hospitalisations (New South Wales, Northern Territory, Queensland, South Australia, Victoria, Western Australia only) where the date of separation was between 1 July 2005 and 30 June 2010.

‡ Average annual age-specific rate per 100,000 population.

§ Includes cases with age unknown. Rates for all ages combined are age-standardised to the Australian Bureau of Statistics Australian population estimates for 2006.

ll Indicates statistically significant, 95% confidence intervals do not overlap 1.0

Comment

Australia is generally regarded as a country with low risk for hepatitis B virus infection. This has been achieved by securing a safe blood supply, implementing a national hepatitis B vaccination program, and providing treatment for people with chronic hepatitis B through the Pharmaceutical Benefits Scheme. However, hepatitis B has been common in some Aboriginal and Torres Strait Islander communities, with the highest rates reported in the Northern Territory,[19] including a death rate from hepatic cancer 10 times that in other people.[20] Surveillance data such as those presented here have serious limitations; they are unlikely to include asymptomatic infections and so may underestimate acute hepatitis B disease, especially in children in whom most infection is asymptomatic. Moreover, they do not reflect the disease burden from chronic hepatitis B or later complications such as liver cirrhosis and hepatocellular carcinoma. The highest notification and hospitalisation rates were in the 25–49 years age group, which suggest continued horizontal transmission of infection.

Studies of markers of infection in Northern Territory Aboriginal and Torres Strait Islander adolescents and young adults who were targeted in the early years of the vaccination program show evidence of substantially lower levels of infection and chronic infection compared to the pre-immunisation period;[21] however, levels are still higher than in non-Indigenous people of the same age.[22] Some studies in some Aboriginal communities have suggested that immune responses to the early hepatitis B vaccines may have been suboptimal.[23] An audit conducted in East Arnhem Land in the Northern Territory concluded that HBV infection was an ongoing public health problem in some Aboriginal communities for all age groups; reasons for this included the existence of a large cohort of susceptible people born before 1990, incomplete immunisation, ongoing vertical transmission despite the use of hepatitis B immunoglobulin, and a poor response to vaccination among Aboriginal children.[22] There may also be a need for booster doses for those vaccinated as infants.[21]

Internationally, Alaska reported a decline in infection rates in both Alaskan Native and non-Native people after 1984, when universal infant vaccination commenced followed by a catch-up program from 1984–1988 for Alaskan Native people. The decline was further helped by ongoing high rates of immunisation.[18]

Substantial impacts of universal hepatitis B vaccination on the earlier manifestations of hepatitis B infection have been demonstrated in Aboriginal and Torres Strait Islander and other Australians,[24,25] and these are likely to be reflected in lower rates of the serious long-term sequelae in coming years. However, there is evidence

that infection continues to occur in Aboriginal and Torres Strait Islander communities that suffered from high levels of disease in the pre-vaccine era. *The Australian Immunisation Handbook 10th edition* recommends that all Aboriginal and Torres Strait Islander people have their risks and vaccination status reviewed, be offered testing and be vaccinated if non-immune.[26]

2.4 Seasonal influenza, pandemic influenza and pneumonia

Influenza is an acute respiratory tract infection caused mainly by influenza type A and type B viruses.[3] Acute febrile influenza illness can range from mild to debilitating, and in some cases may become exacerbated by a variety of secondary complications. The risk of developing serious complications is higher at both extremes of age and also in those with certain underlying conditions. The most common serious complications include exacerbation of underlying chronic pulmonary and cardiopulmonary diseases, such as chronic obstructive pulmonary disease, asthma and congestive heart failure, as well as development of pneumonia (primary viral or bacterial) which may be fatal.[3] Vaccination against the disease is complicated by the capacity of influenza A and B viruses to undergo gradual antigenic change in their two surface antigens, haemagglutinin (HA) and neuraminidase (NA).[3] Antigenic shift in influenza viruses can cause pandemics which are associated with higher rates of illness and death.[3]

Relevant vaccine history

1986

- Seasonal influenza vaccination recommended for individuals at risk of complications or death from influenza: persons >65 years of age, persons with chronic debilitating disease, persons receiving immunosuppressive therapy, persons engaged in medical and health services.

1994

- Seasonal influenza vaccination recommended for Aboriginal and Torres Strait Islander people aged >50 years.

1999

- Seasonal influenza vaccine funded nationally for all Australians aged >65 years and Aboriginal and Torres Strait Islander people aged >50 years or aged 15–50 years with medical risk factors.

2009

- Pandemic influenza A (H1N1) 2009 vaccine registered, recommended and funded: 1 dose for children aged ≥10 years and adults; and 2 doses for children aged 6 months to ≤9 years.

2010

Seasonal influenza vaccine funded for:

- all Aboriginal and Torres Strait Islander people aged ≥15 years
- all persons aged ≥6 months with medical conditions predisposing to severe influenza for whom influenza vaccination is recommended
- pregnant women.

Key points

Severe illness due to seasonal influenza infection is more common in Aboriginal and Torres Strait Islander people; this was also the case with the 2009 pandemic influenza A (H1N1). This is thought to be due to a higher prevalence of risk factors for severe disease in Aboriginal and Torres Strait Islander people. Improvement in vaccination coverage should be a priority.

Disease trends

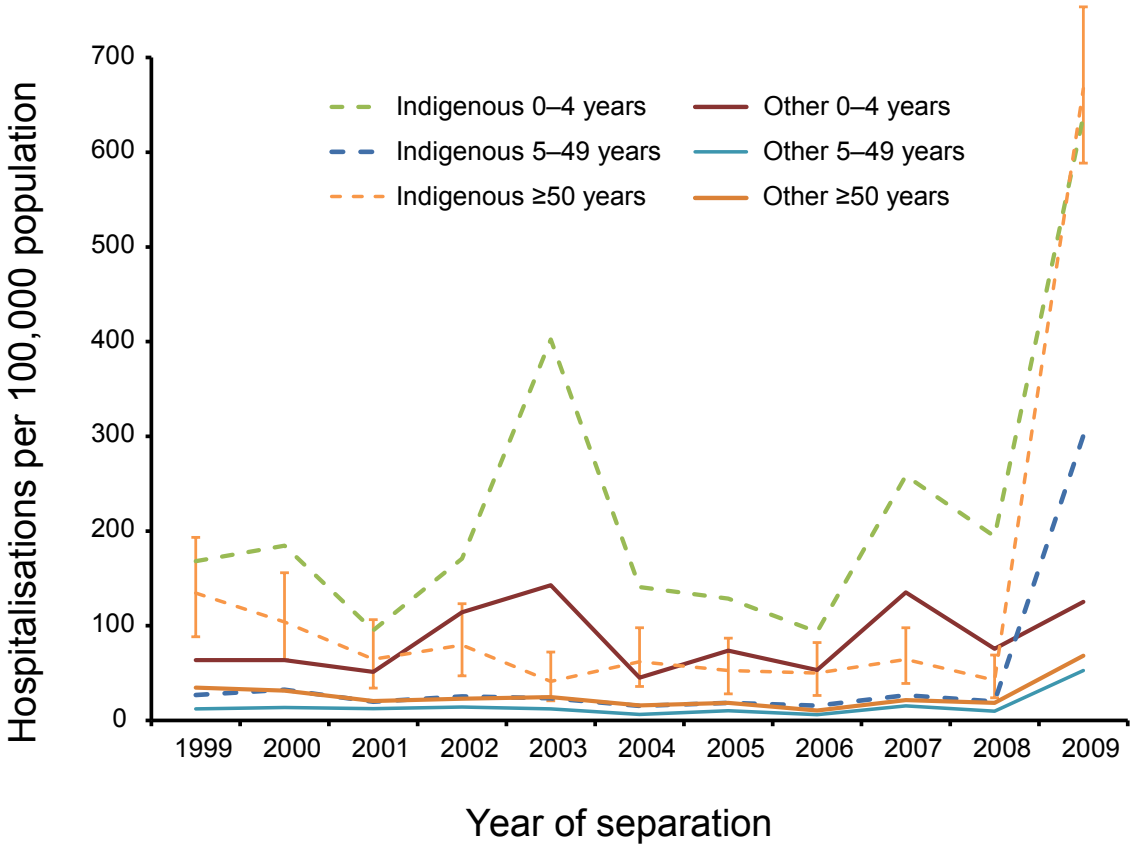
Influenza hospitalisation data are presented for the period 1999–2010 for the Northern Territory, Queensland, South Australia and Western Australia. Time trends in figures are presented by calendar instead of financial years so that each data point includes one full winter season. Data for notifications are not presented due to the low level of completeness of the Indigenous status field in notification records.

Influenza hospitalisation rates for Aboriginal and Torres Strait Islander people aged ≥50 years declined somewhat after the first year of funded vaccination (1999) but confidence intervals for individual years overlapped. Hospitalisation rates in Aboriginal and Torres Strait Islander people in 2005–2010 are still 4.6 times higher than the rates in other people. The peaks in 2003 and 2007 (Figure 2.4.1) reflect more severe influenza seasons nationally. The spike in 2009 coincides with the influenza A (H1N1) pandemic. A much higher impact of the influenza pandemic was noted in Aboriginal and Torres Strait Islander people than in other people (Figure 2.4.1). A more detailed analysis of enhanced data on pandemic influenza A (H1N1) 2009 infection has been included below as a subsection in this chapter.

Hospitalisation rates for influenza and pneumonia combined (Figure 2.4.2) are more than 20 times the rates for influenza alone, with a large difference between Aboriginal and Torres Strait Islander and other people, and little change over time. 'Influenza and pneumonia' is, however, a non-specific diagnosis with multiple aetiologies.

Hospitalisation data for influenza are presented for six jurisdictions (all except Tasmania and the Australian Capital Territory) by financial year for the period July 2005 to June 2010. A total of 22,998 hospitalisations for influenza were recorded during this reporting period, of which 2,245 (10.8%) were reported in Aboriginal

Figure 2.4.1: Influenza* hospitalisation rates, selected Australian states,† 1999 to 2009,‡ by age group and Indigenous status

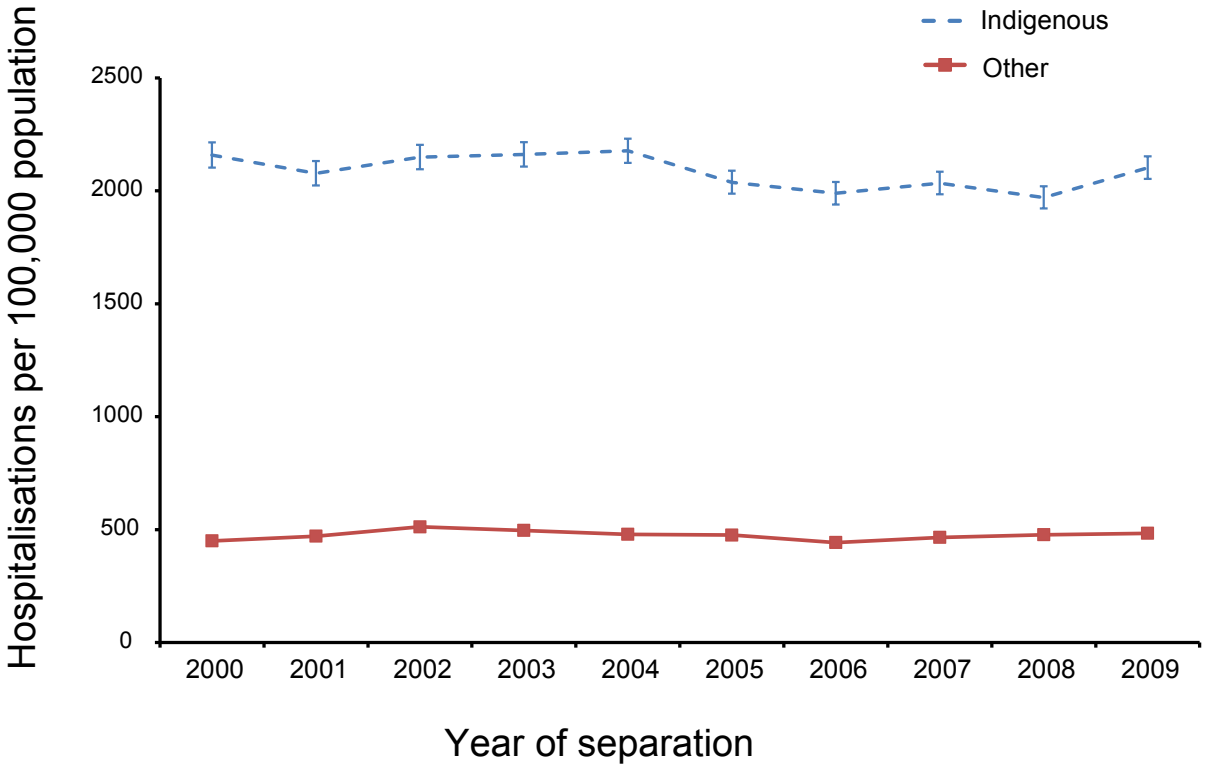


* The ICD-10-AM codes used to identify influenza hospitalisations were: J09 (influenza due to certain identified influenza viruses), J10 (influenza due to identified influenza virus) and J11 (influenza, virus not identified).

† Jurisdictions with satisfactory data quality over the whole time period; see Appendix A (Northern Territory, Queensland, South Australia, Western Australia).

‡ Hospitalisations where the date of separation was between 1 January 1999 and 31 December 2009.

Figure 2.4.2: Influenza and pneumonia* hospitalisation rates and 95% confidence intervals, selected Australian states,† 2000 to 2009,‡ by Indigenous status



* The ICD-10-AM codes used to identify hospitalisations were J09–J18 (Influenza and/or pneumonia).

† Jurisdictions with satisfactory data quality over the whole time period; see Appendix A (Northern Territory, Queensland, South Australia, Western Australia).

‡ Hospitalisations where the date of separation was between 1 January 2000 and 31 December 2009. Rates are age-standardised to the Australian Bureau of Statistics Australian population estimates for 2006.

and Torres Strait Islander people. Rates in Aboriginal and Torres Strait Islander people were consistently higher than in other people across all age groups (Table 2.4.1). The highest hospitalisation rates were seen in Aboriginal and Torres Strait Islander children in the 0–4 years age group (209.7 per 100,000) followed by those in the ≥50 years age group (136.1 per 100,000). The overall Indigenous to non-Indigenous rate ratio was 4.6:1.

There were 235 deaths reported from the five jurisdictions (New South Wales, the Northern Territory, Queensland, South Australia and Western Australia) for the period 2006–2010 with influenza as the underlying cause. Of those deaths, 14 were reported in Aboriginal and Torres Strait Islander people; there were no deaths in the 0–4 years age group, 6 in the 5–49 years age group, and 8 in the ≥50 years age group. There were 7,879 deaths reported with influenza or pneumonia as the underlying cause, of which 183 were reported in Aboriginal and Torres Strait Islander people; 17 in the 0–4 years age group, 68 in the 5–49 years age group and 98 in the ≥50 years age group. There were 341 deaths reported with influenza as the underlying or a contributing cause (20–23 in Aboriginal and Torres Strait Islander people) and 58,268 deaths with pneumonia as the underlying or a contributing cause (1,120 in Aboriginal and Torres Strait Islander people). The ABS provides ranges when absolute numbers of deaths are low.

Table 2.4.1: Influenza hospitalisations, selected Australian states, 2005 to 2010, by age group and Indigenous status

Age group (years)	Indigenous status	Hospitalisations* (2005–2010)		
		n	Rate†	Rate ratio
0–4	Indigenous	657	209.7	2.9§
	Other	4,435	72.0	
5–14	Indigenous	238	38.4	2.6§
	Other	1,876	14.9	
15–24	Indigenous	264	53.3	3.4§
	Other	2,178	15.9	
25–49	Indigenous	678	85.2	5.4§
	Other	5,603	15.7	
≥50	Indigenous	408	136.1	6.3§
	Other	6,661	21.6	
All ages‡	Indigenous	2,245	97.2	4.6§
	Other	20,753	21.0	

* Hospitalisations (New South Wales, Northern Territory, Queensland, South Australia, Victoria, Western Australia only) where the date of separation was between 1 July 2005 and 30 June 2010.

† Average annual age-specific rate per 100,000 population.

‡ Includes cases with age unknown. Rates for all ages combined are age-standardised to the Australian Bureau of Statistics Australian population estimates for 2006.

§ Indicates statistically significant, 95% confidence intervals do not overlap 1.0

Pandemic influenza A (H1N1) 2009

Analysed data for this subsection on pandemic influenza in Aboriginal and Torres Strait Islander people was provided by the Australian Government Department of Health and Ageing (Table 2.4.2).[27] During the 2009 influenza pandemic, cases of laboratory-confirmed influenza A (H1N1) 2009 infections, hospitalisations and deaths were notified to state and territory health departments, which then notified the National Incident Room (NIR) using NetEpi, a web-based outbreak case reporting system. Nationally collected NetEpi data is used here to describe the severity and mortality of pandemic influenza A (H1N1) during 2009 in Aboriginal and Torres Strait Islander Australians.

During 2009, a total of 37,683 notifications of pandemic influenza A (H1N1) 2009 were reported to NetEpi (Table 2.4.2). Of those, 11% (4,063/37,683) were listed as Aboriginal and Torres Strait Islander, 50% (18,832/37,683) were non-Indigenous, and for 39% (14,788/37,683) Indigenous status was missing or unknown. Cases with unknown or missing Indigenous status were included in the 'other' group for further analysis. Other notifications peaked in the 15–19 years age group and then declined with increasing age. For notifications in Aboriginal and Torres Strait Islander people there was no clear trend by age, the highest rate was in the 50–54 years age group (Figure 2.4.3).

Of the total 37,683 notified cases reported to NetEpi, 4,993 (13.3%) were hospitalised, with 17% (830/4,993) of these reported as Aboriginal and Torres Strait Islander and 83% (4,163/4,993) as other (Table 2.4.2). Twenty per cent (830/4,063) of all Aboriginal and Torres Strait Islander notifications and 12.4% (4,163/33,620) of other notifications were hospitalised. In Aboriginal and Torres Strait Islander people, the highest rate of hospitalisation (395 per 100,000) was in the 50–54 years age group; in other people, the highest rate (58 per 100,000) was in the <5 years age group. The age-standardised Indigenous to non-Indigenous rate ratio for admissions to hospital was 8.5.

A total of 489 influenza A (H1N1) 2009 cases notified to NetEpi were admitted to an intensive care unit (ICU) during 2009, representing 1.3% of all notifications (Table 2.4.2). Victoria and Queensland used the Australian and New Zealand Intensive Care Study (ANZICS) to record ICU admissions and so are not represented in this analysis for ICU admissions. The highest rate of ICU admissions for Aboriginal and Torres Strait Islander

people was in the 55–59 years age group with 75.6 admissions per 100,000. For other people, the highest ICU admission rate was in the 50–54 years age group, but it was much lower at 4 per 100,000. The age-standardised Indigenous to non-Indigenous rate ratio for ICU admissions was 7.9.

Pregnant women have also been identified as an important high priority group because of the increased risk of severe health outcomes from influenza.[28] Of the 37,683 notifications for influenza A (H1N1) 2009, 568 (1.5%) were in pregnant women, with 10% (55/568) of those being in Aboriginal and Torres Strait Islander women. Of pregnant women who were notified with influenza A (H1N1) 2009 infection, 53% (300/568)

Table 2.4.2: Pandemic influenza A (H1N1) 2009 infections, Australia, 2009, by Indigenous status

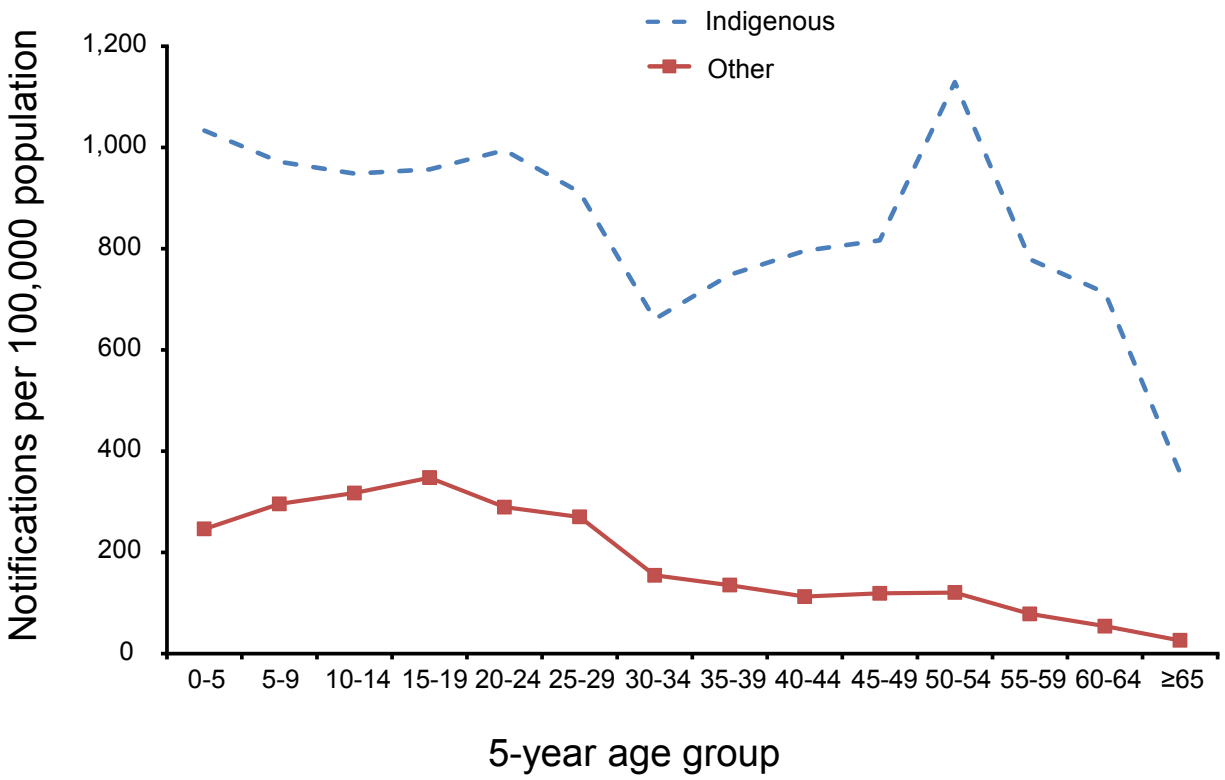
	Indigenous			Other			Rate ratio
	n	Rate*	Median age (95% CI)	n	Rate*	Median age (95% CI)	
Notifications	4,063	892.7	18 (18–19)	33,620	173.3	21 (21–22)	5.2†
Hospitalisations	830	182.4	31 (28–34)	4,163	21.5	31 (29–32)	8.5†
ICU admissions	77	16.9	42 (36–44)	412	2.1	43 (40–47)	7.9†
Deaths	23	5.1	50 (42–56)	168	0.9	50 (47–55)	5.8†

* Age-standardised rate per 100,000 population, standardised to 2006 non-Indigenous population.

† Indicates statistically significant, 95% confidence intervals do not overlap 1.0

Source: H1N1 2009 infections in Australia's Indigenous population in 2009, DOHA27

Figure 2.4.3: Pandemic influenza A (H1N1) 2009 infection reporting rates to NetEpi, Australia, 2009, by age group



Source: H1N1 2009 infections in Australia's Indigenous population in 2009, DOHA27

were hospitalised and 15% (44/300) of those hospitalised were Aboriginal and Torres Strait Islander women. Among women aged 20–34 years, 37% of those requiring hospitalisation and 33% of those requiring ICU admission were pregnant.

Metabolic disorders, diabetes, cardiac conditions, respiratory conditions and obesity were more likely to be found as comorbidities in Aboriginal and Torres Strait Islander people notified with influenza A (H1N1) 2009 than in other people. Aboriginal and Torres Strait Islander people with renal failure and diabetes were more than twice as likely as other people with those conditions to be notified, hospitalised and admitted to ICU with influenza A (H1N1) 2009. Of Aboriginal and Torres Strait Islander people who were hospitalised, almost 50% had at least one comorbidity.

There were a total of 191 deaths from influenza A (H1N1) infection during 2009, representing 0.5% of all notifications (Table 2.4.2). Of those, 23 (12%) were in Aboriginal and Torres Strait Islander people and 168 (88%) were in other people. The age-standardised Indigenous to non-Indigenous rate ratio was 5.8.

Comment

The new influenza A (H1N1) virus which emerged among humans in Mexico in early 2009 generated the first influenza pandemic of the 21st century. The clinical spectrum of disease, severity of illness and the risk factors for complications among confirmed cases of influenza A (H1N1) 2009 illness in Australia were consistent with reports from overseas and were similar to the disease profile of seasonal influenza.[29] The underlying medical conditions associated with pandemic influenza A (H1N1) 2009 and seasonal influenza illnesses diagnosed in the community were also similar. The main differences were that, for pandemic influenza A (H1N1) 2009, there was a shift to a younger age distribution and an even greater risk associated with pregnancy.[29] The reduced susceptibility of the elderly to pandemic influenza A (H1N1) 2009 resulted in a smaller number of influenza-related deaths in that age group than in most other influenza seasons.[28]

Australian public health agencies identified groups vulnerable to poor outcomes from pandemic influenza A (H1N1) 2009 infection and targeted them for priority receipt of antiretroviral treatment and vaccination. These groups were Aboriginal and Torres Strait Islander people, pregnant women, and people with morbid obesity or serious underlying medical conditions.[28] The public health response for Aboriginal and Torres Strait Islander people was facilitated by establishing an Indigenous Influenza Network which held regular teleconferences to coordinate the response.[30] Some reports documenting local efforts to target Aboriginal and Torres Strait Islander people have been published.[31,32]

Increased risks of infection, hospitalisation and death due to pandemic influenza A (H1N1) 2009 in Indigenous populations have been reported in many countries including the United States, Canada, New Zealand and Australia,[33-35] and are also presented in this report. The occurrence of more severe forms of disease has been attributed to a higher susceptibility and prevalence of comorbidities among Indigenous people.[33,34] Diseases including cardiovascular disease, diabetes and chronic respiratory disease are responsible for up to 70% of the observed health gap between Aboriginal and Torres Strait Islander and other Australians.[36] However, there is also evidence of an increased risk of exposure to the virus,[37] as well as higher hospitalisation rates in Aboriginal and Torres Strait Islander people than in others with the same risk factor.[27] A highly mobile population and crowded living conditions are also thought to be risk factors for increased exposure to influenza and other viruses. Other factors causing reduced resilience may also be present in Aboriginal and Torres Strait Islander people.

Reports have indicated that uptake of vaccination against pandemic influenza A (H1N1) 2009 was suboptimal in the general population, at-risk groups, pregnant women and health professionals in most countries.[38] For Australians of all ages, the vaccination coverage was around 20% for Aboriginal and Torres Strait Islander people and 21% for other people.[39] However, this sole national estimate for Aboriginal and Torres Strait Islander people was from a small subset in a national telephone survey. No more detailed breakdowns are available, and there are limitations around using crude telephone surveys in this population with poorer telephone access.[40] Other estimates are limited to Western Australia (20%)[41] and the Northern Territory (41%).[37] It is therefore difficult to evaluate the success of measures to prioritise Aboriginal and Torres Strait Islander people.

Seasonal influenza still remains an issue in vulnerable populations. The data presented here indicate that seasonal influenza rates continue to be higher in Aboriginal and Torres Strait Islander people than in other

people, including in the ≥50 year age group, in which vaccine has been funded for Aboriginal and Torres Strait Islander people since 1999. Since 2010, seasonal influenza vaccine has been funded for all Aboriginal and Torres Strait Islander people aged ≥15 years. This broadening of the vaccination program has the potential to result in a further reduction in the disease burden of seasonal influenza in Aboriginal and Torres Strait Islander people. However, a scarcity of data is an obstacle to program management for seasonal influenza vaccination; there are no coverage estimates for Aboriginal and Torres Strait Islander adults for the first 3 years of this program (also discussed in the 'Vaccination coverage' chapter of this report). Development of strategies to monitor and increase vaccine uptake in high-risk groups should be a priority in tackling seasonal influenza, as well as in preparedness for future pandemics.

2.5 Measles

Measles is an acute and highly communicable disease caused by a morbillivirus. It is characterised by fever, malaise, conjunctivitis, coryza and cough in the prodromal stage followed by appearance of Koplik spots on the buccal mucosa before the onset of maculopapular rash. Complications include otitis media, pneumonia, diarrhoea, post-infectious encephalitis, subacute sclerosing panencephalitis (rare) and death. The risk of serious complications and death is increased in children <5 years of age and adults >20 years of age.[3]

Relevant vaccine history

1975

- Measles vaccine funded for all Australian infants at 12 months of age.

1984

- MM* vaccination of Aboriginal and Torres Strait Islander children in the Northern Territory changed from 12 months to 9 months of age.

1989

- MMR† vaccine recommended and funded on the national schedule at 12 months of age (9 months for Aboriginal and Torres Strait Islander infants in the Northern Territory).

1994

- MMR funded as second dose of measles-containing vaccine for adolescent females.

1996

- MMR funded as second dose of measles-containing vaccine for all adolescents.

1998

- Recommended age for first dose of MMR vaccine for Aboriginal and Torres Strait Islander children in the Northern Territory increased from 9 months to 12 months of age.
- Recommended age for second dose of MMR vaccine lowered to 4–5 years.

2013

- Second dose moved forward to 18 months of age, given as MMRV.‡

Key points

Australia's population is generally well immunised against measles and does not have endemic measles virus transmission. However, measles outbreaks continue to occur with most cases able to be linked to travel or exposure to returned travellers.

* MM: measles and mumps

† MMR: measles, mumps and rubella

‡ MMRV: measles, mumps, rubella and varicella

Disease trends

Notification data are presented for all jurisdictions for the period 2007–2010. Hospitalisation data are presented for six jurisdictions (all except Tasmania and the Australian Capital Territory) by financial year for the period July 2005 to June 2010.

A total of 251 notifications and 139 hospitalisations for measles were recorded during these reporting periods; of these, 3 (1.2%) notifications and 6 (4.3 %) hospitalisations were reported in Aboriginal and Torres Strait Islander people (Table 2.5.1 and Table 2.5.2). Different time periods and jurisdictions were covered by the two datasets (4 years and 6 jurisdictions for notifications, 5 years and 8 jurisdictions for hospitalisations), so direct comparisons of absolute numbers are not possible. However, the disparity between the percentages of notifications and hospitalisations reported as Aboriginal and Torres Strait Islander may be due to these children being less likely to seek healthcare for mild disease, compared to non-Indigenous children. The proportions of notifications and hospitalisations occurring in Aboriginal and Torres Strait Islander people were lower than the proportions (4% and 7%, respectively) in the previous reporting period (2003–2006).

Notification rates have remained low in Aboriginal and Torres Strait Islander people between 2007 and 2010, with no cases recorded in 2008 and only 1 case each in 2009 and 2010. Notification rates were lower in Aboriginal and Torres Strait Islander people than in other people across all age groups. The highest rate among Aboriginal and Torres Strait Islander people occurred in the youngest age group (0.4 per 100,000). The overall Indigenous to non-Indigenous rate ratio for notifications was 0.5:1 (not statistically significantly different to 1:1).

With respect to hospitalisation rates, higher rates were recorded in Aboriginal and Torres Strait Islander children 0–14 years of age than in other children of the same age, but there were no hospitalisations recorded for Aboriginal and Torres Strait Islander people ≥15 years of age. The overall Indigenous to non-Indigenous rate ratio for hospitalisations was 1.8:1, with the highest ratio (4.1:1) in the 5–14 years age group (neither of these ratios was statistically significantly different to 1:1).

There were 5–8 deaths reported in Australia between 2006 and 2010 with measles as a contributing cause (the ABS provides ranges when absolute numbers of deaths are low). None of these deaths had measles recorded as the underlying cause. Of these deaths, 1–4 occurred in the five jurisdictions for which data on Aboriginal and Torres Strait Islander people were available (New South Wales, the Northern Territory, Queensland, South Australia and Western Australia), and none were reported in Aboriginal and Torres Strait Islander people.

Table 2.5.1: Measles notifications, all Australian states, 2007 to 2010, by age group and Indigenous status

Age group (years)	Indigenous status	Notifications* (2007–2010)		
		n	Rate†	Rate ratio
0–4	Indigenous	1	0.4	0.6
	Other	31	0.6	
5–14	Indigenous	1	0.2	0.3
	Other	64	0.6	
15–24	Indigenous	0	0.0	0.0§
	Other	65	0.6	
25–49	Indigenous	1	0.1	0.5
	Other	85	0.3	
≥50	Indigenous	0	0.0	0.0
	Other	3	0.0	
All ages‡	Indigenous	3	0.1	0.5
	Other	248	0.3	

* Notifications where the date of diagnosis was between 1 January 2007 and 31 December 2010.

† Average annual age-specific rate per 100,000 population.

‡ Includes cases with age unknown.

§ Indicates statistically significant, 95% confidence intervals do not overlap 1.0

Table 2.5.2: Measles hospitalisations, selected Australian states, 2005 to 2010, by age group and Indigenous status

Age group (years)	Indigenous status	Hospitalisations* (2005–2010)		
		n	Rate†	Rate ratio
0–4	Indigenous	4	1.3	2.2
	Other	35	0.6	
5–14	Indigenous	2	0.3	4.1
	Other	10	0.1	
15–24	Indigenous	0	0.0	0.0
	Other	26	0.2	
25–49	Indigenous	0	0.0	0.0
	Other	56	0.2	
≥50	Indigenous	0	0.0	0.0
	Other	6	0.0	
All ages‡	Indigenous	6	0.2	1.8
	Other	133	0.1	

* Hospitalisations (New South Wales, Northern Territory, Queensland, South Australia, Victoria, Western Australia only) where the date of separation was between 1 July 2005 and 30 June 2010.

† Average annual age-specific rate per 100,000 population.

‡ Includes cases with age unknown.

§ Indicates statistically significant, 95% confidence intervals do not overlap 1.0

Comment

Australia's population is generally well immunised against measles and does not have endemic measles virus transmission. The data presented here show that measles rates are low in Aboriginal and Torres Strait Islander people and are no different to those in other people.

Before the introduction of measles vaccine, and even until the introduction of a second pre-school dose in 1998, measles epidemics were a feature of Australian life, including severe cases and some deaths.[24] Aboriginal and Torres Strait Islander communities were more severely affected, leading to an accelerated vaccination schedule in the Northern Territory.[42] Today, the elimination of endemic measles transmission may have already been achieved in Australia.[43] Measles cases do still occur, but most are able to be linked to travel or exposure to returned travellers.[44]

Many clusters and some large outbreaks have occurred in New South Wales,[45] Queensland and Victoria in recent years.[46] However, a study of clusters occurring in early 2009 found that they met the World Health Organization elimination criterion of ≥80% of outbreaks having transmission of fewer than 10 cases.[46] Recent measles epidemics in Europe and Africa have led to an increase in the importation of cases into Australia.

Australian adults born between 1966 and 1984 may not have immunity to measles due to them having less exposure to wild measles and receipt of only 1 dose of measles-containing vaccine (the 2-dose schedule was only introduced in Australia in 1984). Identification of adults in this cohort and offering them a dose of measles-containing vaccine, particularly prior to travel to measles-endemic countries, may play an important role in minimising the importation of wild measles virus.[44]

The success of measles immunisation demonstrates the value of universal vaccination programs that include both Aboriginal and Torres Strait Islander and other Australians.

2.6 Meningococcal disease

Meningococcal disease is caused by the meningococcus bacterium (*Neisseria meningitidis*), a Gram-negative endotoxin-producing organism.[3] Meningococcus frequently causes serious and rapidly progressive disease and despite effective antimicrobial therapy and improvements in intensive care, the overall case fatality rate remains at 10%–15%. The most common clinical presentation is that of acute bacterial meningitis; other presentations include pneumonia, septic arthritis and meningococcaemia, which can occur with or without

meningitis.[3] Of the patients who survive meningococcal disease, 10%–20% develop permanent sequelae including gangrene, extensive skin scarring, cerebral infarction, neurosensory hearing loss, cognitive deficits or seizure disorders.[3]

Relevant vaccine history

2003

- Meningococcal C conjugate vaccine added to childhood vaccination schedule at 12 months of age.

2003–2007

- National meningococcal C catch-up vaccination program for all children 2–19 years of age.

Key points

Routine meningococcal C vaccination, implemented in 2003, has resulted in a substantial decrease in cases caused by serogroup C. However, rates of meningococcal disease remain higher in Aboriginal and Torres Strait Islander people than in other people. The predominant serogroup of *N. meningitidis* responsible for disease in both Aboriginal and Torres Strait Islander and other people is serogroup B, for which no vaccine is currently on the National Immunisation Program.

Disease trends

Over the 10-year period from 2000 to 2010, there has been a substantial decline in notifications of serogroup C meningococcal disease in both Aboriginal and Torres Strait Islander and other people (Figure 2.6.1). Comparing pre-vaccine (2000–2002) and post-vaccine (2008–2010) periods, notification rates declined by 74% in Aboriginal and Torres Strait Islander people and by 92% in other people. However, even in the pre-vaccine period, notifications for serogroup B meningococcal disease, for which no vaccine is currently available, were substantially higher than for serogroup C in both Aboriginal and Torres Strait Islander and other people. Notification rates for serogroup B meningococcal disease declined over the same period, but to a lesser extent than serogroup C, and for reasons probably unrelated to vaccination. Serogroup B notifications decreased by 38% in Aboriginal and Torres Strait Islander people and by 36% in other people. Despite the overall downward trends, notification rates remain higher in Aboriginal and Torres Strait Islander people than in other people for both serogroups.

Serogroup data quality has improved over time. The serogroup was recorded for 933 (86%) of the 1,079 notifications in this reporting period, compared with 738 (58%) of 1,263 notifications in the previous period (2003–2006). The serogroup was identified in 92% of the cases in Aboriginal and Torres Strait Islander people and 88% of cases in other people. While serogroup B was relatively more common in Aboriginal and Torres Strait Islander than other people in the pre-vaccine period (73% of cases vs 59% of cases), there was little difference in serogroup B distribution in the most recent period (82% vs 86%).

Notification data are presented for all jurisdictions for the period 2007–2010. Hospitalisation data are presented for six jurisdictions (all except Tasmania and the Australian Capital Territory) by financial year for the period July 2005 to June 2010.

A total of 1,079 notifications and 2,230 hospitalisations for meningococcal disease were recorded during these reporting periods (Table 2.6.1 and Table 2.6.2). Of these, 104 (9.6%) notifications and 189 (8.5%) hospitalisations were reported in Aboriginal and Torres Strait Islander people, which were similar to the proportions (8% for both notifications and hospitalisations) in the previous reporting period (2003–2006).

Notification and hospitalisation rates generally decrease with increasing age in both Aboriginal and Torres Strait Islander and other people. However, rates in 'other' young adults are slightly higher than in older children (Table 2.6.1 and Table 2.6.2).

Both notification and hospitalisation rates were higher in Aboriginal and Torres Strait Islander people than in other people across most age groups. However, in the 15–24 years age group, both the notification and

Table 2.6.1: Meningococcal disease notifications, all Australian states, 2007 to 2010, by age group and Indigenous status

Age group (years)	Indigenous status	Notifications* (2007–2010)		
		n	Rate†	Rate ratio
0–4	Indigenous	62	23.2	3.8§
	Other	322	6.1	
5–14	Indigenous	21	4.1	4.1§
	Other	105	1.0	
15–24	Indigenous	8	1.8	0.8
	Other	283	2.4	
25–49	Indigenous	9	1.3	2.7§
	Other	147	0.5	
≥50	Indigenous	4	1.5	3.4§
	Other	118	0.4	
All ages‡	Indigenous	104	3.2	2.7§
	Other	975	1.2	

* Notifications where the date of diagnosis was between 1 January 2007 and 31 December 2010.

† Average annual age-specific rate per 100,000 population.

‡ Includes cases with age unknown. Rates for all ages combined are age-standardised to the Australian Bureau of Statistics Australian population estimates for 2006.

§ Indicates statistically significant, 95% confidence intervals do not overlap 1.0

Table 2.6.2: Meningococcal disease hospitalisations, selected Australian states, 2005 to 2010, by age group and Indigenous status

Age group (years)	Indigenous status	Hospitalisations* (2005–2010)		
		n	Rate†	Rate ratio
0–4	Indigenous	127	40.5	3.5§
	Other	708	11.5	
5–14	Indigenous	32	5.2	2.3§
	Other	278	2.2	
15–24	Indigenous	13	2.6	0.7
	Other	494	3.6	
25–49	Indigenous	15	1.9	2.1§
	Other	316	0.9	
≥50	Indigenous	2	0.7	0.8
	Other	245	0.8	
All ages‡	Indigenous	189	4.5	2.2§
	Other	2,041	2.1	

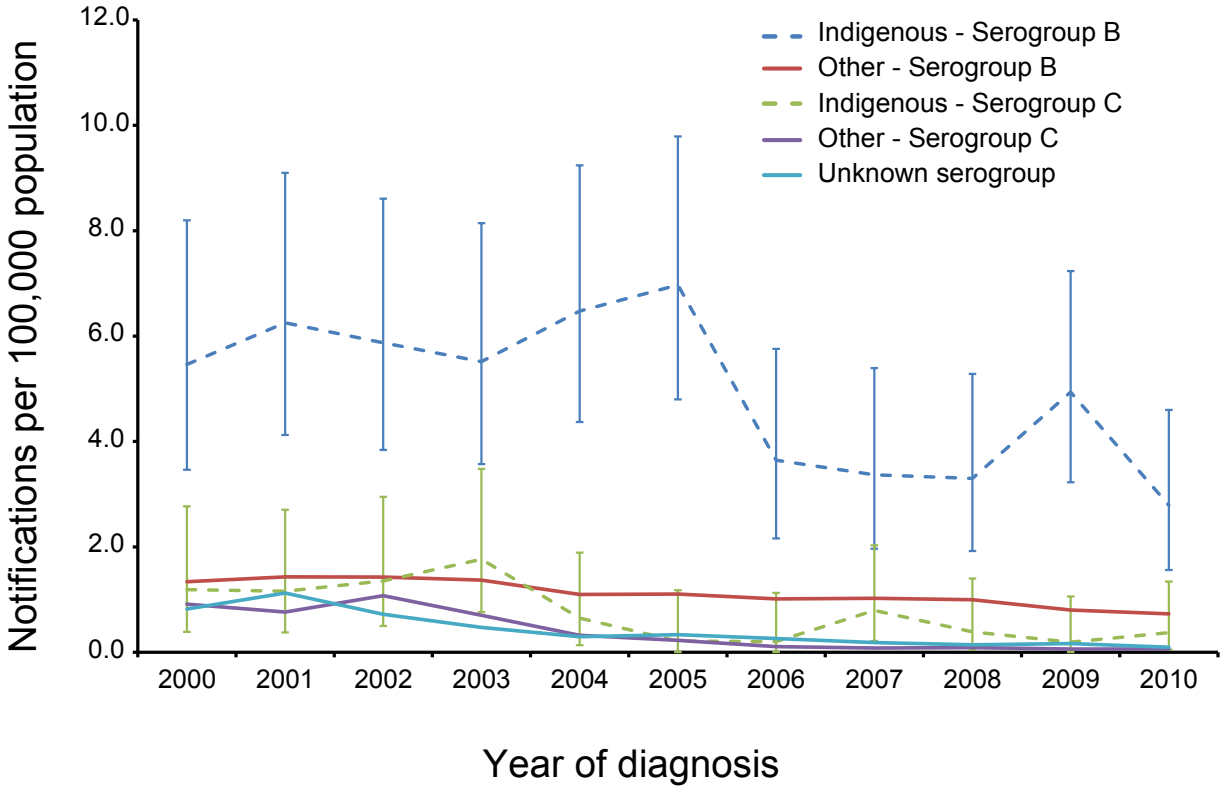
* Hospitalisations (New South Wales, Northern Territory, Queensland, South Australia, Victoria, Western Australia only) where the date of separation was between 1 July 2005 and 30 June 2010.

† Average annual age-specific rate per 100,000 population.

‡ Includes cases with age unknown. Rates for all ages combined are age-standardised to the Australian Bureau of Statistics Australian population estimates for 2006.

§ Indicates statistically significant, 95% confidence intervals do not overlap 1.0

Figure 2.6.1: Meningococcal disease notification rates and 95% confidence intervals, selected Australian states,* 2000 to 2010,† by Indigenous status and serogroup



* Jurisdictions with satisfactory data quality over the whole time period; see Appendix A (New South Wales, Northern Territory, Queensland, South Australia, Victoria, Western Australia).

† Notifications where the date of diagnosis was between 1 January 2000 and 31 December 2010.

hospitalisation rates were slightly higher in other people, although not statistically significantly so (Table 2.6.1 and Table 2.6.2). The highest rates were found in the 0–4 years age group. The overall Indigenous to non-Indigenous rate ratio was 2.7:1 for notifications and 2.2:1 for hospitalisations.

There were 42 deaths reported from the five jurisdictions (New South Wales, the Northern Territory, Queensland, South Australia and Western Australia) for the period 2006–2010 with meningococcal infection as the underlying cause and 45 deaths with meningococcal infection as either the underlying or a contributing cause. There were 1–4 deaths recorded with meningococcal infection as the underlying cause in Aboriginal and Torres Strait Islander people aged <5 years, 1–4 in those aged 5–49 years and none in those aged ≥50 years. The ABS provides ranges when absolute numbers of deaths are low.

Comment

Routine meningococcal C vaccination for infants and the high-school catch-up program, implemented from 2003, have resulted in a significant decrease in cases associated with serogroup C. However, the predominant serogroup responsible for disease in both Aboriginal and Torres Strait Islander and other people remains serogroup B, for which no vaccine is available, and for which the disease burden is higher in Aboriginal and Torres Strait Islander people.

Neisseria meningitidis is carried harmlessly in the nose and throat of approximately 10% of the population, with transmission via prolonged close contact. Conjugate meningococcal vaccines reduce carriage of meningococci, which allows for significant indirect benefits of herd immunity and reduced transmission.[47]

Disease risk has also been demonstrated in different countries to vary among different portions of their population. In the United Kingdom, invasive meningococcal disease incidence and mortality have been found to be

socially patterned, with the most deprived (20%) having twice the incidence rate than that of the most affluent quintile. In New Zealand, Maori and Pacific Islander people were found to have significantly higher rates of invasive meningococcal disease than the European population.[48]

A vaccine protecting against serogroup B disease, now licensed in Australia, could reduce the disparity between Aboriginal and Torres Strait Islander and other people and greatly reduce the overall meningococcal disease burden.

2.7 Mumps

Mumps is an acute viral disease caused by paramyxovirus. The classical presentation is parotitis, although up to 30% of cases will not have salivary gland involvement and may pose difficulty in diagnosis. Some complications of mumps have been known to occur at higher rates in adults than in children. Complications include orchitis, aseptic meningitis, encephalitis, sensorineural hearing loss and pancreatitis.[3]

Relevant vaccine history

1982

- MM* vaccine funded for all Australian infants at 12 months of age.

1984

- MM vaccination of Aboriginal and Torres Strait Islander children in the Northern Territory changed from 12 months to 9 months of age.

1989

- MMR† vaccine recommended and funded on the national schedule at 12 months of age (9 months for Aboriginal and Torres Strait Islander infants in the Northern Territory).

1994

- MMR funded as second dose of mumps-containing vaccine for adolescent females.

1996

- MMR funded as second dose of mumps-containing vaccine for all adolescents.

1998

- Recommended age for first dose of MMR vaccine for Aboriginal and Torres Strait Islander children in the Northern Territory increased from 9 months to 12 months of age.
- Recommended age for second dose of MMR vaccine lowered to 4–5 years.

2013

- Second dose moved forward to 18 months of age, given as MMRV.‡

Key points

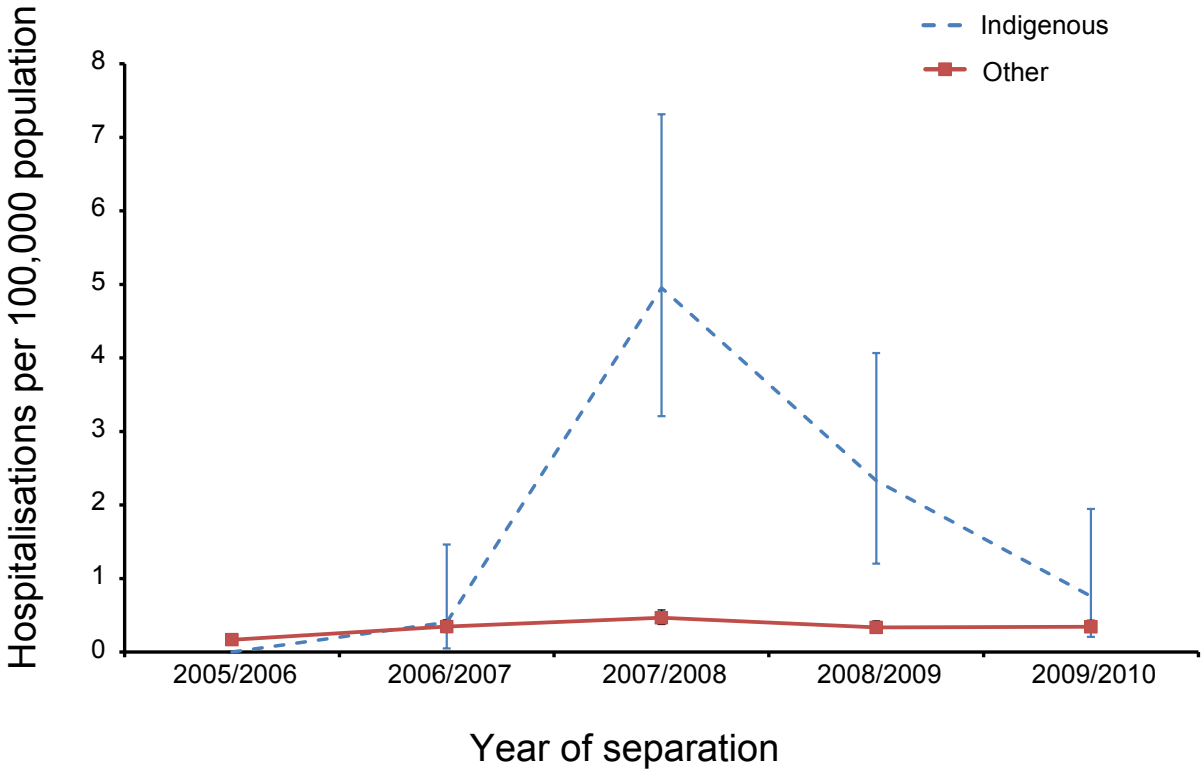
Despite high vaccination coverage in children, mumps outbreaks have been reported in Australia and overseas, predominantly in adolescents and young adults. One outbreak in Australia particularly affected Aboriginal and Torres Strait Islander people, but there was also a national increase in mumps in other adolescents and young adults. Mumps in these age groups more commonly results in serious disease and complications. A third dose of MMR vaccine for adolescents, to combat waning immunity, could be considered if outbreaks continue.

* MM: measles and mumps

† MMR: measles, mumps and rubella

‡ MMRV: measles, mumps, rubella and varicella

Figure 2.7.1: Mumps hospitalisation rates and 95% confidence intervals, selected Australian states,* 2005 to 2010,† by Indigenous status



* Jurisdictions with satisfactory data quality over the whole time period; see Appendix A (New South Wales, Northern Territory, Queensland, South Australia, Victoria, Western Australia).

† Hospitalisations where the date of separation was between 1 July 2005 and 30 June 2010.

Disease trends

Mumps notifications are not included here due to under-reporting of Indigenous status.

During this reporting period, a significant difference in hospitalisation rates was noted between Aboriginal and Torres Strait Islander and other people. There was a spike in the hospitalisation rate for Aboriginal and Torres Strait Islander people in 2007/2008, peaking at 5 per 100,000 (up from 0.4 per 100,000 in 2006/2007); the rates for other people also peaked in that year but were much lower (0.5 per 100,000 in 2007/2008, up from 0.2 per 100,000 in 2005/2006). The rates have since declined among Aboriginal and Torres Strait Islander people to less than 1 per 100,000 population in 2009/2010, which is comparable to the rate in other people (Figure 2.7.1).

Hospitalisation data are presented for six jurisdictions (all except Tasmania and the Australian Capital Territory) by financial year for the period July 2005 to June 2010. Notification data are not presented due to the low level of completeness of the Indigenous status field in notification records.

A total of 372 hospitalisations for mumps were recorded during this reporting period, of which 43 (13%) were reported in Aboriginal and Torres Strait Islander people (Table 2.7.1). The hospitalisation rates were higher in Aboriginal and Torres Strait Islander people than in other people across all age groups. The highest hospitalisation rates in Aboriginal and Torres Strait Islander people were in the 5–14 years age group (2.1 per 100,000 population), followed closely by the 25–49 years age group (2.0 per 100,000 population) indicating a shift in the affected age group from young children to adults since the introduction of vaccination. The overall Indigenous to non-Indigenous rate ratio was 5.1:1, with the highest rate ratio (7.3:1) in the 5–14 years age group.

Table 2.7.1: Mumps hospitalisations, selected Australian states, 2005 to 2010, by age group and Indigenous status

Age group (years)	Indigenous status	Hospitalisations* (2005–2010)		
		n	Rate†	Rate ratio
0–4	Indigenous	5	1.6	3.4§
	Other	29	0.5	
5–14	Indigenous	13	2.1	7.3§
	Other	36	0.3	
15–24	Indigenous	7	1.4	3.1§
	Other	63	0.5	
25–49	Indigenous	16	2.0	5.4§
	Other	132	0.4	
≥50	Indigenous	2	0.7	3.0§
	Other	69	0.2	
All ages‡	Indigenous	43	1.7	5.1§
	Other	329	0.3	

* Hospitalisations (New South Wales, Northern Territory, Queensland, South Australia, Victoria, Western Australia only) where the date of separation was between 1 July 2005 and 30 June 2010.

† Average annual age-specific rate per 100,000 population.

‡ Includes cases with age unknown.

§ Indicates statistically significant, 95% confidence intervals do not overlap 1.0

There were no deaths reported from the five jurisdictions (New South Wales, the Northern Territory, Queensland, South Australia and Western Australia) among Aboriginal and Torres Strait Islander people for the period 2006–2010 with mumps as either the underlying or a contributing cause.

Comment

During this reporting period (July 2005 to June 2010), a prolonged outbreak of mumps was recorded with a peak in hospitalisations in 2007/2008, which predominantly affected Aboriginal or Torres Strait Islander adolescents and young adults. This prolonged outbreak occurred predominantly among two epidemiologically linked Aboriginal populations in the Kimberley region (153 cases)[49] and the Northern Territory (99 cases).[50] Overall, the duration of the outbreak was about 40 weeks. Nearly half of the people affected had received fewer than 2 doses of vaccine (45% in the Northern Territory, 48% in Western Australia).[49,50] The cause of the outbreak was likely to be multifactorial, including social factors such as overcrowding leading to increased opportunities for virus transmission, waning immunity, and lower levels of immunity resulting from the adolescents and young adults having received their first dose of MMR vaccine at 9 months of age in the Northern Territory. The outbreak(s) occurred at the same time as a mumps resurgence in non-Indigenous adolescents and young adults across Australia.[24]

From 1984 to 1998, the initial dose of measles-and-mumps-containing vaccine was recommended at 9 months of age for Aboriginal and Torres Strait Islander children in the Northern Territory but at 12 months of age for non-Indigenous children in that state and for all children in the rest of Australia. This was because Aboriginal and Torres Strait Islander infants were thought to be more vulnerable to the measles epidemics of that era. While vaccination at 9 months of age provides earlier protection, there is a risk of poorer immune response due to immaturity of the immune system and interference by passive (maternal) antibodies. It is possible that this practice led to those children born between 1983 and 1997 in the Northern Territory being at increased risk of mumps due to being less fully protected, even by 2 vaccine doses.[50]

Over the past 6–7 years, mumps has made a re-emergence globally, including in the United States (US) which in 2006 experienced its largest outbreak since 1987.[51] Mumps outbreaks have been reported recently in several other countries including Canada, the United Kingdom (UK), the Netherlands, Israel, Moldova and Belarus.[52] As in Australia, the age group predominantly affected by the outbreaks in the US and the UK has been adolescents and young adults. In the US, multiple outbreaks in university residential communities were

attributed in part to the lower effectiveness of the mumps vaccine and high-density living.[53] This latter factor also applies to remote Aboriginal and Torres Strait Islander communities. Whereas mumps was historically a disease of childhood, the recent outbreaks predominantly involved young adults, nearly all of whom had a history of vaccination during childhood, most with the recommended 2-dose schedule. This evidence of waning immunity has led to suggestions that vaccination with a third dose during adolescence might be an effective measure to prevent outbreaks.[51] However, outbreaks in Australia and overseas have subsided without this being routinely implemented.

2.8 Pertussis

Pertussis (whooping cough) is caused by *Bordetella pertussis*, a Gram-negative bacterium.[3] It is characterised by an insidious onset of symptoms of minor upper respiratory infection, minimal fever and cough which becomes paroxysmal in 1–2 weeks. During the paroxysmal stage, the cough is most severe when the characteristic whoop occurs. Complications include suppurative otitis media, pneumonia, pulmonary hypertension, acute pertussis encephalopathy and nutritional deficiencies due to repeated vomiting.[3]

Relevant vaccine history

1942

- Pertussis vaccination programs started in most states/territories using 3 doses of whole-cell pertussis vaccine (Pw).

1975

- First national vaccination schedule recommended and funded 4 DTPw* doses for infants at 3, 4, 5 and 18 months of age.

1978

- Fourth dose removed from schedule (reinstated 1985).

1994

- Fifth dose added at 4–5 years of age.

1999

- DTPa† recommended and funded for all 5 childhood DTP doses.

2003

- 18-month booster replaced by adolescent dose; the eligible age group varied in different jurisdictions.

2008–2012

- dTpa‡ funded temporarily by various states and territories for parents/contacts of infants under cocoon strategy during an epidemic. Program timing and eligibility criteria differed between jurisdictions.

Key points

Pertussis continues to circulate, causing periodic epidemics in adolescents and adults and transmission to infants who are most vulnerable to severe disease. There is a disproportionate impact on Aboriginal and Torres Strait Islander infants. Timely administration of infant doses is very important. The first dose can now be given at 6 weeks of age.

* DTPw: diphtheria, tetanus and pertussis (whole-cell)

† DTPa: diphtheria, tetanus and pertussis (acellular)

‡ dTpa: diphtheria, tetanus and pertussis (acellular), reduced antigen content

Disease trends

Hospitalisation data (for the Northern Territory, Queensland, South Australia and Western Australia) are presented for the period July 1999 to June 2010. The 10-year trend shows a cyclical pattern of epidemics every 3–5 years. However, there has been an overall upward trend from baseline, especially in Aboriginal and Torres Strait Islander people (Figure 2.8.1).

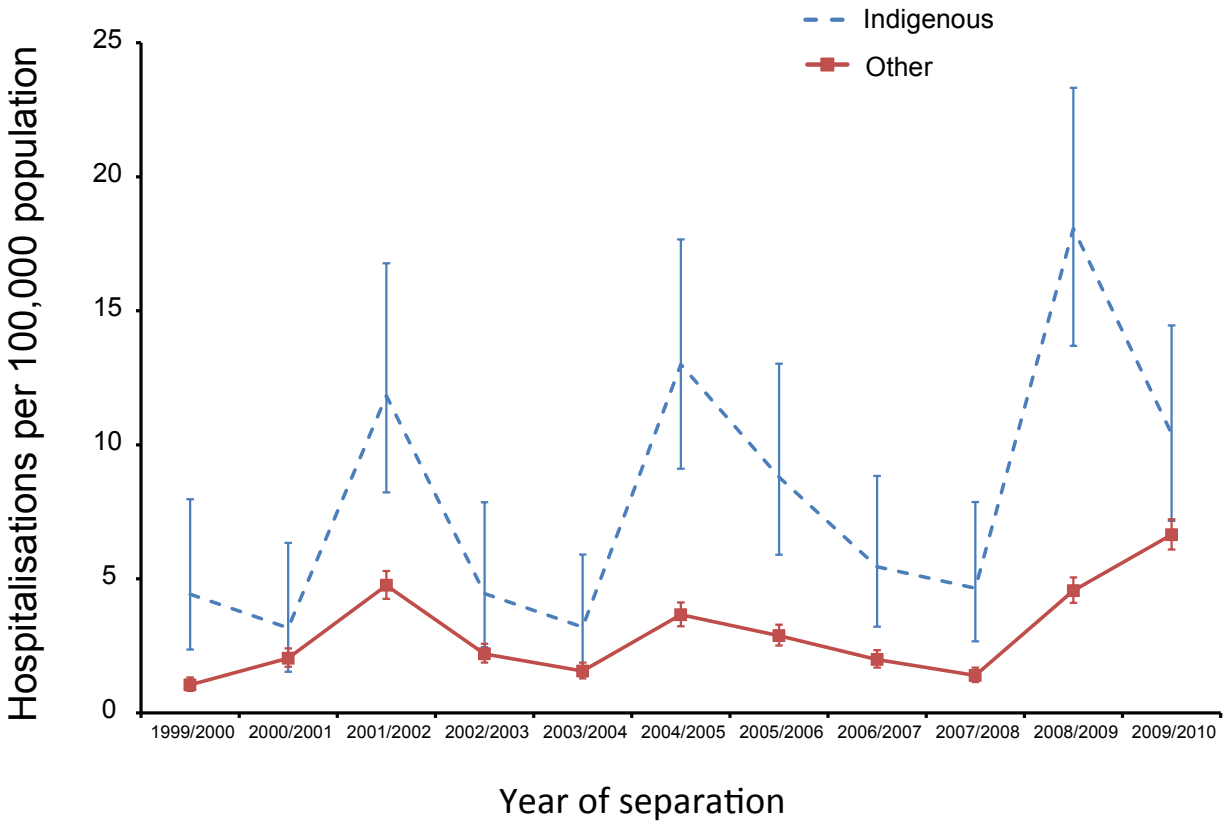
Hospitalisation data are presented for six jurisdictions (all except Tasmania and the Australian Capital Territory) by financial year for the period July 2005 to June 2010. Data for notifications are not presented due to the low level of completeness of the Indigenous status field in notification records.

A total of 3,772 hospitalisations for pertussis were recorded during this reporting period, of which 362 (9.6%) were reported in Aboriginal and Torres Strait Islander people (Table 2.8.1). The rates were higher in Aboriginal and Torres Strait Islander people than in other people across all age groups. The highest hospitalisation rates occurred in the youngest age group (0–4 years) especially in Aboriginal and Torres Strait Islander children (93.2 per 100,000). The overall Indigenous to non-Indigenous rate ratio was 2.9:1, with the highest ratio (3.3:1) in the 0–4 years and 15–24 years age groups.

Those ≥50 years of age have the second highest hospitalisation rates. This pattern is similar for both Aboriginal and Torres Strait Islander and other people.

There were 10 deaths reported from the five jurisdictions (New South Wales, the Northern Territory, Queensland, South Australia and Western Australia) for the period 2006–2010 with pertussis as the underlying cause and 17 deaths with pertussis as either the underlying or a contributing cause. There were 1–4 deaths reported in Aboriginal and Torres Strait Islander people with pertussis as either the underlying or a contributing cause (the ABS provides ranges when absolute numbers of deaths are low).

Figure 2.8.1: Pertussis hospitalisation rates and 95% confidence intervals, selected Australian states,* 1999 to 2010,† by Indigenous status



* Jurisdictions with satisfactory data quality over the whole time period; see Appendix A (New South Wales, Queensland, South Australia, Western Australia).

† Hospitalisations where the date of separation was between 1 July 1999 and 30 June 2010. Rates are age-standardised to the Australian Bureau of Statistics Australian population estimates for 2006.

Table 2.8.1: Pertussis hospitalisations, selected Australian states, 2005 to 2010, by age group and Indigenous status

Age group (years)	Indigenous status	Hospitalisations* (2005–2010)		
		n	Rate†	Rate ratio
0–4	Indigenous	292	93.2	3.3§
	Other	1,748	28.4	
5–14	Indigenous	13	2.1	1.8§
	Other	149	1.2	
15–24	Indigenous	10	2.0	3.3§
	Other	83	0.6	
25–49	Indigenous	25	3.1	2.7§
	Other	420	1.2	
≥50	Indigenous	22	7.3	2.2§
	Other	1,010	3.3	
All ages‡	Indigenous	362	10.0	2.9§
	Other	3,410	3.5	

* Hospitalisations (New South Wales, Northern Territory, Queensland, South Australia, Victoria, Western Australia only) where the date of separation was between 1 July 2005 and 30 June 2010.

† Average annual age-specific rate per 100,000 population.

‡ Includes cases with age unknown. Rates for all ages combined are age-standardised to the Australian Bureau of Statistics Australian population estimates for 2006.

§ Indicates statistically significant, 95% confidence intervals do not overlap 1.0

Comment

Pertussis is the least well controlled of all vaccine preventable diseases with long-standing, well-established vaccination programs. It has the highest notification rates in all age groups for the total Australian population, and higher hospitalisation rates than most other vaccine preventable diseases. Epidemics continue to occur which affect both Aboriginal and Torres Strait Islander and other people, although Aboriginal and Torres Strait Islander people have higher hospitalisation rates. Epidemics result in higher rates of hospitalisation across all age groups, but of particular concern are the rates for infants <1 year of age.

The latest pertussis epidemic commenced midway through the second half of 2008 and spread across all Australian jurisdictions. A recent resurgence in pertussis has been observed in many countries in North America and Europe.[54-56] Increases have been most marked in adolescents and adults in whom the disease is less severe, but infant deaths have also occurred.[55] The most important factors contributing to increased notifications are thought to be improved diagnosis and surveillance, increased awareness of pertussis in adolescents and adults, lower effectiveness of newer (acellular) vaccines,[57] and pathogen strain shifts.[58] The higher sensitivity of molecular tests (PCR) compared to serology and culture, which were the only tests widely available several years ago, contributes to increased diagnosis and reporting. There was also an increase in presentation and testing associated with concern about pandemic influenza in 2009. Research in the Netherlands noted that an increase in notifications, a changing age demographic towards older age groups and increasing disease severity were associated with changes in circulating pertussis strains which expressed increased pertussis toxin production.[58]

In order to protect those who are most vulnerable to severe disease, from 2008 to 2012 DTPa vaccination was funded by various states and territories for parents/contacts of infants under the 'cocoon' strategy. Parents are also now encouraged to have the infant's first vaccination given at 6 weeks of age, instead of the usual 2 months. Timely vaccination of the 4- and 6-month doses is also very important.

2.9 Pneumococcal disease

Pneumococcal disease is caused by *Streptococcus pneumoniae* (pneumococcus), a Gram-positive bacterium. Over 90 capsular antigenic types (serotypes) of this organism have been identified, but only a limited number cause the majority of pneumococcal disease.[59,60] Pneumococci colonise the mucosal surface of the upper respiratory tract with no apparent symptoms in children and less commonly in adults (nasopharyngeal carriage).[3,59] From the nasopharynx, pneumococci may spread locally to cause sinusitis or otitis media, or by

inhalation into the lungs to cause pneumonia. Pneumococci can also enter the bloodstream to cause severe systemic disease such as bacteraemia, meningitis and, rarely, infection in remote sites such as joints, bones and soft tissues.[3,61,62] Invasive pneumococcal disease (IPD) is the clinical condition in which *Streptococcus pneumoniae* is isolated from blood, cerebrospinal or pleural fluid, or other normally sterile sites. In the absence of a sterile site isolate, presumptive diagnosis of pneumococcal pneumonia is often made on the

Relevant vaccine history

1986

- 23vPPV funded for children aged over 2 years with increased risk of pneumococcal disease or complications, due to specified underlying conditions, living in north Western Australia and the Northern Territory.

1991–1993

- 23vPPV funded for all Aboriginal and Torres Strait Islander people aged over 2 years living in north Western Australia.

1995–1996

- 23vPPV funded for Aboriginal and Torres Strait Islander people aged ≥50 years in the Northern Territory (1995) and Far North Queensland (1996, including people 15–49 years with underlying conditions).

1997

- 23vPPV recommended for all Aboriginal and Torres Strait Islander adults aged >50 years.

1998

- 23vPPV funded for Aboriginal and Torres Strait Islander adults aged >50 years and other adults aged >65 years in Victoria.

1999

- 23vPPV funded nationally for all Aboriginal and Torres Strait Islander adults aged ≥50 years or aged 15–49 years with underlying conditions.

2000

- 23vPPV eligibility in the Northern Territory changed to all Aboriginal and Torres Strait Islander people aged ≥15 years. 23vPPV eligibility in central Australia changed to all Aboriginal and Torres Strait Islander children aged 2–5 years.

2001

- 7vPCV funded for all Aboriginal and Torres Strait Islander infants. A booster dose of 23vPPV was funded in the Northern Territory, Queensland, South Australia and Western Australia.

2005

- 7vPCV funded for all children and 23vPPV funded for all adults aged ≥65 years.

2009

- The Northern Territory replaced 7vPCV and 23vPPV in the routine childhood vaccination schedule with the 10vPCV.

2011

- 13vPCV replaced all other pneumococcal vaccines for all children aged <2 years.

Key points

Substantial decreases in invasive disease and reductions in hospitalised pneumonia in some settings have been seen in Aboriginal and Torres Strait Islander children following 7vPCV vaccine introduction. No impact on otitis media has been reported. Impacts of vaccination on Aboriginal and Torres Strait Islander adults are less clear. Reducing vaccination delay in infants and improving coverage in adults are important to maximise the benefits of the existing vaccines.

basis of isolation of the organism in sputum and/or other clinical or radiological features such as characteristic chest X-ray appearance.[63] Acute otitis media is a much more common and less severe non-invasive manifestation of pneumococcal disease in children.[64]

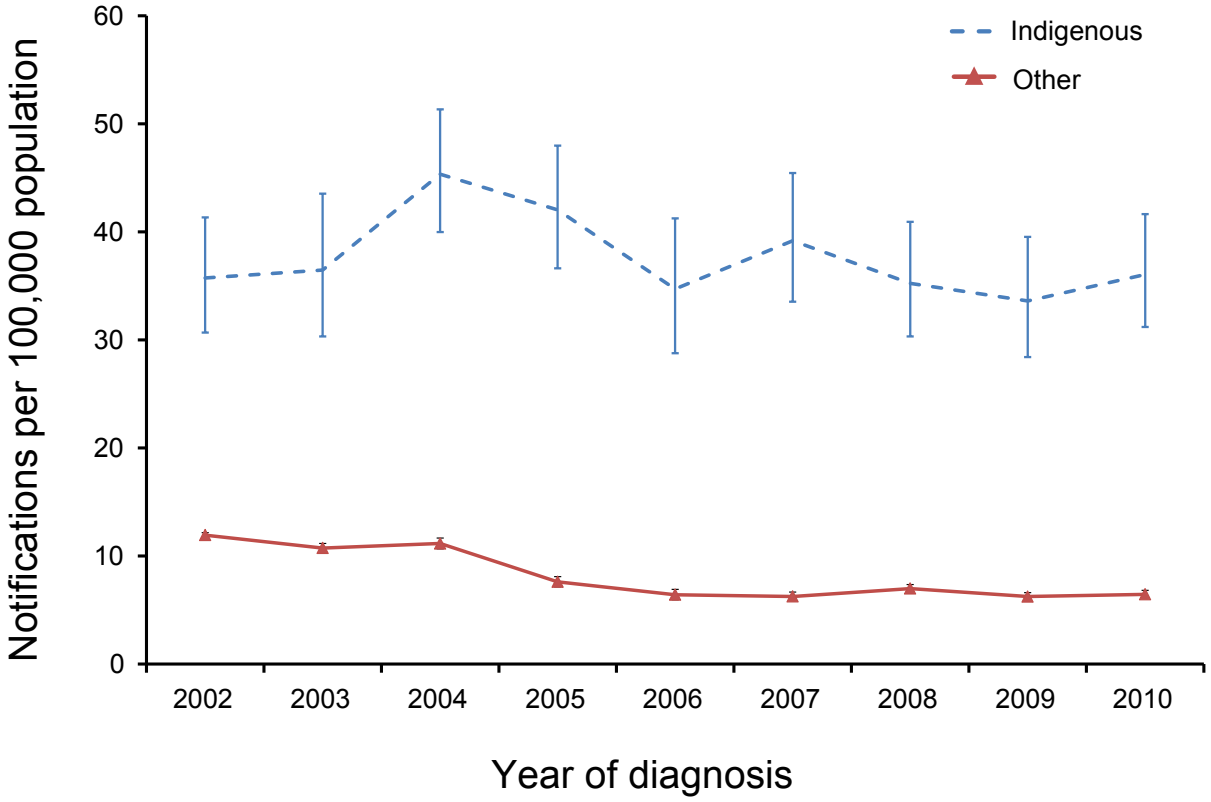
Vaccination programs against pneumococcal disease in Australia over the years has addressed the increased pneumococcal disease burden among Aboriginal and Torres Strait Islander people.[65] When funded pneumococcal vaccination programs using 23-valent polysaccharide vaccine (23vPPV) and the 7-valent conjugate vaccine (7vPCV) were introduced in Australia, they initially targeted only Aboriginal and Torres Strait Islander people.

Disease trends

Notification data for IPD are complete from 2002. Over the 8-year period from January 2002 to December 2010, the rates of total IPD notifications (all ages reported from selected jurisdictions) remained much higher among Aboriginal and Torres Strait Islander people than among other people (Figure 2.9.1), with no clear overall trend. IPD notification rates for other people declined sharply from 2005 onwards, the year in which universal childhood pneumococcal conjugate vaccination and adult 23vPPV vaccination were introduced. Annual rates of total IPD notifications remained stable during 2008–2010 among both Aboriginal and Torres Strait Islander and other people.

In 2002–2004, there was initially a decline in the age-specific IPD notification rates in Aboriginal and Torres Strait Islander children in the 0–4 years age group, to almost equal the rates in other children in the same age

Figure 2.9.1: Invasive pneumococcal disease notification rates and 95% confidence intervals, selected Australian states,* 2002 to 2010,† by Indigenous status



* Jurisdictions with satisfactory data quality over the whole time period; see Appendix A (New South Wales, Northern Territory, Queensland, South Australia, Victoria, Western Australia).

† Notifications where the date of diagnosis was between 1 January 2002 and 31 December 2010. Rates are age-standardised to the Australian Bureau of Statistics Australian population estimates for 2006.

Table 2.9.1: Invasive pneumococcal disease notifications, all Australian states, 2007 to 2010, by age group and Indigenous status

Age group (years)	Indigenous status	Notifications* (2007–2010)		
		n	Rate†	Rate ratio
0–4	Indigenous	137	51.2	2.9§
	Other	925	17.4	
5–14	Indigenous	64	12.4	5.5§
	Other	237	2.3	
15–24	Indigenous	47	10.7	6.3§
	Other	200	1.7	
25–49	Indigenous	306	44.7	11.8§
	Other	1,143	3.8	
≥50	Indigenous	144	53.3	4.6§
	Other	3,100	11.6	
All ages‡	Indigenous	698	42.0	3.6§
	Other	5,606	11.5	

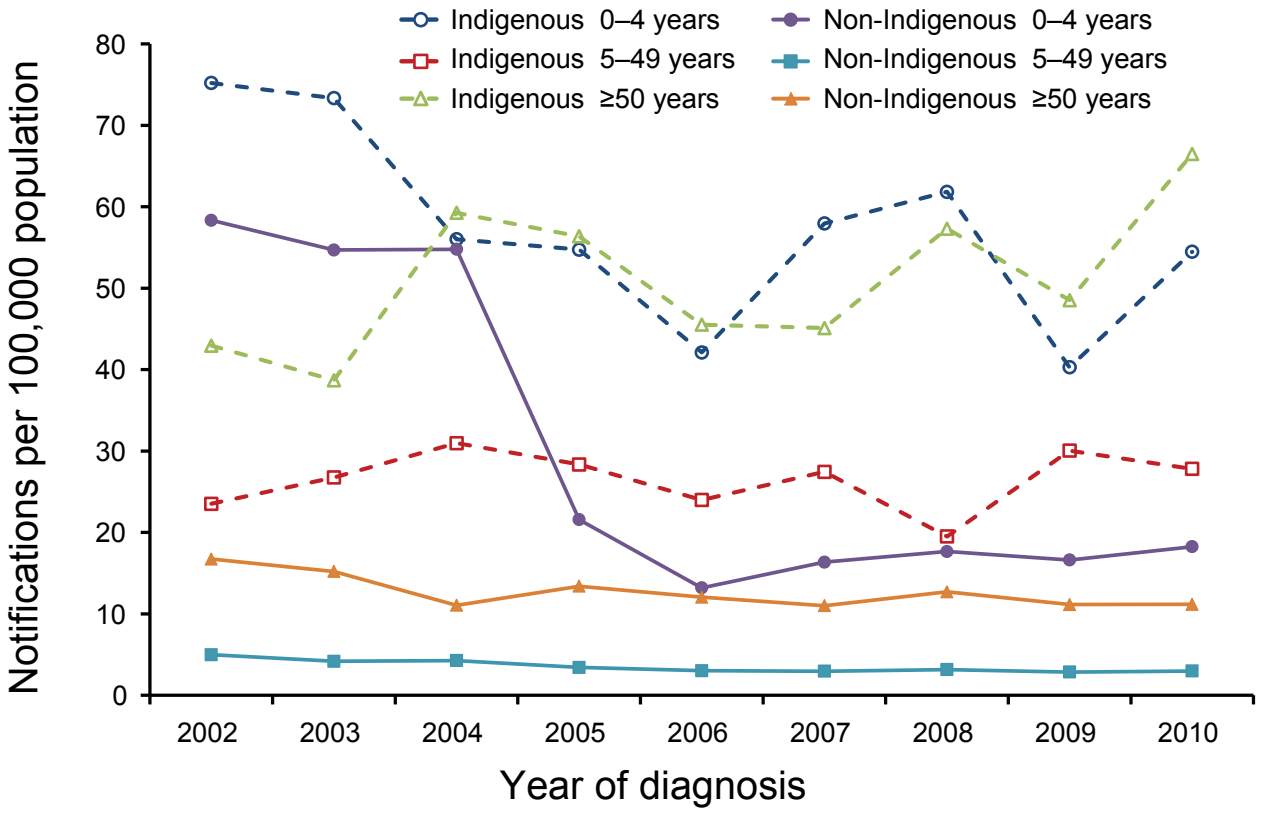
* Notifications where the date of diagnosis was between 1 January 2007 and 31 December 2010.

† Average annual age-specific rate per 100,000 population.

‡ Includes cases with age unknown. Rates for all ages combined are age-standardised to the Australian Bureau of Statistics Australian population estimates for 2006.

§ Indicates statistically significant, 95% confidence intervals do not overlap 1.0

Figure 2.9.2: Invasive pneumococcal disease notification rates, selected Australian states,* 2002 to 2010,† by age group and Indigenous status



* Jurisdictions with satisfactory data quality over the whole time period; see Appendix A (New South Wales, Northern Territory, Queensland, South Australia, Victoria, Western Australia).

† Notifications where the date of diagnosis was between 1 January 2002 and 31 December 2010.

Table 2.9.2: Invasive pneumococcal disease hospitalisations, selected Australian states, 2005 to 2010, by age group and Indigenous status

Age group (years)	Indigenous status	Hospitalisations* (2005–2010)		
		n	Rate†	Rate ratio
0–4	Indigenous	83	26.5	3.7§
	Other	436	7.1	
5–14	Indigenous	32	5.2	5.2§
	Other	124	1.0	
15–24	Indigenous	15	3.0	4.3§
	Other	97	0.7	
25–49	Indigenous	195	24.5	14.2§
	Other	613	1.7	
≥50	Indigenous	72	24.0	3.8§
	Other	1,948	6.3	
All ages‡	Indigenous	397	18.9	6.0§
	Other	3,218	3.2	

* Hospitalisations (New South Wales, Northern Territory, Queensland, South Australia, Victoria, Western Australia only) where the date of separation was between 1 July 2005 and 30 June 2010. The ICD-10-AM codes used to identify hospitalisations were G00.1 (pneumococcal meningitis) and A40.3 (pneumococcal septicaemia) (together considered to be a proxy for IPD).

† Average annual age-specific rate per 100,000 population.

‡ Includes cases with age unknown. Rates for all ages combined are age-standardised to the Australian Bureau of Statistics Australian population estimates for 2006.

§ Indicates statistically significant, 95% confidence intervals do not overlap 1.0

group. But from 2005 onwards, IPD notification rates in Aboriginal and Torres Strait Islander children and other children diverged, with no clear trend in the former and a very steep decline in the latter (Figure 2.9.2). IPD notification rates in the ≥50 years age group also show contrasting trends between the two groups with an overall increase in Aboriginal and Torres Strait Islander people and a slow decline in other people. Among Aboriginal and Torres Strait Islander adults aged ≥50 years, there was a 58% increase in total IPD notifications from 2002 to 2010.

Notification data are presented for all jurisdictions for the period 2007–2010. Hospitalisation data are presented for six jurisdictions (all except Tasmania and the Australian Capital Territory) by financial year for the period July 2005 to June 2010.

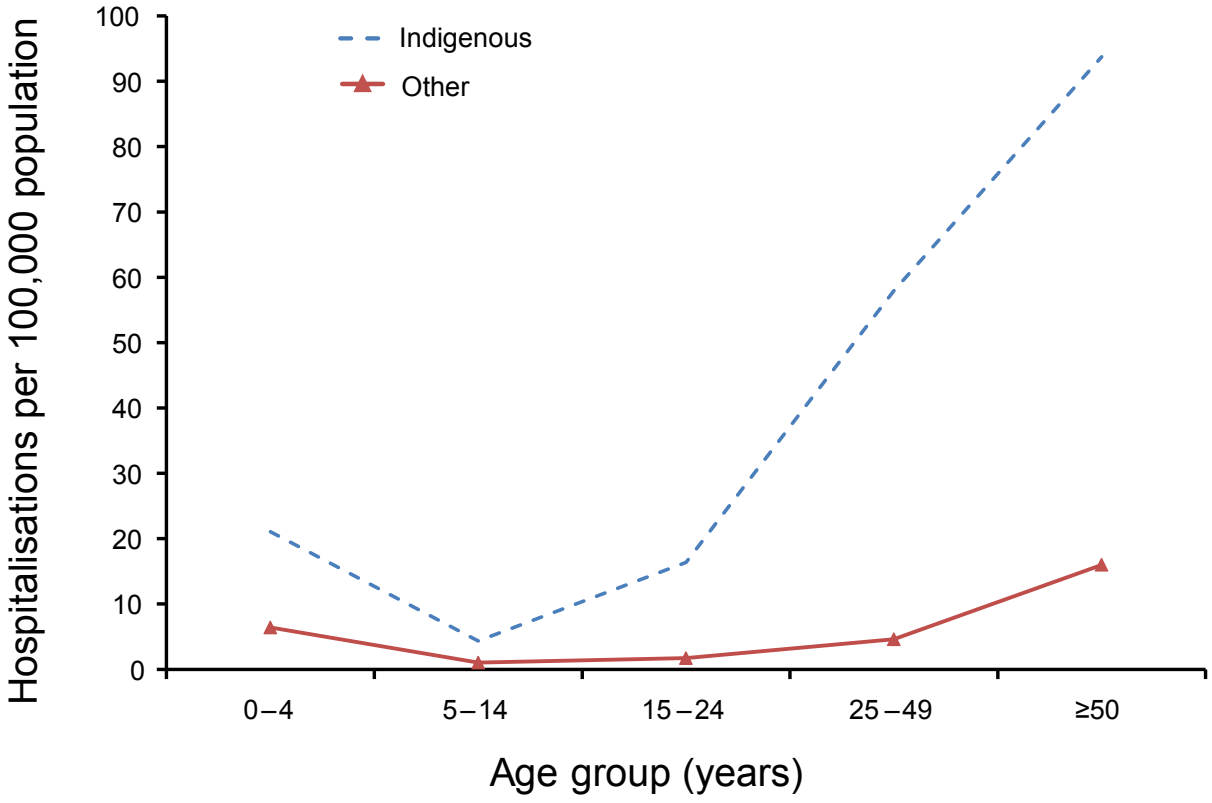
A total of 6,304 notifications of IPD were recorded during this reporting period, of which 698 (11%) were reported in Aboriginal and Torres Strait Islander people (Table 2.9.1). A total of 3,615 hospitalisations coded as pneumococcal meningitis or septicaemia (a proxy for IPD) were recorded during this reporting period, of which 397 (11%) were reported in Aboriginal and Torres Strait Islander people (Table 2.9.2).

The rates of notifications and the number of hospitalisations for IPD in both Aboriginal and Torres Strait Islander and other people were high in the 0–4 years and ≥50 years age groups and lowest in older children and young adults (Table 2.9.1 and Table 2.9.2). Among Aboriginal and Torres Strait Islander people, there was a marked rise in IPD rates from the 15–24 years to the 25–49 years age group, and the highest age-specific IPD notification rate was in the ≥50 years age group. The overall Indigenous to non-Indigenous rate ratios were 3.6:1 for notifications and 6.0:1 for hospitalisations, with the highest ratios (11.8:1 and 14.2:1, respectively) in the 25–49 years age group.

The rates of hospitalisations coded as pneumococcal pneumonia (without codes for pneumococcal meningitis and septicaemia) show hospitalisation rates several-fold higher than for IPD in Aboriginal and Torres Strait Islander adults and the elderly, showing the substantially higher overall disease burden due to *S. pneumoniae* in this group (Figure 2.9.3).

Serotype data were available for 93% (5,858) of the isolates from all IPD notifications. The notification rates of IPD caused by 7vPCV types declined in all age groups in the post-universal vaccination period (2006–

Figure 2.9.3: Pneumococcal pneumonia (not coded as meningitis or septicaemia) hospitalisation rates, selected Australian states,* 2005 to 2010,† by age group and Indigenous status



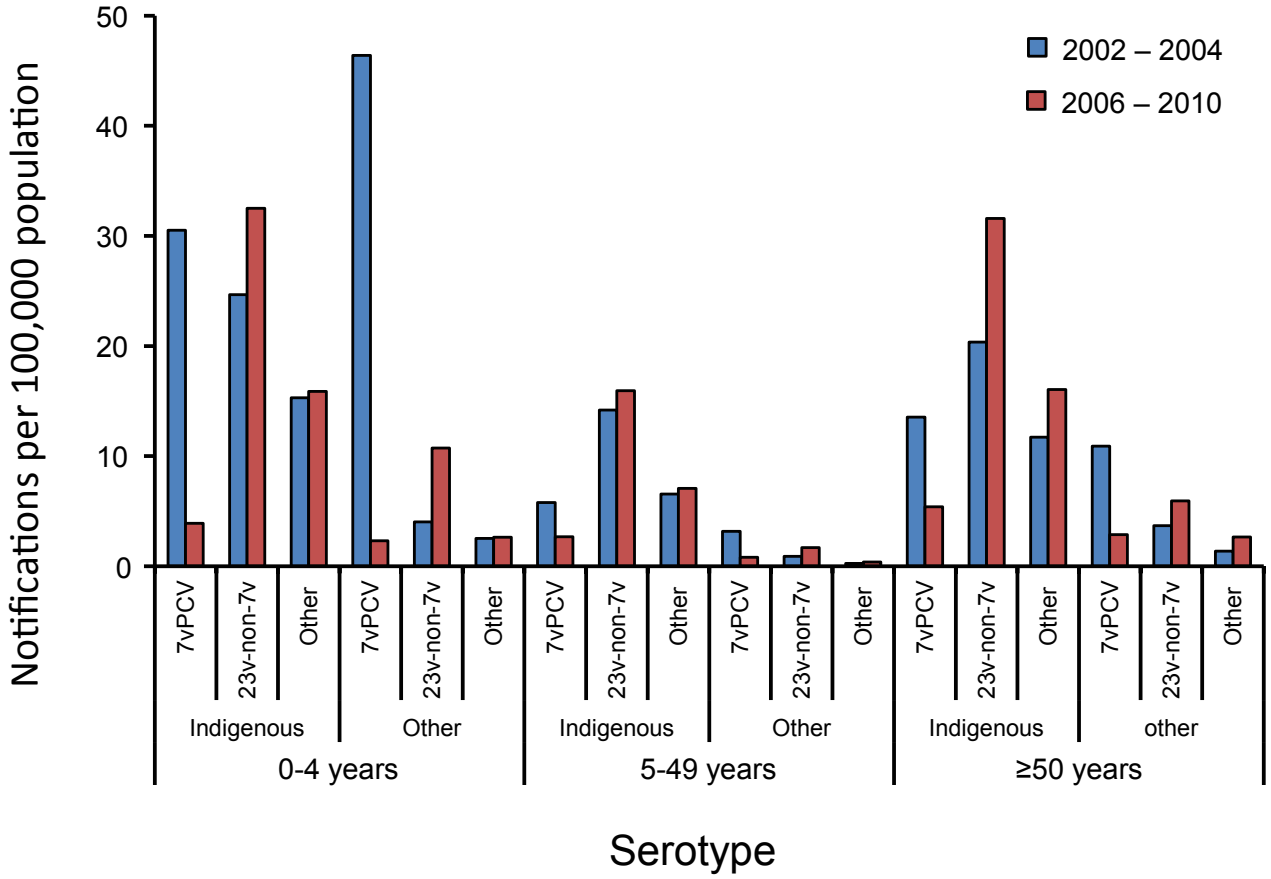
* New South Wales, Northern Territory, Queensland, South Australia, Victoria, Western Australia.

† Hospitalisations where the date of separation was between 1 July 2005 and 30 June 2010. The ICD-10-AM code used to identify hospitalisations was J13 (pneumococcal pneumonia).

2010) compared with the pre-universal vaccination period (2002–2004) in both Aboriginal and Torres Strait Islander and other people (Figure 2.9.4). This decline was most pronounced in the 0–4 years age group (87%). There was a concomitant increase in IPD caused by serotypes contained in 23vPPV but not in 7vPCV (23v-non-7) in all age groups, with the greatest fold increase seen in other children. The proportion of all IPD that was due to 7vPCV types in the 0–4 years age group in 2006–2010 was 6% in Aboriginal and Torres Strait Islander children and 9% in other children. In the same age group, the proportion of serotypes contained in the 13vPCV was significantly less among Aboriginal and Torres Strait Islander children (38%) than among other children (64%, $P<0.0001$). The main contributor to this difference was serotype 19A, which accounted for only 15% of all IPD in Aboriginal and Torres Strait Islander children but 46% in other children. However, the rate of serotype 19A in both groups of children was the same (8 per 100,000). Among adults ≥50 years of age, the proportion of disease caused by serotypes contained in the 23vPPV was similar (68% in Aboriginal and Torres Strait Islander people and 70% in other people).

There were 282 deaths reported to the Australian Bureau of Statistics from five jurisdictions (New South Wales, the Northern Territory, Queensland, South Australia and Western Australia) for the period 2006–2010 with IPD or 'pneumococcal disease' listed as the underlying or a contributing cause, of which 18–30 were reported in Aboriginal and Torres Strait Islander people (the ABS provides ranges when absolute numbers of deaths are low). 'Pneumococcal disease' was primarily the cause in adults (14/15–18), while IPD was more evenly distributed (1–4 each in <5 years, 5–49 years and ≥50 years age groups). Of the total 282 deaths, in 109 IPD (meningitis/septicaemia) or pneumococcal pneumonia was the underlying cause of death. There were 1–4 deaths reported in Aboriginal and Torres Strait Islander people with IPD as the underlying cause in each of the <5 years, 5–49 years and ≥50 years age groups, and 1–4 with pneumococcal pneumonia as the underlying cause in each of the 5–49 years and ≥50 years age groups.

Figure 2.9.4: Invasive pneumococcal disease notification rates for vaccine serotype groups, selected Australian states,* 2002–2004 compared with 2006–2010, by age group and Indigenous status



* New South Wales, Northern Territory, Queensland, South Australia, Victoria, Western Australia.

In national notifications data for the same period and jurisdictions, there were 575 deaths reported as due to IPD, with 6% (n=34) of these in Aboriginal and Torres Strait Islander people. There were 30 deaths in children aged <5 years recorded in the national notifications data; of these, 5 (17%) were in Aboriginal and Torres Strait Islander children.

Comment

The period covered by this report encompasses several years during which pneumococcal vaccination was publicly funded among Aboriginal and Torres Strait Islander as well as other children and adults. Therefore, the data presented here reflect the ongoing effects of those vaccination programs, predominantly using 7vPCV in children and 23vPPV in adults, on pneumococcal disease hospitalisations and notifications.

During this reporting period, sustained reductions in the notification rates for IPD caused by 7vPCV serotypes were seen among both Aboriginal and Torres Strait Islander and other people across all age groups, following the introduction of their respective childhood 7vPCV vaccination programs. The most dramatic decline in 7vPCV-type IPD was seen in other children. A rapid reduction in 7vPCV-type IPD was evident within a year of the introduction of universal vaccination, consequent to rapid achievement of high coverage. In comparison, there was a slower decline and a persistently higher IPD incidence in Aboriginal and Torres Strait Islander children. This is mainly due to the fact that the proportion of IPD caused by the 7vPCV types prior to vaccine introduction was lower in Aboriginal and Torres Strait Islander children than in other children.[66-69]

During the reporting period, the rates of total IPD hospitalisations and notifications were higher in Aboriginal and Torres Strait Islander people than in other people in all age groups. The Indigenous to non-Indigenous rate ratios continued to be lowest in those age groups directly targeted for vaccination where the greatest benefits were expected (i.e. 0–4 years and ≥50 years). Over the years, rates of IPD caused

by 23v-non-7v types has increased to varying degrees in all age groups in both Aboriginal and Torres Strait Islander and other people. Serotype 19A was largely responsible for most of this serotype replacement disease among other children as in other developed countries.[70-73] The increase in 19A was much less marked in Aboriginal and Torres Strait Islander children.[74-76] It is possible but unproven that the childhood booster dose of 23vPPV was responsible for this effect. Among children in different population groups of Native Americans with similarly high levels of exposure to 7vPCV (without a 23vPPV booster) the changes in non-7v type disease varied considerably.[77-79] Therefore there is not a consistent pattern of post-vaccination serotype changes in populations with high levels of pre-existing non-7vPCV IPD incidence. An outbreak of serotype 1 IPD that started in 2010 contributed to 23v-non-7v type disease increases in older Aboriginal and Torres Strait Islander children in central Australia.[80]

The rates of IPD in Aboriginal and Torres Strait Islander adults in the 25–49 years age group were 12–14 times the rates among other people in this age group for this reporting period, as they were in 2002–2005.[13] This is partly a reflection of the greater disparity in the prevalence of risk factors associated with IPD, including smoking, chronic disease and heavy alcohol consumption, between Aboriginal and Torres Strait Islander and other people in this age group.[81,82] The effectiveness of 23vPPV is limited among those with risk factors that include immunosuppression.[83] There has been a remarkable decline in 7vPCV-type IPD in all adult age groups due to the indirect effect from the childhood vaccination program. However, the direct impact of 23vPPV in adults has been less clear in both Aboriginal and Torres Strait Islander and other people. Possible benefits of 23vPPV vaccination have been limited by vaccination coverage (from the few available data) being quite low in this age group.[13] In Western Australian Aboriginal and Torres Strait Islander adults, the rate of IPD was higher in 2005–2007 than in 1997–2001, mainly due to 23v-non-7v types.[69] Rates of all IPD and IPD due to 23v-non-7v types including 19A remained unchanged among Aboriginal and Torres Strait Islander adults in North Queensland and the Northern Territory, although there has been an increase in the range of non-vaccine serotypes causing IPD.[75,76] As some of these serotypes are included in the 13vPCV, the indirect effect from 13vPCV use in children would be expected to result in some benefit to Aboriginal and Torres Strait Islander adults. The 13vPCV is now registered for use for adults ≥50 years of age, but it is not publicly funded and uptake is expected to be low. The results of ongoing large-scale studies of 13vPCV among adults will be vital for making policy decisions on providing public funding for 13vPCV for adults.

Hospitalisations coded as pneumococcal pneumonia are a potential indicator of the burden of non-invasive pneumococcal infections, and changes in the rates of these hospitalisations may reflect the effect of pneumococcal vaccines on non-invasive disease.[24] Several clinical trials have shown that pneumococcal conjugate vaccines are effective in preventing radiologically confirmed as well as clinically diagnosed pneumonia in children, although the greatest effect was on IPD in children.[84,85] The hospitalisation rates for pneumococcal pneumonia were lower in this reporting period than in 2002–2005, with the reduction most pronounced in the 0–4 years age group for both the Aboriginal and Torres Strait Islander and other children.[13] There has been a sustained reduction in hospitalisations coded as pneumonia in Aboriginal and Torres Strait Islander children <2 years of age in Western Australia, initially following targeted pneumococcal vaccination in these children and more pronounced after the introduction of the universal 7vPCV program.[86,87] However, among Aboriginal and Torres Strait Islander children in the Northern Territory, 7vPCV and 23vPPV effectiveness assessments failed to demonstrate a significant effect against either hospitalisations for acute lower respiratory tract infections or radiologically confirmed pneumonia.[88,89] Also, while there has been a reduction in the number of hospital procedures for otitis media in non-Indigenous children, no such impact has been detected for Aboriginal and Torres Strait Islander children.[90,91] Lower serotype coverage of 7vPCV, greater diversity of pathogens, and suboptimal timeliness of vaccination in Aboriginal and Torres Strait Islander children may have limited the pneumococcal vaccine impact in these children.[88,89,92,93] The higher valency vaccines (10vPCV and 13vPCV) that replaced 7vPCV may result in a greater effect on non-invasive as well as invasive disease.

There have been substantial overall reductions in IPD and some impact on pneumonia hospitalisations in Aboriginal and Torres Strait Islander children since the introduction of vaccination. Impacts in Aboriginal and Torres Strait Islander adults have been less obvious. Due to the wide serotype distribution of IPD in Aboriginal and Torres Strait Islander people, the six more serotypes covered by 13vPCV could provide considerable additional benefit to Aboriginal and Torres Strait Islander children and adults. This could include the prevention of serotype 1 outbreaks such as the one that occurred recently in Aboriginal and Torres Strait Islander people in central Australia. However, in the post-7vPCV era, the contribution of 13v-non-7v types to all IPD, and therefore the expected benefits from the 13vPCV, are much less in Aboriginal and Torres Strait Islander people than in other people, largely due to differences in distribution of serotype 19A.

Pneumococcal conjugate vaccines with even broader serotype coverage or novel protein-based serotype-independent pneumococcal vaccines would be required to eliminate the continuing disparity between Aboriginal and Torres Strait Islander people and other people.[94] It is important to continue surveillance of IPD in Aboriginal and Torres Strait Islander people to detect changes in serotype distribution and to match vaccines to disease-causing serotypes.

2.10 Rotavirus

Rotavirus is a common cause of gastrointestinal infection in young children; almost all children will acquire infection and antibodies by the age of 3 years.[3] The severity of rotavirus infection is age dependent. Infections are more likely to be severe in children 3–24 months of age. Rotavirus infection can range from asymptomatic infection to mild diarrhoea to severe gastroenteritis with dehydration. Most disease is mild but about 1 in 75 children will develop severe disease causing dehydration.[3]

Relevant vaccine history

2006

- Vaccination recommended and funded for infants in the Northern Territory using monovalent rotavirus vaccine in a 2-dose schedule (2 and 4 months of age).

2007

- Funded national immunisation commenced, with each state and territory using either a 2-dose schedule of monovalent rotavirus vaccine (2 and 4 months of age) or a 3-dose schedule of pentavalent rotavirus vaccine (2, 4 and 6 months of age).

Key points

Overall, there has been a substantial decline in hospitalisations from rotavirus since the introduction of the Australian rotavirus immunisation program. The decline in Aboriginal and Torres Strait Islander people has been less than that in other people. Low vaccination coverage due to delayed vaccination and upper age limits for rotavirus vaccination, and low vaccine effectiveness for some circulating genotypes may be contributing factors.

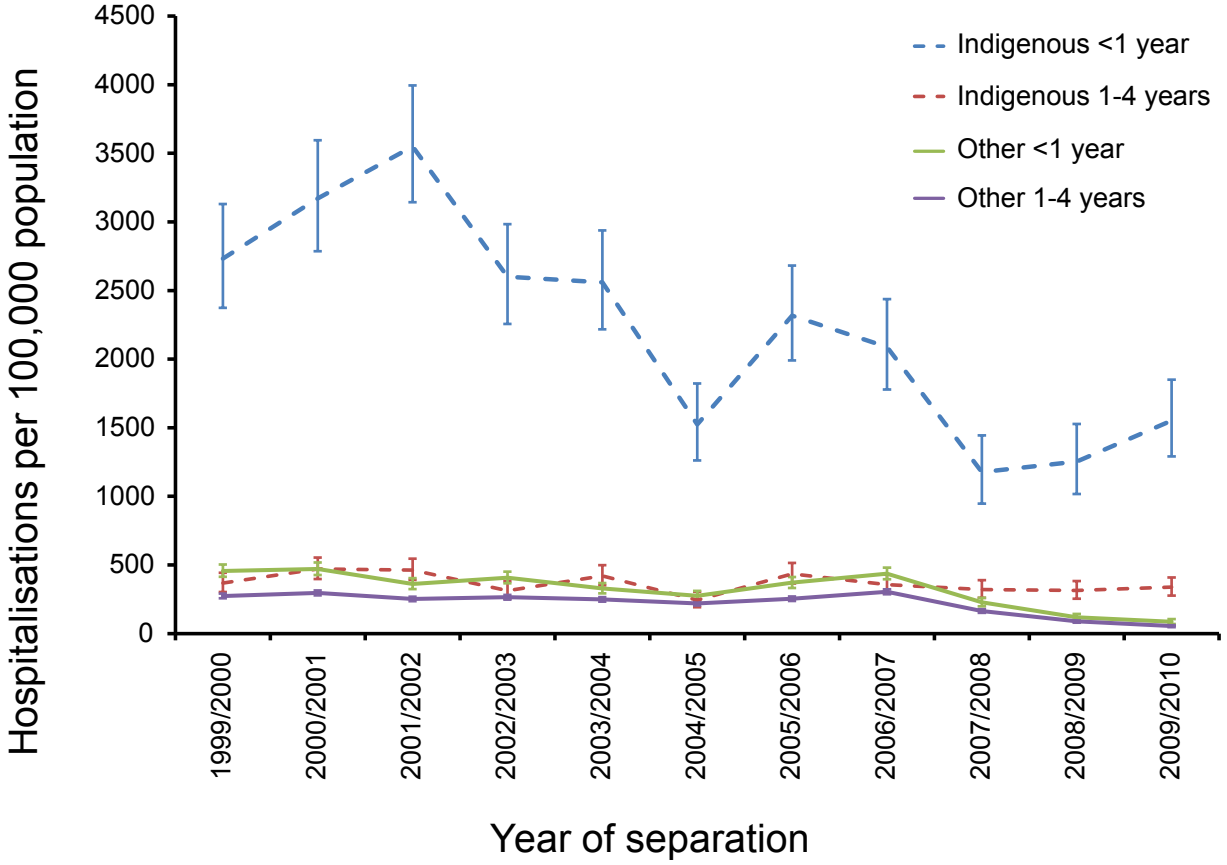
Disease trends

Rotavirus was made notifiable in different jurisdictions at varying times during this reporting period; hence rotavirus notification data are not included in this report. There has been an overall downward trend in rotavirus hospitalisations in children <1 year of age since 2001/2002 (Figure 2.10.1). Vaccination was introduced in the Northern Territory in 2006 and in other states in 2007. Since then, the rates have declined significantly in Aboriginal and Torres Strait Islander infants aged <1 year, while the rates in the 1–4 years age group have remained stable.

Age-specific hospitalisation rates are presented for Aboriginal and Torres Strait Islander and other people, comparing the periods before and after vaccine introduction in 2006/2007. In the period 2002/2003–2009/2010, excluding the vaccine introduction year 2006/2007, there were a total of 8,093 rotavirus hospitalisations in the Northern Territory, Queensland, South Australia and Western Australia. Of those, 2,380 (29%) were aged <1 year, 4,772 (59%) 1–4 years and 941 (12%) ≥5 years. Of the total 8,093 hospitalisations, 1,159 (14%) were reported in Aboriginal and Torres Strait Islander people. There was an overall decline in hospitalisation rates in Aboriginal and Torres Strait Islander children aged <5 years after vaccine introduction, though declines were less than those in other children (Table 2.10.1). There was a 38% reduction in the hospitalisation rate in Aboriginal and Torres Strait Islander children <1 year of age, compared to a 71% reduction in other infants in that age group. There was a small increase in hospitalisation rates in Aboriginal and Torres Strait Islander people, but not in others ≥5 years of age.

There were 1–4 deaths reported from the five jurisdictions (New South Wales, the Northern Territory, Queensland, South Australia and Western Australia) for the period 2006–2010 with rotavirus as the

Figure 2.10.1: Rotavirus hospitalisation rates and 95% confidence intervals, selected Australian states,* 1999 to 2010,† by age group (<5 years) and Indigenous status



* Jurisdictions with satisfactory data quality over the whole time period; see Appendix A (Northern Territory, Queensland, South Australia, Western Australia).

† Hospitalisations where the date of separation was between 1 July 1999 and 30 June 2010.

Table 2.10.1: Rotavirus hospitalisation rates, comparing pre-vaccine period 2002 to 2006 and post-vaccine period 2008 to 2010, selected Australian states,* by age group and Indigenous status

Age group (years)	Pre-vaccine rates† 2002/2003–2005/2006			Post-vaccine rates† 2008/2009–2009/2010		
	Indigenous	Other	Rate ratio	Indigenous	Other	Rate ratio
<1	2,273.4	344.8	6.6§	1,404.1	99.4	14.1§
1–4	351.7	246.0	1.4§	327.3	70.0	4.7§
≥5	1.5	2.3	0.7	2.6	1.9	1.4

* Northern Territory, Queensland, South Australia, Western Australia.

† Average annual age-specific rate per 100,000 population, periods are financial years (July to June).

§ Indicates statistically significant, 95% confidence intervals do not overlap 1.0

underlying cause (the ABS provides ranges when absolute numbers of deaths are low). None of these deaths were reported in Aboriginal and Torres Strait Islander people. There were 5–8 deaths recorded with rotavirus as either the underlying or a contributing cause, of which 1–4 were reported in Aboriginal and Torres Strait Islander people.

Comment

In the pre-vaccine period, there was a higher burden of rotavirus in Aboriginal and Torres Strait Islander children than in other children, especially in those <2 years of age. Aboriginal and Torres Strait Islander children were at higher risk of being notified with or hospitalised due to rotavirus, and when hospitalisation occurred it was at an earlier age and for longer than other children.[95,96]

Since vaccine introduction in Australia, there have been reductions in rotavirus disease notifications[97] and hospitalisations[98-100] in children in the target age group and herd immunity effects in other age groups.[98,100] The reductions in rotavirus-coded hospitalisations reported here in children aged <5 years are an underestimate of the total impact of vaccination; in another study, the reduction in other acute gastroenteritis hospitalisations not coded as due to rotavirus was 6.5 times greater than in those coded as rotavirus.[100] Although there has been a substantial post-vaccine decline in rotavirus hospitalisations in Aboriginal and Torres Strait Islander children, it has been less marked than declines in other children. This is in contrast to the United States where, although there was a greater burden of disease in American Indian and Alaskan Native children than in other children before the introduction of vaccination, the reductions after vaccine introduction have been similar in both groups.[101] Analysis by further age breakdowns in ages ≥5 years has not been presented in this report. However, a modest increase has been reported in national hospitalisation rates in those aged ≥65 years.[100] This may be related to increased testing for rotavirus and other agents of gastroenteritis, such as adenovirus and norovirus, in the elderly.[99]

There are two rotavirus vaccines available; both are oral live attenuated vaccines. Rotarix® is a monovalent human G1P(8) vaccine that requires 2 doses (2 and 4 months of age), and RotaTeq® is a pentavalent human–bovine reassortant vaccine containing G1, G2, G3, G4 and P(8) genotypes, which requires 3 doses (2, 4 and 6 months of age). Immunity from the monovalent vaccine may cover a narrower range of genotypes.[3] Monovalent vaccine effectiveness was found to be 78% in Northern Territory Aboriginal and Torres Strait Islander children during an outbreak of G9P(8) rotavirus in 2007,[102] but only 19% during an outbreak of G2P(4) in 2009.[103] Lower effectiveness of oral vaccines has been shown in developing country settings, possibly related to competition from other gastrointestinal pathogens, poorer immune health, higher maternal antibody levels and/or interference from breast milk,[3] and it is conceivable that these factors may also have an impact in some Aboriginal and Torres Strait Islander communities.

However, while the two vaccines may or may not differ in their effectiveness against particular genotypes, the vaccination coverage achieved from the 2-dose schedule of monovalent vaccine is higher than for the 3-dose pentavalent vaccine. Coverage for Aboriginal and Torres Strait Islander infants at 12 months of age is only 66% for the 3-dose schedule and 77% for the 2-dose schedule. Rotavirus vaccine coverage is much lower (17%) in Aboriginal and Torres Strait Islander children than in other children for the 3-dose schedule, which is a greater disparity than for the 2-dose rotavirus (9%) or non-rotavirus vaccines (7%). This is probably related to the strict upper age cut-offs for rotavirus vaccines and more frequent delayed vaccination of Aboriginal and Torres Strait Islander infants (see 'Vaccination coverage' chapter).

This again underlines the importance of improving timely delivery of immunisation to Aboriginal and Torres Strait Islander children. It also shows the impact of upper age cut-offs for rotavirus vaccines, especially for Aboriginal and Torres Strait Islander children, and an assessment of their costs and benefits may be warranted. Close monitoring of the circulating genotypes of rotavirus and their relationship to vaccine use is also important.

2.11 Varicella-zoster virus infection

The varicella-zoster virus (VZV) causes two distinct diseases, varicella (chickenpox) and herpes zoster (shingles) associated with reactivation of latent VZV. Varicella is highly contagious and generally a benign, self-limiting illness in children, but adults have a significantly higher case morbidity and mortality than children. Acute varicella may be complicated by secondary bacterial infection of the skin, pneumonia, encephalitis, cerebellar ataxia, arthritis, appendicitis, hepatitis, glomerulonephritis, pericarditis and orchitis.[3] Herpes zoster is a reactivation of virus that has lain dormant, usually for years, following varicella infection. It consists of a painful, localised rash. The most common complication of herpes zoster is postherpetic neuralgia; other potential complications include ophthalmic disease, neurological complications, secondary bacterial infections and scarring.[3]

Relevant vaccine history

2003

- Varicella vaccine recommended for all children aged 18 months and 10–13 years without prior history of infection.

2005

- Varicella vaccination funded nationally, at 18 months and 10–13 years of age, for children without prior history of infection.

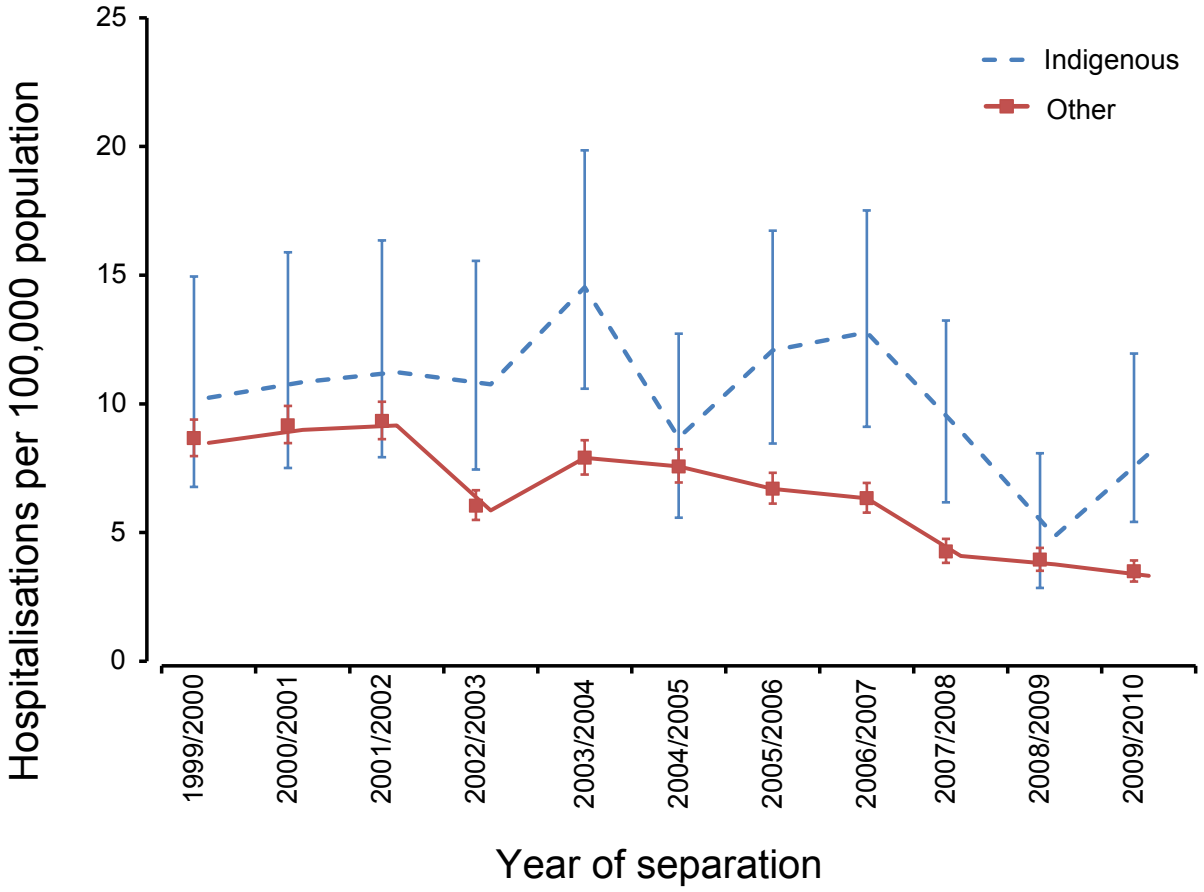
2006

- All jurisdictions commenced school-based catch-up varicella vaccination for one cohort each year of adolescents aged 10–13 years without prior history of infection.

Key points

In the post varicella vaccine period there has been a significant decline in the hospitalisation rate in children due to varicella (chickenpox) in both Aboriginal and Torres Strait Islander and other people.

Figure 2.11.1: Varicella (chickenpox) hospitalisation rates and 95% confidence intervals, selected Australian states,* 1999 to 2010,† by Indigenous status



* Jurisdictions with satisfactory data quality over the whole time period; see Appendix A (Northern Territory, Queensland, South Australia, Western Australia).

† Hospitalisations where the date of separation was between 1 July 1999 and 30 June 2010. Rates are age-standardised to the Australian Bureau of Statistics Australian population estimates for 2006.

Table 2.11.1: Varicella (chickenpox) hospitalisations, selected Australian states, 2005 to 2010, by age group and Indigenous status

Age group (years)	Indigenous status	Hospitalisations* (2005–2010)		
		n	Rate†	Rate ratio
0–4	Indigenous	125	39.9	2.0§
	Other	1,225	19.9	
5–14	Indigenous	67	10.8	1.7§
	Other	798	6.3	
15–24	Indigenous	25	5.0	1.5§
	Other	448	3.3	
25–49	Indigenous	46	5.8	1.4§
	Other	1,452	4.1	
≥50	Indigenous	16	5.3	1.7§
	Other	960	3.1	
All ages‡	Indigenous	279	8.5	1.7§
	Other	4,883	5.0	

* Hospitalisations (New South Wales, Northern Territory, Queensland, South Australia, Victoria, Western Australia only) where the date of separation was between 1 July 2005 and 30 June 2010.

† Average annual age-specific rate per 100,000 population.

‡ Includes cases with age unknown. Rates for all ages combined are age-standardised to the Australian Bureau of Statistics Australian population estimates for 2006.

§ Indicates statistically significant, 95% confidence intervals do not overlap 1.0

Disease trends

Over the period between 1999/2000 and 2009/2010 there has been an overall downwards trend in hospitalisations due to varicella infection in both Aboriginal and Torres Strait Islander and other people. Hospitalisation rates have been persistently higher in Aboriginal and Torres Strait Islander people, with the most recent peak in 2006/2007 followed by a decline to the lowest rates in 2008/2009 (Figure 2.11.1).

In contrast, there has been an upward trend in hospitalisation rates for herpes zoster in Aboriginal and Torres Strait Islander people, after an initial drop in 2003/2004 (Figure 2.11.2). Zoster hospitalisation rates in other people have remained stable over the past 10 years.

Hospitalisation data are presented for six jurisdictions (all except Tasmania and the Australian Capital Territory) by financial year for the period July 2005 to June 2010. Data for notifications are not presented due to the low level of completeness of the Indigenous status field in notification records.

A total of 5,162 hospitalisations for varicella were recorded during this reporting period, of which 279 (5.4 %) were reported in Aboriginal and Torres Strait Islander people. Hospitalisation rates were higher across all age groups in Aboriginal and Torres Strait Islander people (Table 2.11.1). The highest rate (39.9 per 100,000) was in the 0–4 years age group, with an Indigenous to non-Indigenous rate ratio of 2.0:1.

A total of 25,607 hospitalisations for herpes zoster were recorded during this reporting period, of which 376 (1.5 %) were reported in Aboriginal and Torres Strait Islander people. Hospitalisation rates for herpes zoster increased with age, as expected (Table 2.11.2). Rates were higher in Aboriginal and Torres Strait Islander people than in other people across all age groups except those ≥50 years of age. The overall Indigenous to non-Indigenous rate ratio was 1.1:1 (not statistically significantly different to 1:1).

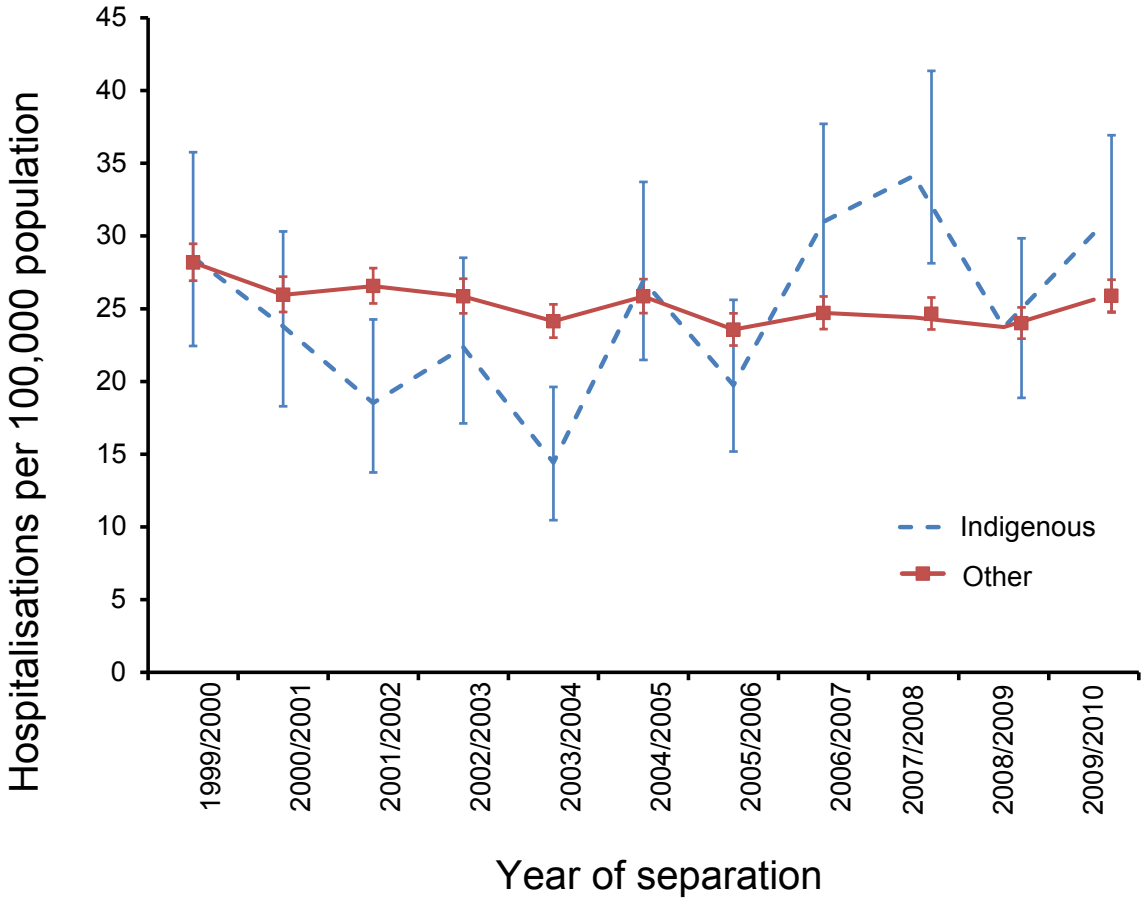
There were 20 deaths reported from the five jurisdictions (New South Wales, the Northern Territory, Queensland, South Australia and Western Australia) for the period 2006–2010 with varicella (chickenpox) as the underlying cause and 37 deaths with varicella (chickenpox) as either the underlying or a contributing cause. There were no deaths reported in Aboriginal or Torres Strait Islander people with varicella as either the underlying or a contributing cause.

There were 75 deaths reported from the five jurisdictions (New South Wales, the Northern Territory, Queensland, South Australia and Western Australia) for the period 2006–2010 with herpes zoster as the underlying cause and 295 deaths with herpes zoster as either the underlying or a contributing cause. There were 1–4 deaths reported in Aboriginal and Torres Strait Islander people with herpes zoster as either the underlying or a contributing cause, all of which were in people aged ≥50 years (the ABS provides ranges when absolute numbers of deaths are low). Overall, 97% of deaths with herpes zoster as either the underlying or a contributing cause were recorded in people ≥50 years of age.

Comment

In the post varicella vaccine period there has been a significant decline in varicella hospitalisation rates in both Aboriginal and Torres Strait Islander and other people, most markedly after national funding in 2005, but also from 2003 when there was significant use of the vaccine in the private market.[104] The decreases have occurred only in the 0–4 years age group (data not shown). Although rates continue to be higher in Aboriginal and Torres Strait Islander people than in other people, varicella (chickenpox) has not been known to be of particular concern in Aboriginal and Torres Strait Islander communities.

Figure 2.11.2: Herpes zoster (shingles) hospitalisation rates and 95% confidence intervals, selected Australian states,* 1999 to 2010,† by Indigenous status



* Jurisdictions with satisfactory data quality over the whole time period; see Appendix A (Northern Territory, Queensland, South Australia, Western Australia).

† Hospitalisations where the date of separation was between 1 July 1999 and 30 June 2010. Rates are age-standardised to the Australian Bureau of Statistics Australian population estimates for 2006.

Table 2.11.2: Herpes zoster (shingles) hospitalisations, selected Australian states, 2005 to 2010, by age group and Indigenous status

Age group (years)	Indigenous status	Hospitalisations* (2005–2010)		
		n	Rate†	Rate ratio
0–4	Indigenous	9	2.9	2.0§
	Other	90	1.5	
5–14	Indigenous	29	4.7	1.7§
	Other	348	2.8	
15–24	Indigenous	26	5.2	2.0§
	Other	369	2.7	
25–49	Indigenous	111	13.9	2.5§
	Other	1,999	5.6	
≥50	Indigenous	201	67.0	0.9
	Other	22,425	72.6	
All ages‡	Indigenous	376	26.0	1.1
	Other	25,231	23.8	

* Hospitalisations (New South Wales, Northern Territory, Queensland, South Australia, Victoria, Western Australia only) where the date of separation was between 1 July 2005 and 30 June 2010.

† Average annual age-specific rate per 100,000 population.

‡ Includes cases with age unknown. Rates for all ages combined are age-standardised to the Australian Bureau of Statistics Australian population estimates for 2006.

§ Indicates statistically significant, 95% confidence intervals do not overlap 1.0

The burden of hospitalisation due to zoster is actually larger than for varicella, as cases are more likely to require hospitalisation. However, there are no clear trends in zoster hospitalisation rates over the past decade. Universal varicella vaccination was hypothesised to contribute to zoster increases, due to reduced natural boosting.[105] Apparent increases in zoster-related disease seen in Australia and elsewhere have been attributed to ageing populations and changes in drug use patterns and have also occurred in the absence of vaccine use.[106]

This analysis is limited by the absence of suitable data on the vast majority of varicella and zoster disease – cases which do not require hospitalisation. Data on notifications and emergency department admissions for varicella and zoster in Aboriginal and Torres Strait Islander people would provide valuable information on future emerging issues such as breakthrough disease and zoster incidence.

2.12 Rare diseases

Four vaccine preventable diseases that are rare in Australia are considered together in this chapter – diphtheria, tetanus, poliomyelitis and rubella. The following data are presented for these diseases: notification data for all jurisdictions for the period 2007–2010 (Table 2.12.1); hospitalisation data for six jurisdictions (all except Tasmania and the Australian Capital Territory) by financial year for the period July 2005 to June 2010 (Table 2.12.2); and death data for New South Wales, the Northern Territory, Queensland, South Australia and Western Australia for the period 2006–2010. Please refer to the 2010 report *Vaccine preventable diseases in Australia, 2005 to 2007* for more detailed information on these diseases.[24]

Diphtheria

Diphtheria is a severe upper respiratory illness caused by *Corynebacterium diphtheriae,* a Gram-positive bacterium. The major threat of diphtheria is fatal acute respiratory obstruction. Diphtheria skin infections can also occur in skin lesions in warm climates and under conditions of poor hygiene.[3] The severe symptoms of diphtheria are associated with toxin produced by the organism. Non-toxigenic forms can cause mild respiratory and skin infections.

Table 2.12.1: Notifications for diphtheria, tetanus, poliomyelitis and rubella, all Australian states, 2007 to 2010, by age group and Indigenous status

Age group (years)	Indigenous status	Diphtheria		Tetanus		Poliomyelitis		Rubella	
		n	Rate*	n	Rate*	n	Rate*	n	Rate*
0–4	Indigenous	0	0.0	0	0.0	0	0.0	0	0.0
	Other	0	0.0	0	0.0	0	0.0	12	0.2
5–14	Indigenous	0	0.0	0	0.0	0	0.0	0	0.0
	Other	0	0.0	0	0.0	0	0.0	6	0.1
15–24	Indigenous	0	0.0	0	0.0	0	0.0	0	0.0
	Other	0	0.0	0	0.0	1	<0.01	31	0.3
25–49	Indigenous	0	0.0	0	0.0	0	0.0	0	0.0
	Other	0	0.0	1	<0.01	0	0.0	86	0.3
≥50	Indigenous	0	0.0	0	0.0	0	0.0	0	0.0
	Other	0	0.0	11	0.04	0	0.0	8	0.03
All ages	Indigenous	0	0.0	0	0.0	0	0.0	0	0.0
	Other	0	0.0	12	0.01	1	<0.01	143	0.2

* Average annual age-specific rate per 100,000 population.

Table 2.12.2: Hospitalisations for diphtheria, tetanus, poliomyelitis and rubella, selected Australian states,* 2005 to 2010,† by age group and Indigenous status

Age group (years)	Indigenous status	Diphtheria		Tetanus		Poliomyelitis		Rubella	
		n	Rate‡	n	Rate‡	n	Rate‡	n	Rate‡
0–4	Indigenous	7	2.2	1	0.3	0	0.0	2	0.6
	Other	2	0.03	0	0.0	0	0.0	16	0.3
5–14	Indigenous	6	1.0	0	0.0	0	0.0	1	0.2
	Other	3	0.02	1	0.01	0	0.0	7	0.1
15–24	Indigenous	3	0.6	0	0.0	1	0.2	0	0.0
	Other	1	0.01	5	0.04	1	0.02	17	0.1
25–49	Indigenous	27	3.4	1	0.1	0	0.0	3	0.4
	Other	14	0.04	22	0.1	1	0.01	66	0.2
≥50	Indigenous	4	1.3	0	0.0	0	0.0	0	0.0
	Other	35	0.1	57	0.2	20	0.1	28	0.1
All ages	Indigenous	47	1.9	2	0.08	1	0.04	6	0.2
	Other	55	0.1	85	0.09	22	0.06	134	0.1

* New South Wales, the Northern Territory, Queensland, South Australia, Victoria, Western Australia.

† Hospitalisations where the date of separation was between 1 July 2005 and 30 June 2010.

‡ Average annual age-specific rate per 100,000 population.

There were no notifications and a total of 102 hospitalisations recorded in this reporting period. Notifications are regarded as the most reliable source of data on toxigenic diphtheria, as public health follow-up and laboratory confirmation is routine. Of the total hospitalisations, 47 (46%) were reported in Aboriginal and Torres Strait Islander people with higher rates than other people in all age groups. The highest rate was among Aboriginal and Torres Strait Islander people aged 25–49 years (3.4 per 100,000), with an Indigenous to non-Indigenous rate ratio of 89:1. In the absence of any notifications during this period, the hospitalisations are most likely to have been due to non-toxigenic or culture-negative suspected diphtheria cases or coding errors.

There were 1–4 deaths reported from the five jurisdictions (New South Wales, the Northern Territory, Queensland, South Australia and Western Australia) for the period 2006–2010 with diphtheria as either the underlying or a contributing cause of death, none of which were reported in Aboriginal and Torres Strait Islander people. At least 1 death recorded as due to diphtheria in 2006 has been previously investigated and reported as likely to be a coding error;[24] hence the data need to be interpreted with caution.

Diphtheria has become rare in Australia but sporadic cases do occur occasionally in unvaccinated individuals. Historical reports document that cutaneous toxigenic *C. diphtheriae* was endemic in the Central Australian desert region and the tropical north of Australia as late as the 1990s, while diphtheria had become a rare occurrence in other states of Australia since the introduction of the diphtheria-tetanus-pertussis vaccine in the 1950s.[107]

A case of cutaneous toxigenic diphtheria acquired in East Timor and notified in 2001 was the first Australian case notified since 1993.[24] Since then 4 cases have been reported, all in 2011 (not included in this reporting period); this included a cluster of 3 cases, one of them fatal respiratory diphtheria, in an unvaccinated family, where contact with a returned traveller was the suspected source of infection.[108] This reinforces the importance of primary vaccination, and also booster doses to prevent travel-associated *C. diphtheriae* infection. Non-toxigenic skin infections still occur, largely limited to Aboriginal and Torres Strait Islander people in the Northern Territory. These infections are not vaccine preventable and underline the importance of improvements to hygiene in remote communities.

Tetanus

Tetanus is unique among vaccine preventable diseases in that it is not communicable.[3] It is caused by *Clostridium tetani* a Gram-positive, spore-forming, anaerobic bacterium that grows at the site of injury and produces toxin with local and systemic neuromuscular effects.[3]

A total of 12 notifications and 87 hospitalisations (with tetanus as either the primary or a contributing cause) were recorded during this reporting period; of these 0 notifications and 2 hospitalisations were reported in Aboriginal and Torres Strait Islander people. One of the hospitalisations occurred in a young child and the other was in a young adult. Tetanus was recorded as the primary cause in 55 hospitalisations; none were among Aboriginal and Torres Strait Islander people. No deaths were reported in Aboriginal and Torres Strait Islander people, and there were 1–4 deaths in other people (the ABS provides ranges when absolute numbers of deaths are low).

Tetanus is a rare disease in Australia, although the exact number of cases is difficult to estimate. Tetanus is likely to be under-notified, as with many clinically diagnosed diseases, while hospitalisations may contain inter-hospital transfers for the same incident, and coding errors. Tetanus predominantly occurs among the adult population who are likely to be unimmunised or insufficiently immunised. A booster dose of a tetanus- and diphtheria-containing vaccine is recommended for adults who are ≥50years of age and have not received a tetanus-containing vaccine in the previous 10 years.[26] It is thought that the booster dose is poorly utilised in Australia.[24] Strategies to improve uptake in this age group should be investigated.

Relevant vaccine history

Diphtheria and tetanus

No difference in vaccination programs between Aboriginal and Torres Strait Islander and other people.

1932

- School-based diphtheria vaccination programs commenced.

1975

- First national vaccination schedule recommended and funded 4 DTPw* doses for infants at 3, 4, 5 and 18 months of age; booster doses of tetanus toxoid recommended every 5 years.

1978

- Fourth dose removed from schedule (reinstated 1985).

1982

- Booster doses of tetanus toxoid recommended every 10 years.

1994

- Fifth dose added at 4–5 years of age.

1999

- DTPa† recommended and funded for all 5 childhood DTP doses.

2003

- 18-month booster replaced by adolescent dose; the eligible age group varied in different jurisdictions.

2008–2012

- dTpa‡ funded temporarily by various states and territories for parents/contacts of infants under cocoon strategy during pertussis epidemic. Program timing and eligibility criteria differed between jurisdictions.

Current schedule

DTPa at 2, 4, 6 months and 4 years of age, dTpa at 10–17 years of age.

* DTPw: diphtheria, tetanus and pertussis (whole-cell)

† DTPa: diphtheria, tetanus and pertussis (acellular)

‡ dTpa: diphtheria, tetanus and pertussis (acellular), reduced antigen content

Poliomyelitis

Poliovirus infection involves the gastrointestinal tract and may progress to the central nervous system. Poliovirus exposure in a person susceptible to poliomyelitis results in one of the following consequences: asymptomatic infection, minor illness, non-paralytic poliomyelitis (aseptic meningitis) or paralytic poliomyelitis. Post-polio syndrome, which encompasses the late manifestations of acute paralytic polio, occurs in 25%–40% of paralytic cases. In recent years, because the feasibility of polio eradication has been demonstrated, the disease has gained renewed attention.[3]

During this reporting period there was 1 notification of polio; the case was in a non-Indigenous person. This case occurred in 2007 and was due to wild poliovirus being acquired by an Australian resident while visiting relatives in Pakistan.[109] This individual had received oral polio vaccine during childhood.

A total of 23 hospitalisations were recorded, of which 1 was in an Aboriginal and Torres Strait Islander person. Notifications are individually reviewed by an expert panel, and are therefore regarded as a more reliable indicator of true polio disease than hospitalisations. The disparity between notification and hospitalisation might be explained by the occurrence of post-polio syndrome rather than acute cases, since the majority of those hospitalisations occurred in those ≥50 years of age. They may also represent cases of acute flaccid paralysis

where polio could not be excluded. No deaths were recorded with poliomyelitis as the underlying cause. Poliomyelitis was recorded as a contributing cause in 11 deaths, none of which occurred in those identifying as Aboriginal and Torres Strait Islander people. This might again indicate people with post-polio syndrome.

Australia was declared polio free in August 2000, indicating the absence of circulation of wild poliovirus.[110] However, a major obstacle to polio eradication appears to be through international spread via travellers, whether they are refugees, pilgrims, traders or tourists.[111] Until the disease has been eradicated there is still a risk of acquiring polio and of re-introduction into polio-free areas.[111]

Relevant vaccine history

Poliomyelitis

No difference in vaccination programs between Aboriginal and Torres Strait Islander and other people.

1956

- IPV* programs commenced in individual jurisdictions.

1966

- IPV replaced by OPV.†

1975

- First national vaccination schedule recommended and funded for infants aged 6, 8 and 10 months.

2005

- IPV funded to replace OPV for children in combination vaccine formulations.

Current schedule

IPV given at 2, 4, 6 months and 4 years of age in combination vaccines.

* IPV: inactivated poliomyelitis vaccine

† OPV: live attenuated oral poliomyelitis vaccine

Rubella

Rubella is caused by the rubella virus. The most common symptoms of rash and lymphadenopathy are usually transient and benign, but infection may be asymptomatic. The severity of the disease increases with age,[112] as does the risk of complications such as thrombocytopenia, encephalitis and a late syndrome of progressive rubella panencephalitis. Rubella is of particular importance when acquired in the first trimester of pregnancy because it is associated with spontaneous abortion or in surviving babies with abnormalities of congenital rubella syndrome (CRS) including cataracts, retinopathy, deafness, heart defects and neurological deficit.[3]

A total of 143 notifications, 140 hospitalisations and no deaths were recorded during the current reporting periods; of these, 0 notifications and 6 hospitalisations were reported in Aboriginal and Torres Strait Islander people. Two of these hospitalisations occurred in children <4 years of age. There were 23 cases in which rubella was recorded as the primary cause of hospitalisation; none of these were in Aboriginal and Torres Strait Islander people. There were 2 notifications of CRS but none in Aboriginal and Torres Strait Islander infants.

Australian seropositivity, vaccine coverage and notification data all support the hypothesis that endemic transmission of rubella may have been eliminated in Australia.[112] However, sporadic cases continue to occur, although the absolute number is difficult to estimate. Notified cases may underestimate total cases as they require laboratory confirmation. The level of laboratory confirmation of hospitalisations is not known, and they may include people diagnosed with rubella but hospitalised for another primary cause. Only 1 case of CRS was notified to the Australian Paediatric Surveillance Unit during this reporting period, in the infant of a woman born overseas.[113] Cases of rubella and CRS related to overseas travel are likely to continue to occur

in Australia while transmission continues in other countries. Women born in countries with low vaccination coverage have been identified as the highest priority for rubella screening and vaccination if non-pregnant or post-partum vaccination if pregnant. Aboriginal and Torres Strait Islander women from some remote communities have been identified as having lower levels of rubella immunity than other women,[114] underlining the importance of screening and vaccination in this group.

Relevant vaccine history

Rubella

1971

- Rubella vaccine funded for females aged 12–14 years (school-based program) and for susceptible women prior to pregnancy.

1989

- MMR* vaccine recommended and funded on the national schedule at 12 months of age (9 months for Aboriginal and Torres Strait Islander infants in the Northern Territory).

1996

- MMR funded as second dose of rubella-containing vaccine for all adolescents.

1998

- Recommended age for first dose of MMR vaccine for Aboriginal and Torres Strait Islander children in the Northern Territory increased from 9 months to 12 months of age.
- Recommended age for second dose of MMR vaccine lowered to 4–5 years.

2013

- Second dose moved forward to 18 months of age, given as MMRV.†

* MMR: measles, mumps and rubella

† MMRV: measles, mumps, rubella and varicella

Comment

These four diseases have been well controlled in Australia, and none are of particular concern in Aboriginal and Torres Strait Islander people. This underlines the substantial health benefits to Aboriginal and Torres Strait Islander and other Australians from effective vaccines in a national vaccination program. However, all of these diseases are still endemic in many developing countries around the world, and could be easily acquired by travellers or imported by returning travellers, posing a threat to unimmunised individuals in Australia.[108,109] Maintaining high vaccination coverage and timely vaccination among children and adults as recommended should remain an important goal for achieving and sustaining elimination of these diseases.

3. Vaccination coverage

Key points

- By 12 months of age, 86% of Aboriginal and Torres Strait Islander children were 'fully vaccinated'.* This proportion has remained stable in recent years and is 6% lower than that of other children.
- By 2 years of age, 91% of Aboriginal and Torres Strait Islander children were 'fully vaccinated',* the same as for other children.
- At 5 years of age, 85% of Aboriginal and Torres Strait Islander children were 'fully vaccinated',* 4 percentage points lower than in other children.
- Coverage of vaccines specifically recommended for Aboriginal and Torres Strait Islander people remains suboptimal.
- A greater proportion of Aboriginal and Torres Strait Islander children are vaccinated after the recommended schedule point than other children; however, this has improved in recent years.
- The age cut-offs for rotavirus vaccine are resulting in lower coverage for this vaccine, and a greater disparity between Aboriginal and Torres Strait Islander and non-Indigenous children.
- The absence of any coverage data for Aboriginal and Torres Strait Islander adolescents, and any coverage data for adults since 2004/2005, is a serious obstacle to program monitoring.

* 'Fully vaccinated' definition from 1996 to June 2013: at 12 months of age – defined as receipt of 3 doses of diphtheria, tetanus, pertussis, Hib, hepatitis B and polio, but did not include rotavirus and pneumococcal vaccines, which are also due at the same schedule points; at 24 months of age – included 3 or 4 doses of Hib and hepatitis B, and 1 dose of measles, mumps, rubella, but did not include meningococcal C or varicella vaccines; at 5 years (60 months) – included a fourth dose of diphtheria, tetanus, pertussis, polio and a second dose of measles, mumps and rubella.

3.1 Methods

Children aged <7 years

Data on vaccination coverage from the Australian Childhood Immunisation Register (ACIR) were provided by Medicare Australia. The ACIR, established in 1996 and administered by the Australian Government Department of Health, is a national register of vaccination doses provided to children <7 years of age. The ACIR includes records for all children who have been registered with Medicare (approximately 99% of Australian children),[115] as well as children who are not registered but for whom details of vaccination doses have been reported to the register.[115,116] Details of vaccination doses provided are reported to the ACIR by vaccination providers.

ACIR data as at June 2011 were used for this report, in most instances using 12-month birth cohorts. Each cohort includes children born between 1 January and 31 December for each respective 12-month period, 2007, 2008, 2009 and 2010. The use of the term 'fully vaccinated' in this report is consistent with its use by Medicare Australia, at the time of publication, for eligibility for incentive payments for carers and immunisation service providers. It does not necessarily include all vaccines provided under the NIP at particular schedule points. 'Fully vaccinated' at 12 months of age is defined as receipt of 3 doses of diphtheria, tetanus, pertussis, Hib, hepatitis B and polio, but does not include rotavirus and pneumococcal vaccines, which are also due at the same schedule points. 'Fully vaccinated' at 24 months of age includes Hib and hepatitis B, and 1 dose of measles, mumps, rubella, but does not include meningococcal C or varicella vaccines. 'Fully vaccinated' at 5 years (60 months) of age includes a fourth dose of diphtheria, tetanus, pertussis and polio and a second dose of measles, mumps and rubella. However, from July 2013, additional vaccines will be included in these calculations: pneumococcal conjugate vaccine, but not rotavirus, at 12 months of age, and meningococcal and varicella vaccines at 24 months of age. This is discussed in more detail in Appendix A. Coverage estimates for the 'old' and 'new' definitions are included in this report.

Adolescents and adults

Vaccination uptake data for Aboriginal and Torres Strait Islander adults, derived from the *2004–05 National Aboriginal and Torres Strait Islander health survey*, was provided in the previous report in this series.[13] At the time of writing, more recent data were not available, with the exception of limited information on influenza vaccine uptake among Aboriginal and Torres Strait Islander adults in the *2009 national adult vaccination survey*.[117]

Records of human papillomavirus (HPV) vaccination are captured and reported by the National HPV Vaccination Program Register (NHVPR),[118-120] as this vaccine is administered to adolescents beyond the 7 year cut-off age of the ACIR. At the time of writing, specific HPV vaccination uptake data for Aboriginal and Torres Strait Islander people have not been published.

3.2 The Australian National Immunisation Program 2007 to 2013

The current NIP schedule applicable to Aboriginal and Torres Strait Islander people is presented in Table 3.2.1. Changes since 2007 are also outlined below.

Children

Rotavirus vaccination was introduced to the NIP in July 2007. Rotavirus vaccination is funded for all children born after 1 May 2007. The rotavirus vaccine schedule is dependent on the brand of vaccine given, and includes at least 2 doses, one at 2 months and one at 4 months of age, with a third dose at 6 months of age for the brand that requires a 3-dose schedule. In the Northern Territory, the 10-valent pneumococcal conjugate vaccine (10vPCV) was used instead of the 7-valent vaccine (7vPCV) from October 2009 to September 2011 in a 4-dose schedule, at 2, 4, 6 and 18 months of age. The 13-valent conjugate vaccine (13vPCV) replaced other pneumococcal vaccines due at 2, 4 and 6 months of age in September 2011 in the Northern Territory, and in July 2011 in all other jurisdictions. A catch-up program for children <3 years of age was also conducted in 2011. From October 2012, 13vPCV also replaced the other pneumococcal booster vaccines due at 18–24 months of age for Aboriginal and Torres Strait Islander children in the Northern Territory, Queensland, South Australia and Western Australia. From July 2013, the combined measles-mumps-rubella-varicella vaccine (MMRV) at 18 months of age replaced the 18-month varicella and 4-year MMR doses for all children.[26]

Adolescents

Funding of HPV vaccination was announced by the Australian Government in November 2006. A quadrivalent HPV vaccine was added to the NIP in April 2007, and an alternative bivalent HPV vaccine was subsequently added in July 2008. The national HPV program is delivered to girls aged 12–13 years via school-based immunisation. A catch-up program for females aged 14–26 years was implemented between 2007 and 2009. From 2013, the national HPV program was expanded to include boys aged 12–13 years, with catch-up vaccination provided for boys aged 14–15 years for a 2-year period.[26,121]

Adults

Since January 2010, seasonal influenza vaccine became available under the NIP for Aboriginal and Torres Strait Islander people aged ≥15 years, pregnant women, and people aged ≥6 months with underlying medical conditions which predispose them to severe influenza.[26]

Additional or specific vaccination recommendations for Aboriginal and Torres Strait Islander people

There are a number of differences in the vaccine recommendations for Aboriginal and Torres Strait Islander Australians compared with those for other Australians. These differences are due to the much higher rates of disease suffered by Aboriginal and Torres Strait Islander people and these epidemiological differences may occur nationally or within limited geographic areas or communities.

Bacille Calmette-Guérin (BCG) vaccine is recommended at birth for Aboriginal and Torres Strait Islander neonates in areas of high risk (the Northern Territory, Queensland and northern South Australia), for the prevention of disseminated tuberculosis.[26]

Hepatitis A vaccine is funded under the NIP for all Aboriginal and Torres Strait Islander children aged 12–24 months living in the Northern Territory, Queensland, South Australia and Western Australia, due to historically higher rates of hepatitis A infection in Aboriginal and Torres Strait Islander Australians in these states, particularly within rural and remote communities.[26]

Table 3.2.1: The Australian National Immunisation Program Schedule for Aboriginal and Torres Strait Islander people, effective 1 July 2013

Age	Vaccine												
Birth	HepB												BCG* ‖‖‖
2 months	HepB	DTPa	Hib	IPV				13vPCV			Rotavirus		
4 months	HepB	DTPa	Hib	IPV				13vPCV			Rotavirus		
6 months	HepB	DTPa	Hib	IPV				13vPCV			Rotavirus†		
12 months			Hib		MMR		MenCCV	13vPCV‡,§		Influenza‖,¶			
12–18 months									HepA‡ ‖‖‖				
18 months					MMRV								
18–24 months									HepA‡ ‖‖‖				
4 years		DTPa		IPV				23vPPV§					
10–13 years	HepB**					VV**							
12–13 years		dTpa										HPV	
15–17 years													
15–49 years								23vPPV††,‡‡,‖‖‖		Influenza‖ ‖‖‖			
≥50 years								23vPPV‡‡,§§		Influenza‖,§§			

* For Aboriginal and Torres Strait Islander infants living in areas of high risk.

† Third dose is dependent on vaccine brand used.

‡ For Aboriginal and Torres Strait Islander children living in areas of higher risk (Northern Territory, Queensland, South Australia and Western Australia).

§ For children with medical risk conditions.

|| Annual vaccination for those in whom vaccine is recommended.

¶ For people medically at risk aged >6 months.

** Should be given only if there is no prior history of disease or vaccination.

†† For Aboriginal and Torres Strait Islander people medically at risk.

‡‡ See recommendations regarding revaccination with 23vPPV in The Australian Immunisation Handbook, 10th edition.

§§ For people aged ≥65 years.

|||| For Aboriginal and Torres Strait Islander people.

See list of Abbreviations for vaccine descriptions.

The 23vPPV was funded under the NIP as a pneumococcal booster vaccine at 18–24 months of age for Aboriginal and Torres Strait Islander children living in the Northern Territory, Queensland, South Australia, and Western Australia. In October 2012, it was replaced by 13vPCV, given at 12–18 months of age.[26]

The 23vPPV is also funded nationally for all Aboriginal and Torres Strait Islander adults aged ≥50 years and Aboriginal and Torres Strait Islander people aged 15–49 years who have an underlying condition which predisposes them to invasive pneumococcal disease.[26]

The seasonal influenza vaccine is funded under the NIP for all Aboriginal and Torres Strait Islander people aged ≥15 years.[26]

3.3 Vaccination coverage estimates from the Australian Childhood Immunisation Register for Aboriginal and Torres Strait Islander versus other children

'Fully vaccinated' coverage using the 'old' (1996 – June 2013) inclusion criteria

'Fully vaccinated' coverage estimates for Australian children born in 2009 (assessed at 12 months of age), 2008 (assessed at 24 months of age) and 2005 (assessed at 60 months of age), using the inclusion criteria which applied from 1996 to June 2013, are presented in Table 3.3.1. 'Fully vaccinated' coverage estimates by cohort, Indigenous status and jurisdiction are presented in Appendix E.

At 12 months of age, the proportion of Aboriginal and Torres Strait Islander children who were recorded as 'fully vaccinated' was 85.5%, compared with 91.9% for all other children. Coverage was lower in Aboriginal and Torres Strait Islander children than in other children in all jurisdictions, with the difference ranging from 4 to 13 percentage points.

At 24 months of age, 'fully vaccinated' coverage estimates in Aboriginal and Torres Strait Islander children were similar to those in other children – 91.3% and 92.0%, respectively. This was the case in most jurisdictions; however, in Western Australia and South Australia coverage was lower in Aboriginal and Torres Strait Islander children than in other children by 4 and 3 percentage points, respectively. In the Australian Capital Territory, 24-month coverage was 5 percentage points higher in Aboriginal and Torres Strait Islander children than in other children.

At 60 months of age, 'fully vaccinated' coverage was 4 percentage points lower in Aboriginal and Torres Strait Islander children (85.3%) than in other children (89.2%). Coverage at 60 months of age was between 2 and 8 percentage points lower in Aboriginal and Torres Strait Islander children than in other children in all jurisdictions, except the Northern Territory, where it was 5 percentage points higher among Aboriginal and Torres Strait Islander children (89.2%).

'Fully vaccinated' coverage using the 'new' extended inclusion criteria (from July 2013)

Vaccination coverage estimates using the new 'fully vaccinated' criteria are presented in Table 3.3.1. Coverage estimates by cohort, Indigenous status and jurisdiction are presented in Appendix E.

'Fully vaccinated' coverage estimates at 12 months of age using the new criteria (including 3 doses of PCV) are little different to those using the old criteria – 0.5 percentage points lower for Aboriginal and Torres Strait Islander children and 1.9 percentage points lower for other children. However, if rotavirus vaccine was also to be included, coverage would be substantially lower: 16 percentage points lower for Aboriginal and Torres Strait Islander children, and 8 percentage points lower for other children. This discrepancy is accounted for by the significantly lower coverage achieved for rotavirus vaccine, particularly in jurisdictions with a 3-dose schedule for rotavirus vaccination, and more so in Aboriginal and Torres Strait Islander children. Therefore, the inclusion of rotavirus and pneumococcal vaccines in the 'fully vaccinated' definition at 12 months of age results in a wider disparity (24 percentage points) between Aboriginal and Torres Strait Islander and other children.

At 24 months of age, the 'fully vaccinated' estimates using the new criteria (including varicella and meningococcal C vaccines) are substantially lower than estimates excluding these vaccines. This is mainly due to the lower coverage for varicella vaccine. However, the difference is similar in scale in both Aboriginal and Torres Strait Islander and other children, resulting in little change in the disparity between the two groups.

Table 3.3.1: Percentage of Australian children immunised, by vaccine type and Indigenous status

Age	Vaccine	Indigenous (%)	Other (%)
Coverage at 12 months of age (born January – December 2009)	DTP 3 doses	85.7	92.6
	Polio 3 doses	85.7	92.6
	Hib (2 or 3 doses)	85.7	92.4
	Hep B (2 or 3 doses)	85.6	92.1
	7vPCV 3 doses	85.3	91.7
	Rotavirus (3-dose states)	66.4	83.4
	Rotavirus (2-dose states)	77.4	86.5
	'Fully vaccinated'*	85.5	91.9
	'Fully vaccinated'† (including 7vPCV)	85.0	90.0
	'Fully vaccinated' (including 7vPCV) + rotavirus	69.9	83.7
Coverage at 24 months of age (born January – December 2008)	DTP 3 doses	94.1	94.7
	Polio 3 doses	94.0	94.6
	Hib (2 or 3 doses)	94.0	94.4
	Hep B (2 or 3 doses)	94.0	93.9
	MMR first dose	94.4	93.8
	MenC 1 dose	93.9	93.3
	Varicella 1 dose	82.3	82.9
	'Fully vaccinated'*	91.3	92.0
	'Fully vaccinated'† (including varicella and MenC)	79.4	81.1
Coverage at 60 months (born January – December 2005)	MMR 2 doses	86.1	89.6
	DTP-polio	85.0	88.8
	'Fully vaccinated'*	85.3	89.2

* 'Fully vaccinated' definition from 1996 to June 2013: at 12 months of age – defined as receipt of 3 doses of diphtheria, tetanus, pertussis, Hib, hepatitis B and polio, but did not include rotavirus and pneumococcal vaccines, which are also due at the same schedule points; at 24 months of age – included 3 or 4 doses of Hib and hepatitis B, and 1 dose of measles, mumps, rubella, but did not include meningococcal C or varicella vaccines; at 5 years (60 months) – included a fourth dose of diphtheria, tetanus, pertussis, polio and a second dose of measles, mumps and rubella.

† 'Fully vaccinated' definition from July 2013: pneumococcal vaccine added at 12 months, varicella and meningococcal vaccines added at 24 months, unchanged at 60 months.

Source: ACIR, data as at June 2011.

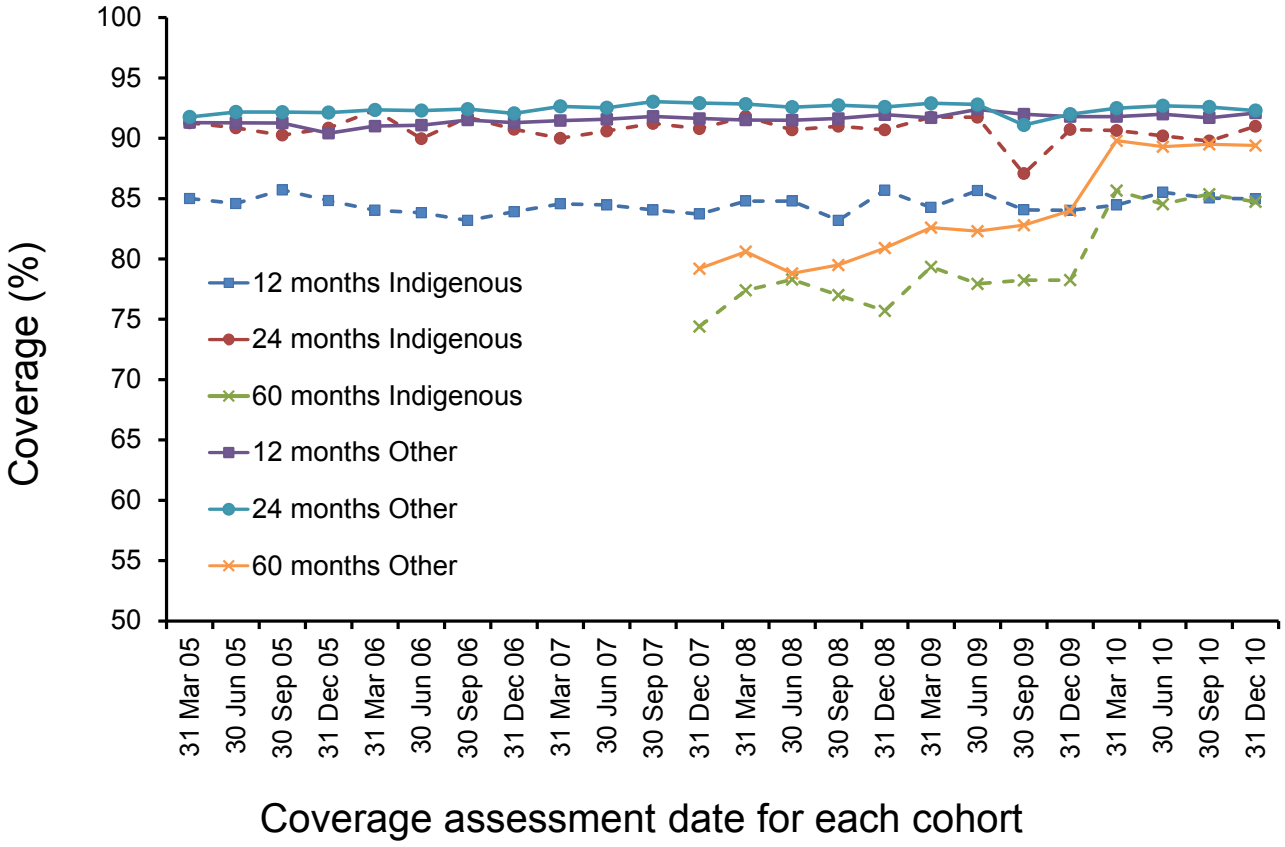
Between 2005 and 2010, 12-month and 24-month vaccine coverage in Aboriginal and Torres Strait Islander children has remained stable (Figure 3.3.1).

Individual vaccines or antigens

Australian vaccination coverage estimates for specific vaccines are presented in Table 3.3.1. Tables of vaccine-specific coverage estimates by cohort, Indigenous status and jurisdiction are presented in Appendix E. The comparison of coverage estimates between Aboriginal and Torres Strait Islander children and other children for specific vaccines is consistent with the comparisons of 'fully vaccinated' coverage. However, rotavirus coverage is substantially lower in Aboriginal and Torres Strait Islander children than in other children, by 11 percentage points in jurisdictions with the 2-dose schedule and by 17 percentage points in jurisdictions with the 3-dose schedule (Table 3.3.1).

Hepatitis A vaccine is recommended for all Aboriginal and Torres Strait Islander children in the Northern Territory, Queensland, South Australia and Western Australia. The 2 doses are scheduled at 12 and 18 months of age in the Northern Territory and Western Australia, and at 18 and 24 months of age in Queensland

Figure 3.3.1: Trend in 12-, 24- and 60-month 'fully vaccinated' coverage in Aboriginal and Torres Strait Islander and other children, 2005 to 2010, by 3-month birth cohorts



Source: ACIR, data as at coverage assessment date.

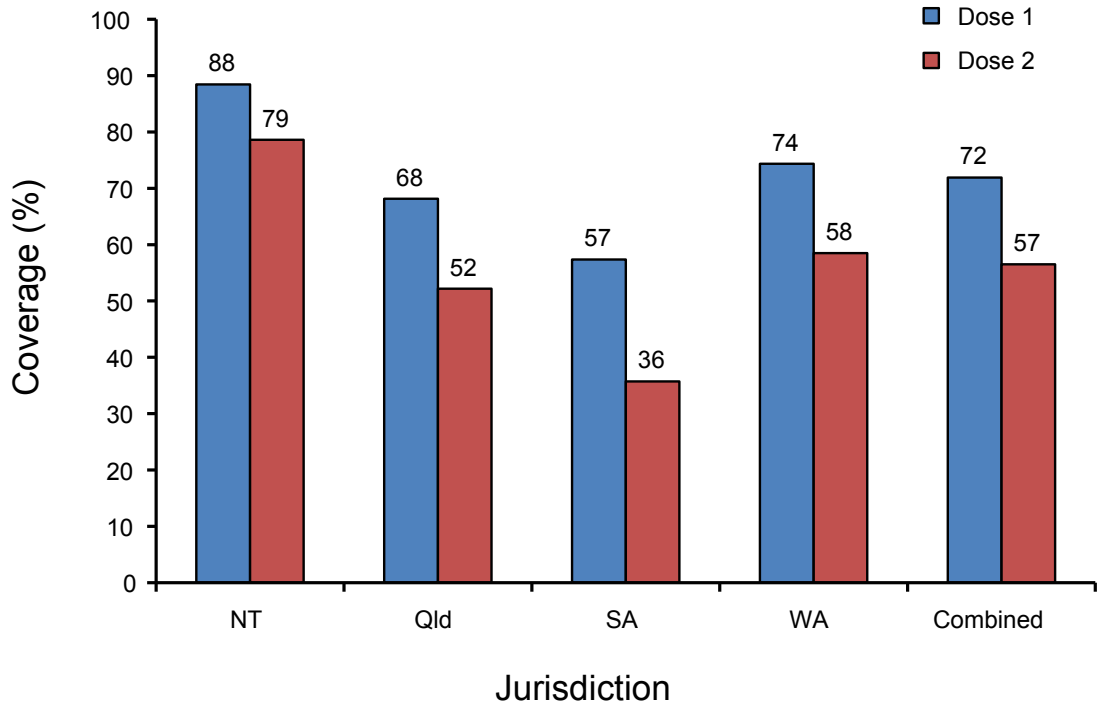
and South Australia. Vaccination coverage for both dose 1 and dose 2 of hepatitis A vaccine is presented in Figure 3.3.2. Coverage for hepatitis A vaccine remains substantially lower than the coverage achieved for universally recommended vaccines. However, hepatitis A coverage has increased dramatically since the previous reporting period in which combined coverage for children born in April to June 2004 was reported to be 29%.[13]

The 23vPPV was recommended for Aboriginal and Torres Strait Islander children in the Northern Territory, Queensland, South Australia and Western Australia, as a booster dose following the routine course of primary vaccination with 7vPCV. This dose was scheduled at 18 months of age in the Northern Territory and Western Australia, and at 24 months of age in Queensland and South Australia. All children in the Northern Territory received the 10-valent pneumococcal conjugate vaccine (10vPCV) instead of the 7vPCV from October 2009 to September 2011, and a fourth dose of 10vPCV at 18 months of age replaced the 23vPPV.[122] Pneumococcal booster coverage is presented in Figure 3.3.3. Similar to hepatitis A coverage, pneumococcal booster coverage is substantially lower than coverage for universally recommended vaccines. Coverage across all the four jurisdictions has increased since the previous reporting period (44%, for 2003–2004 birth cohorts), and increases from 2007 to 2008 occurred in Queensland, the Northern Territory and overall (four jurisdictions combined).

Geographic variations of vaccination coverage

'Fully vaccinated' coverage at 12 months of age is presented by Statistical Division in Figure 3.3.4. This figure should be interpreted with caution as the number of Aboriginal and Torres Strait Islander children within some Statistical Divisions is small. Despite this, the map demonstrates the significant variation in vaccination coverage within jurisdictions, in particular lower coverage in the remote regions of South Australia and Western Australia, and generally higher coverage in areas in the north and south-east of the country.

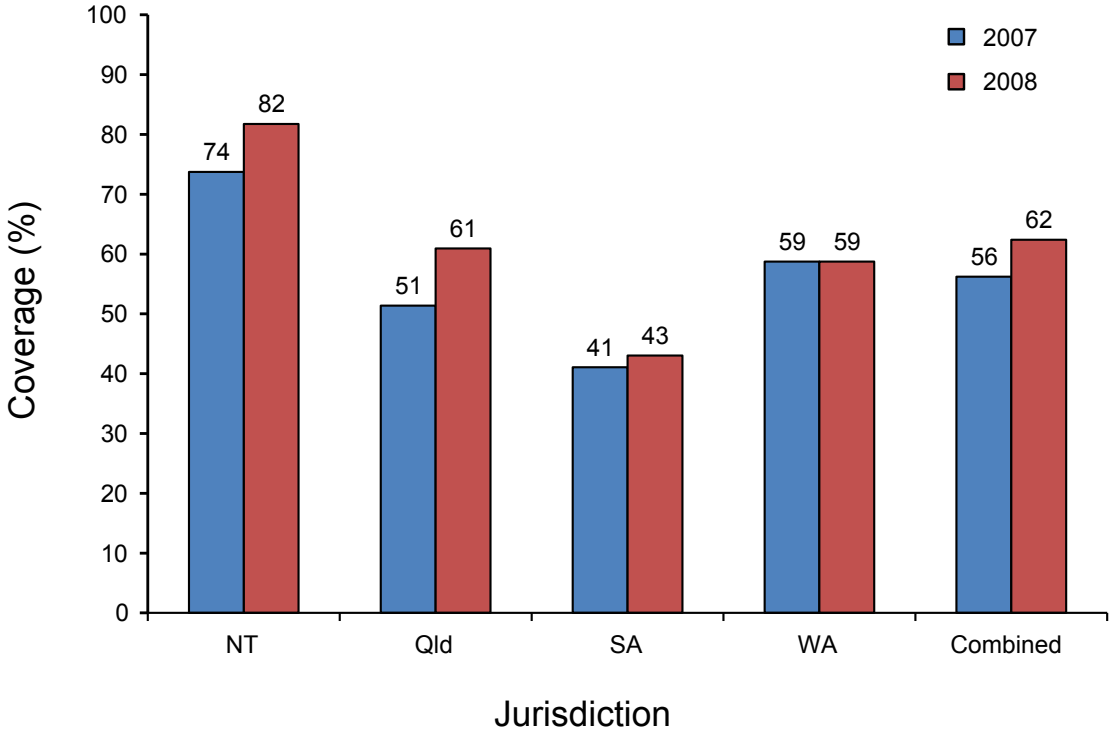
Figure 3.3.2: Percentage of Aboriginal and Torres Strait Islander children born in 2008 who had received 1 or 2 doses of Hepatitis A vaccine within 6 months of the relevant schedule point



NT: Northern Territory; Qld: Queensland; SA: South Australia; WA: Western Australia

Source: ACIR, data as at June 2011.

Figure 3.3.3: Percentage of Aboriginal and Torres Strait Islander children who had received a pneumococcal booster dose* within 6 months of the relevant schedule point, 2007 and 2008, by year of birth

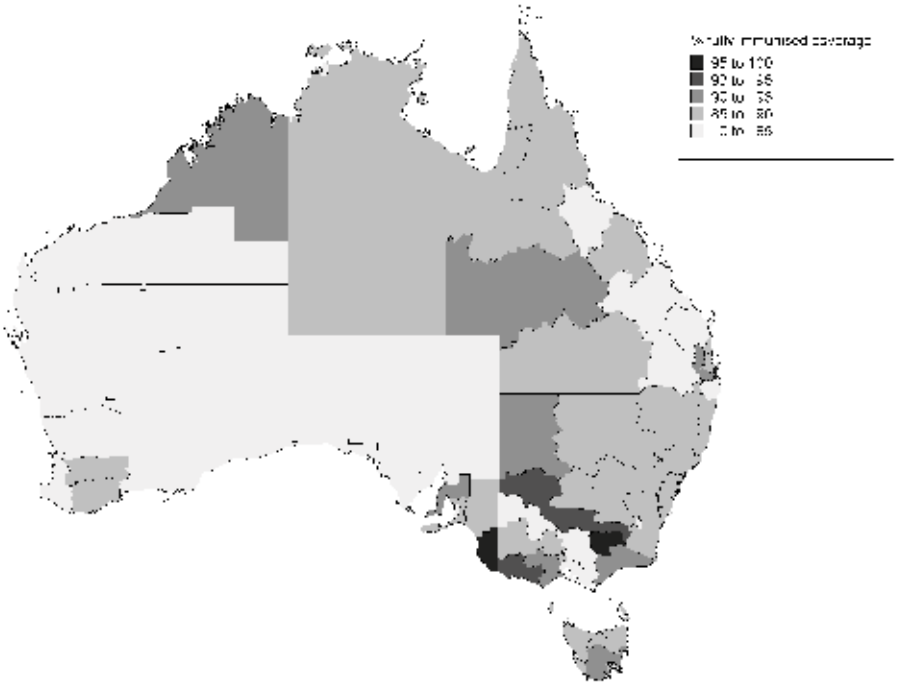


* 23-valent, 10-valent or 13-valent vaccines

NT: Northern Territory; Qld: Queensland; SA: South Australia; WA: Western Australia

Source: ACIR, data as at June 2011.

Figure 3.3.4: 'Fully vaccinated' coverage at 12 months of age in Aboriginal and Torres Strait Islander children born in 2009, Australia, by Statistical Division



Source: ACIR, data as at September 2011

Timeliness of vaccination

Delays in receipt of the second dose of the DTPa vaccine, the second dose of 7vPCV, and the first dose of MMR are used here as indicators for assessing timeliness of vaccination.

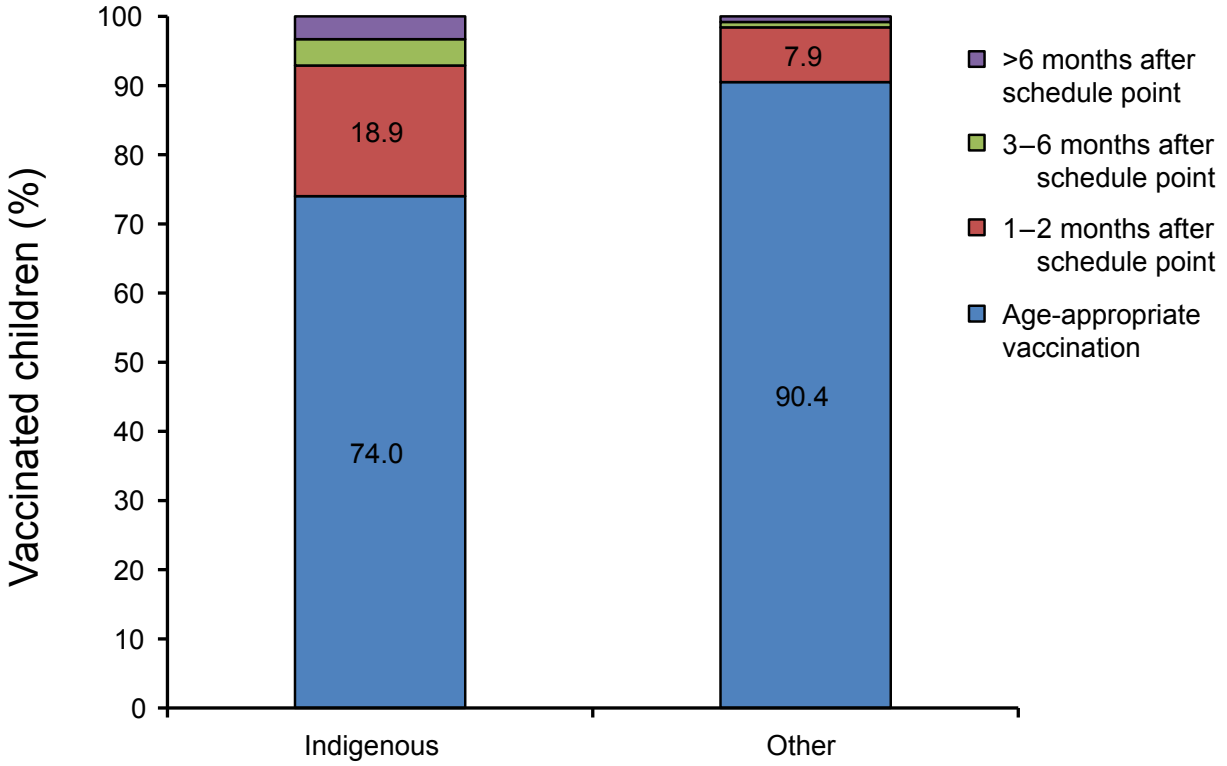
Timeliness of the second dose of DTPa vaccine is presented in Figure 3.3.5. Among Aboriginal and Torres Strait Islander children, 74.0% of the cohort born in 2009 received age-appropriate vaccination (before 7 months of age), compared with 90.4% of other children. In addition, substantially larger proportions of Aboriginal and Torres Strait Islander children were vaccinated 3 or more months after the schedule point (7.1% vs 1.6% of other children). However, the overall proportion of children within the cohort who eventually received the second dose of DTPa by 18 months of age was similar in Aboriginal and Torres Strait Islander children and other children (92.4% and 94.0%, respectively).

Timeliness of the second dose of DTPa among Aboriginal and Torres Strait Islander children varied by jurisdiction (Figure 3.3.6). Jurisdictions achieved coverage between 88.0% and 96.2% for receipt of the second dose of DTPa by 18 months of age. However, timeliness varied between jurisdictions, with 63.6% to 85.1% of Aboriginal and Torres Strait Islander children born in 2009 receiving age-appropriate vaccination.

Trends over time in timeliness of the first dose of MMR vaccine are presented in Figure 3.3.7. There has been an improvement in vaccination timeliness for the first dose of MMR vaccine between 2004 and 2008 in both Aboriginal and Torres Strait Islander and other children. Despite the improvement over time, the proportion of Aboriginal and Torres Strait Islander children with delayed vaccination remains higher than in other children.

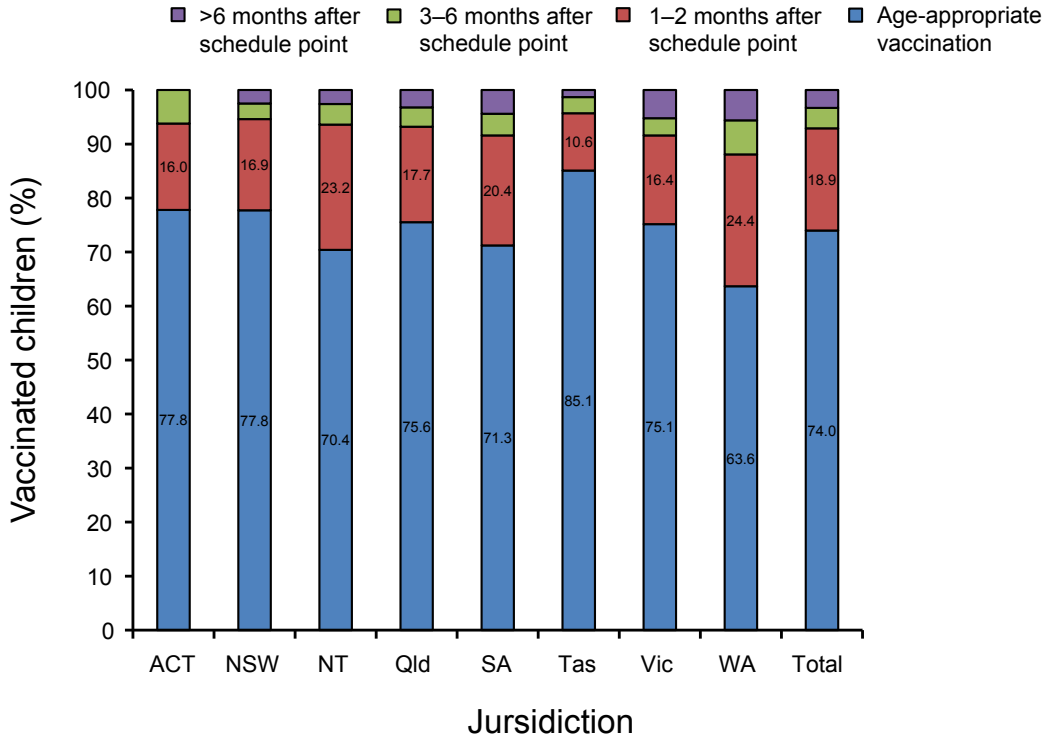
Table 3.3.2 shows vaccination delay by remoteness. It shows that poorer timeliness for Aboriginal and Torres Strait Islander children is consistent across both remote and accessible regions, and there is relatively little difference by remoteness classification.

Figure 3.3.5: Timeliness of the second dose of DTPa vaccine, children born in 2009, by Indigenous status



Source: ACIR, data as at June 2011.

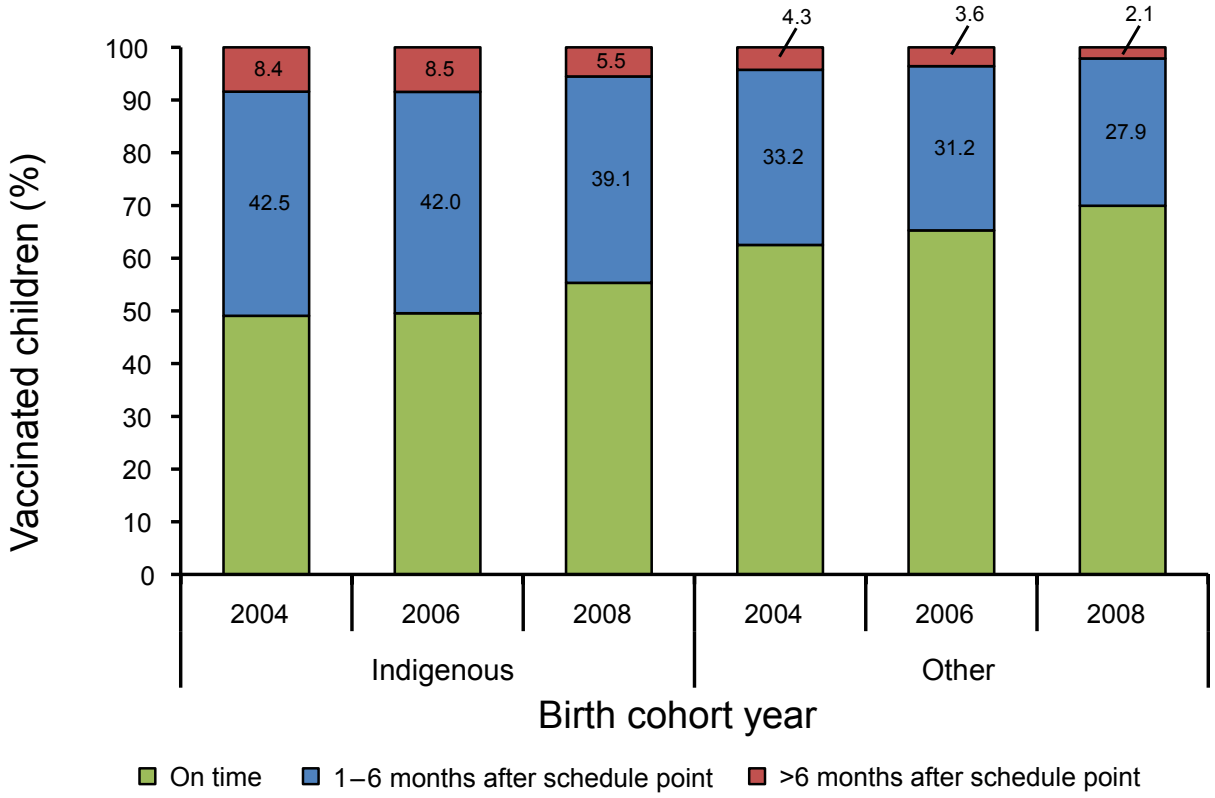
Figure 3.3.6: Timeliness of the second dose of DTPa vaccine, Aboriginal and Torres Strait Islander children born in 2009, by jurisdiction



ACT: Australian Capital Territory; NSW: New South Wales; NT: Northern Territory; Qld: Queensland; SA: South Australia; Tas: Tasmania; Vic: Victoria; WA: Western Australia

Source: ACIR, data as at June 2011.

Figure 3.3.7: Timeliness of the first dose of MMR vaccine, by Indigenous status and year of birth, 2004 to 2008



Source: ACIR, data as at June 2011.

Table 3.3.2: Vaccination delay in children born in 2008, Australia, by Indigenous status and remoteness status

Vaccine dose	Indigenous status	Remoteness	1–6 months after schedule point (%)	>6 months after schedule point (%)
DTP second dose	Indigenous	Accessible*	21.0	3.5
		Remote†	27.0	3.0
	Other	Accessible	8.7	0.8
		Remote	8.9	0.6
MMR first dose	Indigenous	Accessible	33.2	5.6
		Remote	32.9	4.4
	Other	Accessible	25.1	2.1
		Remote	24.0	1.9

* Areas classified as 'Highly accessible', 'Accessible' or 'Moderately accessible' by the Accessibility/Remoteness Index of Australia.

† Areas classified as 'Remote' or 'Very remote' by the Accessibility/Remoteness Index of Australia.

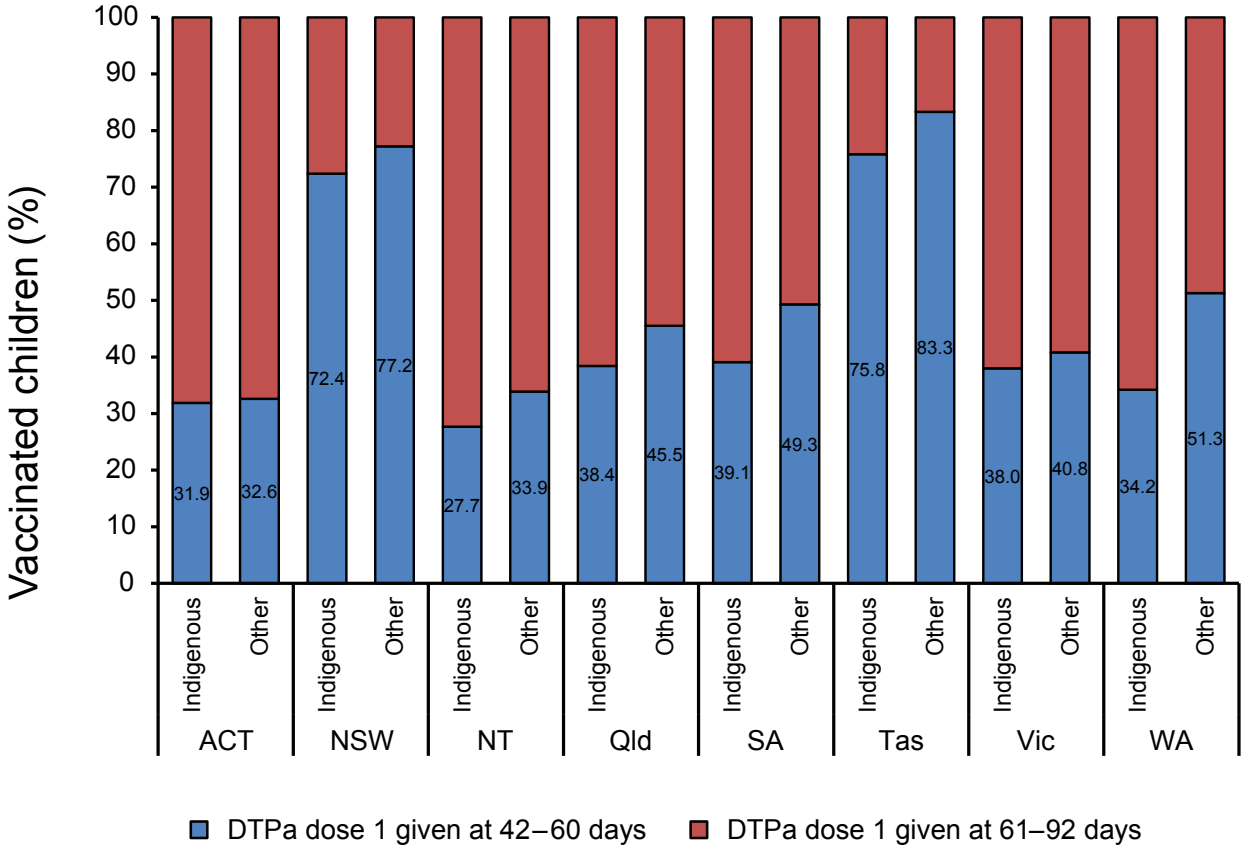
Uptake of first dose of pertussis-containing vaccine before 2 months of age, by jurisdiction

In March 2009, the Australian Technical Advisory Group on Immunisation recommended that the first dose of DTPa vaccine be given at 6 weeks instead of the usual 2 months (8.5 weeks) of age. This was to provide protection to infants as early as possible during a pertussis epidemic. This change was promoted by some jurisdictions more than others, partly depending on the severity of their respective pertussis epidemics at the time. Among the cohort of Australian children born in 2010, there was evidence of the implementation of the earlier first dose of DTPa vaccine in New South Wales and Tasmania (Figure 3.3.8). In these two states, the proportion of age-appropriately vaccinated children who received a first dose of DTPa between 42 and 60 days of age was greater than 70% for both Aboriginal and Torres Strait Islander and other children. In all other jurisdictions, the majority of age-appropriately vaccinated children received their first dose of DTPa after 60 days of age. The proportion of children vaccinated at 42–60 days tended to be slightly higher in non-Indigenous children than in Aboriginal and Torres Strait Islander children, but in general, where this policy was implemented, it was implemented for both Indigenous and non-Indigenous children.

Vaccine refusal

Among all Australian children born between 2004 and 2008, 1.6% were recorded as vaccine refusers. The proportion of Aboriginal and Torres Strait Islander children who were recorded as vaccine refusers was less than one-quarter of that in other children (0.37% vs 1.65%). The difference in proportions was statistically significant (P<0.0001).

Figure 3.3.8: Proportion of on-time vaccinated infants born October–December 2009 who received the first dose of pertussis-containing vaccine at 6–8.5 weeks versus 2–<3 months, by jurisdiction



ACT: Australian Capital Territory; NSW: New South Wales; NT: Northern Territory; Qld: Queensland; SA: South Australia; Tas: Tasmania; Vic: Victoria; WA: Western Australia

Source: ACIR, data as at June 2011.

3.4 Comment

For universally recommended childhood vaccines, Aboriginal and Torres Strait Islander children continue to have lower vaccination coverage at 12 months of age than other children. The difference in 'fully vaccinated' coverage, using the 'old' (1996 – June 2013) definition, was 6 percentage points during this reporting period. By 24 months of age, the difference was less than 1 percentage points. As no vaccines given between 12 and 24 months of age were included in this calculation, this suggests the role of a higher level of delayed vaccination in Aboriginal and Torres Strait Islander children than in other children. Coverage for the pre-school doses due at 4 years of age reflects a similar situation. Between 2003 and 2006, when 'fully vaccinated' coverage was measured at 72 months (6 years) of age, it was comparable between Aboriginal and Torres Strait Islander and other children. However, in this report, where coverage is now measured at 60 months (5 years) of age, it is 4 percentage points lower in Aboriginal and Torres Strait Islander children than in other children.

However, there have been improvements in recent years. The promotion of administering the first dose at 6 weeks of age in New South Wales and Tasmania in 2009 was effective in both Aboriginal and Torres Strait Islander and other children. Also in 2009, the change in due and overdue rules for the pre-school doses, which classified children as overdue at 4 years and 1 month instead of 5 years of age, resulted in substantial improvements in timeliness in Aboriginal and Torres Strait Islander and other children. Improvements over time have also been seen in coverage for all children for doses due at 12 months of age, although this does not appear to be directly related to a specific policy initiative. For individual vaccines, coverage estimates are quite similar to those for 'fully vaccinated', with the exceptions of rotavirus and varicella vaccines. Rotavirus vaccine coverage at 12 months of age in 'other' infants is 6–9 percentage points lower than for other vaccines given at this milestone. This is probably a result of the strict upper age limits for administering these vaccines, as infants arriving too late to receive rotavirus vaccine can still receive other vaccines. However, for Aboriginal and Torres Strait Islander infants, this discrepancy is more marked in areas using a 3-dose schedule, where the proportion of Aboriginal and Torres Strait Islander infants who have received rotavirus vaccine by 12 months of age is 17 percentage points lower than in other infants. Varicella vaccine coverage by 24 months of age is lower than for other vaccines, but there is little difference in coverage between Aboriginal and Torres Strait Islander and other children. This underlines the importance of continuing to monitor, and have access to, coverage data on vaccines not included in the 'fully vaccinated' definition.

Coverage for vaccines recommended only for Aboriginal and Torres Strait Islander children continues to be substantially lower than that for universal vaccines, as shown in this report for hepatitis A vaccine and pneumococcal vaccine boosters. Both these vaccines are limited to the Northern Territory, Queensland, South Australia and Western Australia. Coverage varies much more substantially between jurisdictions for these vaccines than for universally recommended vaccines. This underlines the importance of immunisation providers establishing the Indigenous status of their clients, particularly in urban areas, so they can offer them the additional vaccines they may require.

There has been relatively little research on the specific causes of low coverage and delayed vaccination in Indigenous people worldwide. Greater mobility of Aboriginal and Torres Strait Islander people may contribute in some instances. However, the evidence suggests many of the barriers are those common to people of low socioeconomic status.[123,124] Measures that are effective in other settings can also be effective in Indigenous communities; these include the elimination of financial barriers, vaccination by non-medical staff, patient and provider education, patient reminders, diverse delivery models such as outreach and inpatient as well as primary care,[125] and clinical quality improvement activities. The introduction of a personalised calendar to improve timeliness is an Australian example of the successful implementation of a measure that had been effective in other settings being applied to Aboriginal and Torres Strait Islander children.[126] The use of Medicare items for child and adult health checks for Aboriginal and Torres Strait Islander people can also be effective for catch-up vaccination.[127] Monitoring milestones earlier than at 12 months of age may also be worth consideration.[128]

Unlike previous editions of this report, updated data from the National Aboriginal and Torres Strait Islander Health Survey was not available. At the time of writing a survey was in progress, the first since the 2004/2005 survey reported on in our previous edition. The only other data on Aboriginal and Torres Strait Islander adult vaccination coverage available during this 8-year gap were from the 2009 Adult Vaccination Survey. This survey estimated coverage of 23% for the pandemic H1N1 vaccine and 28% for the 2009 seasonal influenza vaccine among Aboriginal and Torres Strait Islander people aged ≥18 years.[117] No coverage data are available for the first 2 years of funded influenza vaccine for Aboriginal and Torres Strait Islander people aged ≥15 years. This highlights the need for more frequent and detailed data to support program managers.

For Aboriginal and Torres Strait Islander adolescents, no vaccine coverage data have been published or publicly released.

Indigenous status is collected on the National HPV Register but this has not been included in HPV coverage data released to date. Coverage data for the general adolescent population for vaccines other than HPV are not collated nationally, and are infrequently published by states and territories. New Zealand Maori adolescents have been shown to have lower consent form returns and lower vaccination rates from school-based programs.[129] A similar finding in Australia for Aboriginal and Torres Strait Islander adolescents would not be unexpected. Given the focus on school-based delivery and lower rates of school attendance by Aboriginal and Torres Strait Islander students,[130] good coverage data for this age group are urgently needed.

4. Discussion

Vaccination has had a substantial positive impact on the health of Aboriginal and Torres Strait Islander people. This report documents further recent improvements in vaccine delivery to, and impact on, Aboriginal and Torres Strait Islander people, while highlighting some areas for improvement.

4.1 Disease impact

In Australia, the endemic transmission of many diseases which caused significant burden in the past, disproportionately affecting Aboriginal and Torres Strait Islander people, has been largely controlled. Diseases like diphtheria, tetanus and poliomyelitis have become rare in Australia due to vaccination programs. There have also been significant declines more recently in diseases like measles, varicella and hepatitis A, which are not, or are no longer, of particular concern in Aboriginal and Torres Strait Islander communities. However, all of these diseases are still endemic in some or many developing countries around the world, and could be easily acquired by travellers or imported by returning travellers, posing a threat to unimmunised individuals. Hence, maintaining high vaccination coverage and improving timeliness of vaccination among both children and adults should remain an important goal for achieving and sustaining the elimination of these diseases.

Although rates of Hib disease have decreased significantly since the introduction of Hib vaccines in 1993, the plateauing in Aboriginal and Torres Strait Islander children, and increasing disparity with other children, are concerning. Continuing Hib nasopharyngeal carriage, increased susceptibility to Hib disease, and poor immune responses to immunisation have been implicated as driving forces behind continuing disease in Aboriginal and Torres Strait Islander children. The progressive withdrawal of PRP-OMP vaccines (3 doses) in Australia from 2005 to 2009, replaced by 4 doses of PRP-T, was due to an international shortage of PRP-OMP vaccine. While it is possible that higher disease rates in young infants could be associated with the later age of protection from PRP-T vaccine, it is also possible that the higher immunogenicity of the PRP-T vaccine will result in reduced Hib carriage. Close monitoring is important to detect any re-emergence of disease as soon as possible.

Some diseases like pertussis have still not been controlled and outbreaks continue to occur. Pertussis is the least well controlled of the diseases that have long-standing, well-established vaccination programs. Of these diseases, pertussis has the highest notification rates (in all age groups) and higher hospitalisation rates. Epidemics continue to occur in Australia which affect both Aboriginal and Torres Strait Islander and other people, although Aboriginal and Torres Strait Islander people have higher hospitalisation rates. In order to protect the most vulnerable in the community from severe disease, from 2008 to 2012, DTPa vaccination was funded by various states and territories for parents/contacts of infants under the 'cocooning' strategy. Parents are also now encouraged to have their infant's first vaccination given at 6 weeks of age, instead of the usual 2 months, and this is being implemented for Aboriginal and Torres Strait Islander as well as other infants. Timely administration of the 4- and 6-month doses is also very important.

Although there have been substantial declines in severe rotavirus disease, mumps, meningococcal disease and hepatitis B infection, the rates are still higher in Aboriginal and Torres Strait Islander people than in other people. The decline in severe rotavirus disease after vaccine introduction was most pronounced in infants <1 year of age, but less marked in Aboriginal and Torres Strait Islander than in other infants. By far the highest hospitalisation rates continue to occur in Northern Territory Aboriginal and Torres Strait Islander children. Vaccination delay is having a substantial impact on coverage for rotavirus vaccine due to the upper age limits for vaccination and this is most marked for the 3-dose schedule. Consideration of the role of age cut-offs and 2-dose versus 3-dose schedules may be necessary. Genotype surveillance is critically important to be able to detect any possible future emergence of genotypes for which there is lower vaccine-derived immunity.

Mumps has been reported to have recently made a re-emergence globally. During this reporting period, a prolonged outbreak was recorded with a peak in hospitalisations in 2007/2008. This outbreak predominantly affected Aboriginal and Torres Strait Islander adolescents and young adults. The rates among Aboriginal and Torres Strait Islander people have declined from the highest of 5 per 100,000 in 2007/2008 to less than 1 per 100,000 in 2009/2010. Whereas mumps was historically a disease of childhood, recent outbreaks have predominantly involved young adults, nearly all of whom had a history of vaccination during childhood, most with the recommended 2-dose schedule. Evidence of waning immunity has led to suggestions that vaccination with a third dose during adolescence might be an effective measure to prevent outbreaks.[1] However, outbreaks in Australia and overseas have subsided without this being routinely implemented as yet.

Routine meningococcal C vaccination for infants and the high-school catch-up program, implemented from 2003, have resulted in a significant decrease in cases associated with serogroup C. However, the predominant serogroup responsible for disease remains serogroup B and vaccines in development are keenly awaited.[131]

Pandemic and seasonal influenza and pneumonia are other diseases with higher rates in Aboriginal and Torres Strait Islander people than in other people. This has been attributed to higher susceptibility to influenza disease and the prevalence of comorbidities. For Aboriginal and Torres Strait Islander people aged ≥50 years there is evidence of a decline in influenza hospitalisations from 1999, suggesting some possible impact of the National Indigenous Pneumococcal and Influenza Immunisation (NIPII) Program. Since 2010, seasonal influenza vaccine has been funded for all Aboriginal and Torres Strait Islander people aged ≥15 years. This broadening of the vaccination program has the potential to result in a further reduction in the disease burden of seasonal influenza in this population. However, infrequent but low coverage estimates in the 50–64 years age group and the lack of coverage data in the 15–49 years age group, highlight the need for improved coverage data to facilitate program implementation.

For invasive pneumococcal disease, a substantial overall reduction in notifications of IPD caused by serotypes contained in the 7vPCV has been seen among Aboriginal and Torres Strait Islander children after the introduction of vaccination. However, due to a lower proportion of IPD being caused by 7vPCV types prior to vaccine introduction, delayed vaccination, and a higher prevalence of risk factors associated with IPD among Aboriginal and Torres Strait Islander people, the decline in IPD notifications has been less marked than in other people. The higher valency vaccines (10vPCV and 13vPCV) that replaced 7vPCV may result in a greater effect on both invasive and non-invasive disease. Recent evidence of an increase in IPD in Aboriginal and Torres Strait Islander people aged ≥50 years, together with infrequent, unvalidated and low coverage estimates, highlight the need for improved coverage data.

4.2 Vaccination coverage

High coverage for universal vaccines, but greater vaccination delay, continue to be features for Aboriginal and Torres Strait Islander children. There have been some improvements in vaccination timeliness in recent years, but disparities remain between Aboriginal and Torres Strait Islander children and other children. This reduces the potential benefits of vaccination for Aboriginal and Torres Strait Islander children, most importantly for pneumococcal, Hib and rotavirus vaccines in infants. The age cut-offs for rotavirus vaccines present a particular challenge for timely vaccination. Coverage achieved by the 2-dose rotavirus schedule is around 8 percentage points lower in both Aboriginal and Torres Strait Islander and other infants compared to other vaccines, but the disparity between Aboriginal and Torres Strait Islander and other children has not substantially changed. However, the proportion of children who complete the 3-dose schedule is 17 percentage points lower in Aboriginal and Torres Strait Islander children than in other children, with 34% of Aboriginal and Torres Strait Islander children remaining incompletely immunised or unimmunised.

Coverage for vaccines recommended only for Aboriginal and Torres Strait Islander children (hepatitis A and pneumococcal boosters in some jurisdictions) continues to remain substantially lower than that for universally recommended vaccines. This underlines the importance of immunisation providers establishing the Indigenous status of their clients, so they can offer them the additional vaccines they may require.

However, the absence of any coverage data for Aboriginal and Torres Strait Islander adolescents or for adults since 2004/2005 is a substantial obstacle to implementing and improving programs in these groups.

Appendix A: Technical notes on methods and interpretation of data

A.1 Vaccine preventable diseases data

The methods used in this report are adapted from and align with the methods previously applied in the series of reports on Vaccine preventable diseases and vaccination coverage in Aboriginal and Torres Strait Islander people, Australia.[13,132]

General issues regarding data on vaccine preventable diseases

Three sources of routinely collected data were used for this report. Notification data were obtained from the National Notifiable Diseases Surveillance System (NNDSS) and hospitalisation data from the Australian Institute of Health and Welfare (AIHW) National Hospital Morbidity Database. Mortality data were provided by the Australian Bureau of Statistics (ABS).

Comparisons between the notification, hospitalisation and death data should be made with caution since these datasets differ in their purposes of collection, reporting mechanisms, accuracy, timeliness and period of reporting.

In this report, in order to provide the most recent information available, and to accommodate the varied reporting formats, data of different time periods (representing the available data for the most recent years since the previous report in the series) have been selected from each dataset for review. As there are no unique identifying codes to link records for the same individuals across these datasets, and due to differences in defining a case and in the completeness and the accuracy of the data in each dataset, it is not possible to interpret deaths and hospitalisations as subsets of notifications.

For some diseases, there are no specific ICD codes that correspond to the particular disease condition of interest. This will limit the validity of comparisons between notification data and hospitalisation or death data (e.g. for invasive pneumococcal disease). The algorithms used to select surrogate ICD codes to match the hospitalisation case definitions for invasive pneumococcal disease are explained in the disease chapter. Hospitalisation data for invasive *Haemophilus influenzae* type b disease has not been presented because no type-specific ICD code exists.

Indigenous status identification in the datasets used in this report

In this report, the term 'Indigenous status' refers to a field in a dataset in which information is recorded on whether or not a person has been identified as Aboriginal and/or Torres Strait Islander. It is reported as either one of two categories: an individual who was identified in a record as an Aboriginal and/or Torres Strait Islander person is categorised as 'Indigenous'; an individual whose Indigenous status was recorded as non-Indigenous or was not stated or inadequately described is categorised as 'Other'.

The quality of Aboriginal and Torres Strait Islander health statistics depends on the accuracy of Indigenous population estimates and completeness and reporting accuracy in the collection of Indigenous status information for the disease of interest. Considerable work has been done in recent years by agencies such as the ABS, AIHW and state and territory governments on assessing and improving the quality of Aboriginal and Torres Strait Islander statistics in national and state and territory administrative data collections.[133-136] More work is needed to improve the quality of the data, as there are large variations in quality between data collections (particularly for notifications), and within the same data collections, there are variations between jurisdictions and over time.

Notifications

The NNDSS was established in its current form in 1991, and includes de-identified information about cases of notifiable vaccine preventable diseases (VPDs) notified to state and territory authorities under their respective public health legislation. Prior to 2004, state and territory notification criteria were based on the National Health and Medical Research Council (NHMRC) surveillance case definitions,[137] with various modifications applied in different jurisdictions. Since 2004, all jurisdictions have applied the new national case definitions for notifiable diseases endorsed by the Communicable Diseases Network Australia.[138] The case definitions for notifications for each of the included VPDs are described in Appendix B.

The data collected by the NNDSS are continually updated by jurisdictions. There could be minor variations between NNDSS data in this report and annual reports of the NNDSS (Australia's Notifiable Disease Status reports)[16] and other reports that include national notifiable diseases data, since different data versions were used for analysis. In this report, disease notifications primarily consist of cases with a date of diagnosis between 1 January 2007 and 31 December 2010 (4 years), as at August 2011. Historical notification trend data included in this report have been updated from previous reports in this series.[13,132] Previous reports on data prior to 2005 analysed notifications by date of onset as collected from the clinical history, where available, or the specimen collection date for laboratory-reported cases. As of mid-2005, a date of diagnosis field was generated for all NNDSS records. For each notification record, a date of diagnosis is derived from the date of onset, or, where that is not supplied, the earliest date recorded among the following fields: date of specimen, date of notification, or date when the notification was received (the only mandatory date field).[16]

The variables extracted for analysis of each disease were: date of diagnosis, Indigenous status, age at diagnosis, and the state or territory from which the notification was received. Data for specific serotypes/serogroups of the causal organisms have been presented for invasive pneumococcal disease and meningococcal disease. Following an assessment of the completeness of reporting in the Indigenous status data field (see below), all jurisdictions were included for reporting of VPD notifications in this report for the period 2007 to 2010. This contrasts with previous reports in this series where notification data from only selected jurisdictions were analysed and reported.

Notification data are presented for invasive Hib disease, hepatitis A, acute hepatitis B, measles, meningococcal disease and pneumococcal disease in their respective chapters, and rare diseases (diphtheria, tetanus, poliomyelitis and rubella) in a combined chapter. Summary data are presented in Appendix C. No notification data are presented for influenza, mumps or pertussis, due to the low level of completeness of the Indigenous status field across most jurisdictions. Data on rotavirus and HPV disease notifications have not been included in this report as these diseases are not nationally notifiable. Notification data for varicella-zoster infections (including chickenpox and herpes zoster) have not been included in this report because national notifications are not available prior to vaccine funding, and due to the low level of completeness of Indigenous status in these data.

Indigenous status identification in notification data

The proportion of notifications that lack identification of Indigenous status was examined by jurisdiction, year and disease. An acceptable level of completeness of Indigenous status identification in the records was defined as at least 60% for a substantial majority of the diseases analysed. This level of completeness was achieved for all jurisdictions during this reporting period of 2007–2010. After establishing that notification incidence estimates were not dominated by any one of the jurisdictions (data not shown), estimates are presented for all jurisdictions combined.

Tasmania and the Australian Capital Territory were excluded from the disease trend data and graphs covering the period 2000–2010, due to varying but generally low levels of Indigenous status identification over the whole of this period.

Other issues to be noted when interpreting notification data

A major limitation of the notification data is that they represent only a proportion of all the cases occurring in the community, due to under-reporting. This proportion may vary between diseases, over time, and across jurisdictions.[16] An infectious disease that is diagnosed by a laboratory test is more likely to be notified than if it is diagnosed only on clinical grounds. Data accuracy may also vary among jurisdictions due to the use of different case definitions for surveillance (prior to adoption of the national case definitions) and varying reporting requirements and mechanisms by medical practitioners, hospitals and laboratories.

Hospitalisations

The AIHW National Hospital Morbidity Database has received administrative, demographic and clinical information about patients admitted to public and private hospitals in Australia since 1993. Almost all hospitalisation episodes in public and private hospitals are captured.[139] Data are received by financial year of hospital 'separation' (the process by which an admitted patient completes an episode of care by being discharged, dying, transferring to another hospital, or changing type of care).[139] The four most recent (financial) years for which data were available (2005/2006, 2006/2007, 2007/2008, 2009/2010) are included

in this report. Available data for analysis were final, and future revisions are not anticipated. Trend data for pneumococcal disease and 'influenza and pneumonia' by age group have been analysed and presented by calendar years rather than financial years to facilitate comparison with notification data, taking into account the seasonality of these conditions.

Data from jurisdictions in which Indigenous status identification of hospitalisation records exceeded 80% within the reporting periods are included for reporting of hospital rates and rate trends by Indigenous status, in accordance with AIHW recommendations[136,139] (see 'Indigenous status identification in hospitalisation data' below). For hospitalisation records where data on the jurisdiction of residence are missing, the jurisdiction in which the hospitalisation occurred is used to replace the jurisdiction of residence datum.

Data were extracted based on the International Statistical Classification of Diseases and Related Health Problems, 10th Revision, Australian Modification (ICD-10-AM). Most states and territories began using ICD-10-AM in 1998/1999, and since 1999/2000, all jurisdictions use the new classification. The codes used to select the specific condition(s) for reporting of each of the included VPDs are described in Appendix B and/or in the respective disease chapter. Eligible separations included those with the code of interest listed in the principal diagnosis (the diagnosis chiefly responsible for the episode of hospital admission) or in any other additional diagnosis fields (i.e. conditions or complaints either coexisting with the principal diagnosis or arising during the episode of care).[139] For hepatitis B, only hospitalisation records with acute hepatitis B as the principal diagnosis were included, consistent with previous practice in this report series and the series of national surveillance reports on VPDs.[13,24,132,140-143]

The variables extracted for analysis of each disease were: age at admission, state or territory of residence, Indigenous status, year of separation, and separation (discharge) diagnoses (principal and additional diagnoses – up to 31 diagnoses prior to 2003/2004, and up to 50 diagnoses since 2003/2004, were recorded for each hospital separation).

Hospitalisation data are presented for hepatitis A, acute hepatitis B, influenza, influenza and pneumonia, measles, meningococcal disease, mumps, pertussis, pneumococcal disease, rotavirus and varicella-zoster infections (chickenpox and herpes zoster, separately) in their respective chapters, and rare diseases (diphtheria, tetanus, poliomyelitis, and rubella) in a combined chapter. Summary data are presented in Appendix D. No hospitalisation data are presented for invasive Hib disease, as there is no type-specific code for invasive Hib disease within the ICD-10 classification system.

HPV

HPV hospitalisations are not included in this report, as the conditions measured by hospitalisation are of limited value in monitoring HPV-related disease. The most appropriate indicators are Pap test abnormalities and genital warts. The relevant datasets for these are outside the scope of this report and they have been reported on elsewhere.[144,145] However, data on Aboriginal and Torres Strait Islander people has not been published.

Indigenous status identification in hospitalisation data

The previous (second) report in this series included aggregated hospital separation data from five jurisdictions: New South Wales, the Northern Territory, Queensland, South Australia and Western Australia.[13] In this report, data from six Australian jurisdictions (all except Tasmania and the Australian Capital Territory) were included for reporting of aggregated hospital separations and rates by Indigenous status for the 4-year period July 2005 to June 2010, as recommended by the AIHW.[136] (About 96% of the Aboriginal and Torres Strait Islander population resides in these six jurisdictions.) Subsequently, it has been recommended that data from 2011–2012 onwards may include all jurisdictions.[146] For trend reporting for the period 1999/2000–2009/2010, data were included for only four jurisdictions (the Northern Territory, Queensland, South Australia and Western Australia), where Indigenous status data quality has been demonstrated as satisfactory over the whole period. (About 60% of the Aboriginal and Torres Strait Islander population resides in these four jurisdictions.) This data inclusion/exclusion selection is consistent with AIHW recommendations regarding acceptable data quality for analysis based on Indigenous identification.[136,139] Data from these selected jurisdictions are not necessarily representative of Aboriginal and Torres Strait Islander people living in other jurisdictions.[139]

Jurisdictional differences in data quality, including the degree of Indigenous under-identification, should be considered when interpreting the results. The analysis of hospitalisation rates over time should also be interpreted with caution, as hospitalisation rates for Aboriginal and Torres Strait Islander patients may be affected to a varying degree by improved identification over the period being analysed.[133,136]

Other issues eto be noted when interpreting hospitalisation data

Hospitalisations generally represent the more severe end of the morbidity spectrum of a disease, and the extent to which ICD-coded hospitalisation data can reflect the burden of the disease of interest varies between diseases.

There are also limitations associated with the use of ICD codes to identify cases. Errors that cause the ICD code to differ from the true disease include both random and systematic measurement errors. These errors may occur either along the patient pathway (e.g. level of details documented in medical records, clinicians' experience) or along the paper trail (e.g. transcribing errors, coder errors such as miss-specification, unbundling (assigning codes for all the separate parts of a diagnosis rather than the overall diagnosis) and upcoding (using reimbursement values to determine the order of coding)).[147] It is difficult to gauge the relative importance of hospitalisations where the coded disease of interest was not the principal diagnosis but was recorded as an additional diagnosis for that hospitalisation episode.

In the National Hospital Morbidity Database, there is one record for each hospital admission/separation episode. This means that there are separate records for each readmission or inter-hospital transfer. This is unlikely to have a major impact on the numbers reported for most diseases reviewed in this report, as they are mostly acute illnesses, but the implications of the potential but unquantified impact of this limitation need to be considered for each VPD individually.

Hospitalisation data may also be affected by variations in admission practices over time, between public and private sectors, and across various states and territories.[139] Variation in availability and access to hospitals across different geographic regions should also be noted when comparing and interpreting hospitalisation data for different population groups.

Deaths

The registration of deaths is the responsibility of the eight individual state and territory Registrars of Births, Deaths and Marriages. As part of the registration process, information on the cause of death is supplied by the medical practitioner certifying the death, or by a coroner. The information is provided by individual Registrars to the ABS for coding and compilation into aggregate statistics. In addition, the ABS supplements this data with information from the National Coroners Information System.[148]

Since 1997, the International Classification of Diseases, 10th Revision (ICD-10) has been used to identify the cause of death. The problems associated with the accuracy of ICD coding used for hospital separations, discussed above, may also be relevant for mortality data. The codes used to select the specific condition(s) for reporting of each of the included VPDs are described in Appendix B.

Unit file records of registered deaths were not available for analysis for this report. Unpublished mortality data on selected VPDs, by Indigenous status, aggregated for the period 2006–2010 were obtained directly from the ABS. In this current data release, 2006, 2007 and 2008 data are final, but 2009 and 2010 data are subject to further revisions.[148]

Due to incomplete identification of Indigenous status in the records, data on deaths in Aboriginal and Torres Strait Islander people are included from five jurisdictions only (New South Wales, the Northern Territory, Queensland, South Australia and Western Australia), as per recommendations of the ABS.[148] In November 2010, the Queensland Registrar of Births, Deaths and Marriages advised the ABS of an initiative which resulted in the registration (in 2010) of some 374 previously unregistered deaths that occurred between 1992 and 2006 or at an unknown time. Of these, around three-quarters (284) were deaths of Aboriginal and Torres Strait Islander Australians.[148] After consulting with the ABS, it was decided that, for the purposes of this report, these death records would be excluded from the dataset from which deaths attributed to VPDs were derived.

This report includes data on death records where the disease of interest was documented as the 'underlying cause of death' (i.e. the single disease that 'initiated the train of morbid events leading directly to death'),[148] and separately where a disease was one of the multiple causes of death (i.e. 'either the underlying cause, the

immediate cause, or any intervening causes, and those conditions which contributed to death but were not related to the disease or condition causing death').[148] In this report, deaths where the disease was one of the multiple causes, but not the underlying cause, are referred to as a 'contributing' cause.

As per ABS requirements to protect the confidentiality of individuals,[148] some of the exact counts of <5 deaths in aggregate cannot be reported, but instead are reported as a range. Additional counts have to be reported in ranges to prevent possible back calculations. For diseases where the total number (or subtotal by age group) of recorded deaths from selected jurisdictions is small, only the total counts or range for all ages by Indigenous status are reported. Where possible, for selected diseases, death counts are reported by three age groups (<5 years, 5–49 years and ≥50 years) by Indigenous status.

Other issues

Mortality data are reported and analysed by the year in which the death was registered rather than by the year in which the death occurred. This avoids problems associated with incomplete data for the latest available year. In recent years, less than 5% of deaths in a particular calendar year are registered in the subsequent year,[149] the bulk of which are deaths that occurred in December of that calendar year.

In Australia, information on the cause of death is reported routinely for every death on a standard Medical Certificate of Cause of Death completed by a medical practitioner or a coroner. The person completing the certificate must nominate the underlying (principal) cause of death and any associated conditions.[149] The accuracy in ascertaining the cause of death may vary according to the experience of the practitioner, the complexity of the disease process, the circumstances of the death, and whether post-mortem autopsy was performed. Studies comparing clinical and autopsy diagnoses have found that infectious diseases were not uncommonly a missed or discordant diagnosis, although vaccine preventable diseases were not specifically identified.[150,151] In the case of pertussis and tetanus, studies have documented that deaths due to these diseases, which can be otherwise identified through disease surveillance systems and hospitalisation records, sometimes go unrecorded on death certificates.[152,153]

Calculations and statistical methods

Calculation of rates

All rates were calculated using the mid-year estimated resident populations released by the ABS as the population denominator. Rates are presented as annual rates or average annual rates per 100,000 total population, or population by Indigenous status and by age groups as appropriate.

Age-standardised rates by Indigenous status, for all age groups in aggregate, are also reported. The direct standardisation method is used to calculate rates for all age groups combined, using the ABS 2006 population estimates as the standardising population. Interpretation and comparison of the standardised rates for each disease between 'Indigenous' and 'Other' Australians should take into account the limitations of age-standardised rates in representing the overall disease burden, including the issues arising from small number of events, age distribution of events, misclassification, population size and distribution, and under-identification of Indigenous status. Accordingly, rates were not standardised when case numbers were less than 20.[154]

Rate ratios for Indigenous versus non-Indigenous Australians were calculated for most of the reported diseases, including age-specific rate ratios, where appropriate.

STATA version12 was used for statistical analysis and to calculate 95% confidence intervals for rates. The method of Draper was used for confidence intervals for age-standardised rates.[155]

It is also important to note that high disease rates may be observed even with small absolute numbers of cases in jurisdictions with small populations (e.g. the Australian Capital Territory, the Northern Territory, Tasmania), and a small change in the numbers may result in a relatively large change in rates.

The statistical significance of time trends was assessed based on whether or not confidence intervals for individual years overlapped.

Population denominators for calculation of rates

For notification data, all rates were calculated using the mid-year estimated resident populations for the corresponding calendar year for the respective age group and/or jurisdiction as the population denominator.

For hospitalisation data, the mid-year population estimate for the first half of the financial year was used as the denominator; for example, the 2009 mid-year population estimate is used to calculate rates for 2009/2010. (This is consistent with previous reports in this series.)

Estimates from the B series of ABS experimental estimates and projections, Aboriginal and Torres Strait Islander Australians, 1991 to 2021 (based on the 2006 Census)[156] were used as the population denominators for calculations of rates for Indigenous people. This differs from AIHW recommendations to use the most recent Census with a year ending in one (1), i.e. 2001.[154] Therefore, if age-standardised rates are produced by other organisations that cover the same time periods and groups of ICD codes as this report, there may be some inconsistencies between the two. However, it was thought more appropriate to use the 2006 Census as those population estimates are closer to the periods analysed in this report.

The population denominators for calculation of rates for 'other' (presumed non-Indigenous) people were derived by subtracting the corresponding estimates for Aboriginal and Torres Strait Islander population of the relevant jurisdictions and age groups from the estimates of the total ABS-estimated resident population as at June of the corresponding year (based on latest estimates as at November 2011).

A.2 Vaccination coverage data

Calculating vaccination coverage estimates from the Australian Childhood Immunisation Register

The cohort method for calculating vaccination coverage was applied to ACIR data to measure vaccination coverage. This method has been routinely used in Australia for the reporting of vaccination coverage, and details have been previously published.[116] For the purpose of this report, 12-month birth cohorts have been used. Each cohort includes children born between 1 January and 31 December for each respective 12-month period, 2007, 2008, 2009 and 2010. Vaccination coverage has previously been reported for children at 12, 24 and 72 months of age.[13] Since 2008, 'fully vaccinated' coverage estimates have been reported at 60 months of age rather than 72 months.[157] The assessment date for determining vaccination coverage was at least 3 months following each milestone age for the cohort, to allow for delayed reporting to the ACIR.

For the purpose of calculating vaccination coverage estimates, only children who were registered with Medicare were included in the analysis, to minimise the potential for duplicate records in the ACIR. As per previous coverage reports, the 'third-dose assumption', by which record of receipt of a later dose of a vaccine with a multiple dose schedule implies receipt of earlier doses of the schedule, was used.[116] Evaluation of this assumption has concluded that it is appropriate for the reporting of population vaccination coverage rates.[158,159]

Vaccination coverage for Aboriginal and Torres Strait Islander children was calculated by dividing the numerator (children registered with Medicare as Aboriginal and Torres Strait Islander and reported as vaccinated) by the denominator (total number of children registered with Medicare as Aboriginal and Torres Strait Islander) within each birth cohort. Children for whom Aboriginal and Torres Strait Islander status was missing or unknown on their ACIR record have been categorised as other for the purpose of this report. Vaccination coverage of these children was calculated in a similar manner. The definition of a child being 'fully vaccinated' is based on the National Immunisation Program (NIP) schedule as per *The Australian Immunisation Handbook, 9th edition*.[9] Additional vaccines will be included in this calculation from July 2013, and results for these extended definitions are also presented (Table A.1.1).

Vaccination coverage expressed as children 'fully vaccinated' was calculated for Aboriginal and Torres Strait Islander and other children to allow for comparison. The standard definition of 'fully vaccinated' at 12 months of age is documented receipt on the ACIR of a third dose of a diphtheria-, tetanus- and acellular pertussis-containing vaccine; a third dose of polio vaccine; a second or third dose of a PRP-OMP-containing vaccine or a third dose of any other Hib vaccine; and a second or third dose of a Comvax hepatitis B vaccine or a third dose of any other hepatitis B vaccine.

The extended definition for 'fully vaccinated' at 12 months of age is as per the standard definition, but with the addition of a third dose of 7vPCV (as per the definition applicable from July 2013), and also with the addition of a second or third dose of rotavirus vaccine.

The standard definition for 'fully vaccinated' at 24 months of age is documented receipt on the ACIR of a third dose of a diphtheria-, tetanus- and acellular pertussis-containing vaccine; a third dose of polio vaccine; a third or fourth dose of a PRP-OMP-containing vaccine or a fourth dose of any other Hib vaccine; a third or fourth dose of Comvax hepatitis B vaccine or a fourth dose of any other hepatitis B vaccine; and a first dose of a measles-, mumps- and rubella-containing vaccine.

The extended definition for 'fully vaccinated' at 24 months of age is as per the standard definition, but with the addition of a first dose of varicella vaccine and a first dose of meningococcal C vaccine, as per the definition applicable from July 2013.

The definition used for 'fully vaccinated' at 60 months of age is documented receipt on ACIR of a fourth or fifth dose of a diphtheria-, tetanus- and acellular pertussis-containing vaccine; a fourth dose of polio vaccine; and a second dose of a measles-, mumps- and rubella-containing vaccine.

The differing number of doses in the inclusion criteria for various vaccines (such as Hib and hepatitis B vaccines) corresponds to the recommended schedule for the specific formulation of vaccine that a child received.

Vaccination coverage was also calculated for selected individual vaccines provided under the NIP, including conjugate and polysaccharide pneumococcal vaccines and hepatitis A, rotavirus and MMR vaccines.

Calculating vaccination timeliness from the Australian Childhood Immunisation Register

In addition to vaccination coverage, we report timeliness of vaccination. As per other national reports,[157] age-appropriate vaccination was defined as receipt of a scheduled vaccine dose within 30 days of the schedule point (age) recommended in The Australian Immunisation Handbook.[160]

For the purpose of reporting, vaccine doses given too early or not reported were excluded from the analysis. The proportion of children who received the dose within 30 days of the schedule point were defined as age-appropriately immunised. Remaining children were categorised by the length of time between the vaccine schedule point and when the vaccine dose was administered (i.e. vaccine received 1–2 months after schedule point, 3–6 months after schedule point, or >6 months after schedule point).

In response to high rates of pertussis infection in Australia since 2008,[161] the Australian Technical Advisory Group on Immunisation, in February 2011, endorsed the recommendation to bring forward the first dose of DTP vaccine for infants from 8 weeks (2 months) of age to 6 weeks of age. To assess the implementation of this intervention, timeliness of the first dose of DTP was calculated, comparing the proportion vaccinated at 42–60 days of age with the proportion vaccinated at 61–92 days of age.

Geographic variations of vaccination coverage

Vaccination coverage estimates for being 'fully vaccinated' at 12 months of age by Australian Statistical Division (SD) were used as an indicator for geographic variations of vaccination coverage among Aboriginal and Torres

Table A.1.1: Inclusion criteria for 'fully vaccinated' definitions, by milestone

'Fully vaccinated' category	Vaccine								
	DTPa	Polio	Hib	HepB	MMR	MenC	7vPVC	Rotavirus	Varicella
12 months – old*	✓	✓	✓	✓					
12 months – new†	✓	✓	✓	✓			✓		
12 months – new inc. rotavirus	✓	✓	✓	✓			✓	✓	
24 months – old*	✓	✓	✓	✓	✓				
24 months – new†	✓	✓	✓	✓	✓	✓			✓
60 months	✓	✓			✓				

* Applicable from 1996 to June 2013

† Applicable from July 2013

Strait Islander children. ACIR child records were allocated to SDs by the postcode of residence recorded in the ACIR. Coverage estimates were calculated for each SD using the methods described previously; results were then mapped.

Vaccination coverage data and timeliness data were analysed by remoteness status based on the Accessibility/Remoteness Index of Australia (ARIA), developed by the Australian Government Department of Health.[162] The ARIA system is based on the road distance of a location to the nearest service centre, with locations categorised into five groups. For the purpose of this report, the two groups with the most restricted access to services (remote and very remote) are classified as 'remote'; the other three categories (highly accessible, accessible and moderately accessible) are classified as 'accessible'. Vaccine timeliness of the second dose of DTPa (DTP2) and the first dose of MMR (MMR1) was assessed for both remote and non-remote categories.

Vaccine refusal

The parents of Medicare registered children may lodge a conscientious objection to immunisation if they do not wish their children to be immunised. The percentage of conscientious objectors was measured by dividing the total number of objectors by the total number of Medicare registered children. The proportion of conscientious objectors was calculated from children born in the cohort from January 2004 to December 2008, to allow a large enough sample for statistical accuracy and sufficient time for parents to have lodged a conscientious objection form with Medicare. The proportions of conscientious objectors among Aboriginal and Torres Strait Islander and other children were compared.

Data quality and notes on interpreting coverage data

General under-reporting in the Australian Childhood Immunisation Register

It should be noted that there is a potential for general under-reporting of children and vaccine doses to the ACIR. Firstly, vaccination coverage estimates do not account for children not registered with Medicare; however, the impact of this is minimal. It is reported that by 12 months of age 99% of Australian children have been registered with Medicare.[115] Secondly, the ACIR is dependent on reporting of vaccination doses by service providers. Providers report to the ACIR via the Medicare Australia website or through submission of a paper form which is then centrally updated to the ACIR.[160] A national population-based survey was conducted in 2001 to measure the proportion of under-reporting to the ACIR and to calculate corrected vaccination coverage estimates. The study found that at 12 months of age the ACIR underestimated vaccination coverage by 2.7% (95% CI 2.4–3.0) and at 24 months of age by 6.5% (95% CI 6.1–6.9).[163] However, since the time of this study, incentives have been introduced for both parents and providers to encourage reporting of vaccination to the ACIR (detailed elsewhere)[160] and rates of electronic reporting have increased markedly. The impact of these incentives on reporting to the ACIR by service providers and the subsequent effect on vaccination coverage estimates has not been evaluated.

Indigenous status identification in the Australian Childhood Immunisation Register

In addition to general under-reporting, completion and accuracy of Indigenous status in the ACIR has, in the past, been suboptimal. Despite past data quality issues, significant improvements to data quality have been made since the inception of the ACIR. The first national surveillance report on vaccine preventable diseases and vaccination coverage in Aboriginal and Torres Strait Islander Australians, which covered the period 1999 to 2002, analysed coverage data from New South Wales, the Northern Territory, South Australia, Victoria and Western Australia only, as Indigenous status reporting was inadequate in other states.[132] Indigenous status identification greatly improved between 2003 and 2004 and in the second of these reports, for the period 2003 to 2006, data quality had improved enough to allow all states and jurisdictions to be included in the analysis.[13]

The improvement in data quality is supported by several studies. Vlack et al. demonstrated that Aboriginal and Torres Strait Islander coverage estimates obtained from the ACIR were comparable to estimates provided by a survey of Aboriginal and Torres Strait Islander children in Queensland.[164] Rank and Menzies demonstrated that Aboriginal and Torres Strait Islander status identification in the ACIR increased from 42% of the national cohort of Aboriginal and Torres Strait Islander children aged 12–14 months in 2002 to 95% in 2005.[135]

Appendix B: Case definitions in effect during data collection period

NNDSS case definitions are also available at www.health.gov.au/casedefinitions, and previous versions described in Vaccine preventable diseases in Australia, 2005 to 2007.[24]

Diphtheria

Notifications

Both confirmed cases and probable cases are notifiable. A confirmed case requires laboratory definitive evidence only. A probable case requires laboratory suggestive evidence and clinical evidence, or clinical evidence and an epidemiological link to a laboratory-confirmed case.

Laboratory definitive evidence:

- Isolation of toxigenic *Corynebacterium diphtheriae* or toxigenic *C. ulcerans*.

Laboratory suggestive evidence:

- Isolation of *C. diphtheriae* or *C. ulcerans* (toxin production unknown).

Clinical evidence:

At least one of the following:

- pharyngitis and/or laryngitis (with or without) membrane), or
- toxic (cardiac or neurological) symptoms.

Hospitalisations and deaths

The ICD-10-AM/ICD-10 code A36 (diphtheria) was used to identify hospitalisations and deaths.

Hib

Notifications

Invasive Hib infection

Only confirmed cases are notifiable. A confirmed case requires laboratory definitive evidence only.

Laboratory definitive evidence:

- Isolation of *Haemophilus influenzae* type b from a normally sterile site where typing has been confirmed at an approved reference laboratory, or
- Detection of Hib antigen in cerebrospinal fluid when other laboratory parameters are consistent with meningitis.

Hospitalisations and deaths

There were no ICD-10-AM/ICD-10 codes which specified Hib as a causative organism. The ICD-10-AM/ICD-10 code used to identify presumed Hib cases was G00.0 (*Haemophilus meningitis*). The ICD-10-AM/ICD-10 codes for *H. influenzae* pneumonia, *H. influenzae* septicaemia, *H. influenzae* infection and acute epiglottitis were not included as these were considered insufficiently specific for invasive *H. influenzae* type b disease.

Hepatitis A

Notifications

Both confirmed cases and probable cases are notifiable. A confirmed case requires laboratory definitive evidence only. A probable case requires clinical evidence and an epidemiological link to a laboratory-confirmed case.

Laboratory definitive evidence:

- Detection of anti-hepatitis A virus IgM antibody (in the absence of recent vaccination), or
- Detection of hepatitis A virus by nucleic acid testing.

Clinical evidence:

- Clinical hepatitis (jaundice and/or bilirubin in urine) without a non-infectious cause.

Hospitalisations and deaths

The ICD-10-AM/ICD-10 code B15 (hepatitis A) was used to identify hospitalisations and deaths.

Acute hepatitis B (newly acquired)

Notifications

Only confirmed cases are notifiable. A confirmed case requires laboratory definitive evidence only.

Laboratory definitive evidence:

- Detection of hepatitis B surface antigen (HBsAg) in a patient shown to be negative within the last 24 months, or
- Detection of HBsAg and IgM to hepatitis B core antigen, in the absence of prior evidence of hepatitis B virus infection, or
- Detection of hepatitis B virus by nucleic acid testing, and IgM to hepatitis B core antigen, in the absence of prior evidence of hepatitis B virus infection.

Hospitalisations

The ICD-10-AM code B16 (acute hepatitis B) was used to identify hospitalisations. As in the previous reports, hospitalisations were included only where the relevant ICD code was the principal diagnosis.

Deaths

The ICD-10 code B16 (acute hepatitis B) was used to select deaths from acute hepatitis B.

* Queensland implemented a consistent but less comprehensive definition for laboratory notification in December 2005 for 'Hepatitis B (acute)': HBsAg positive *and* anti-HBc IgM positive. However, the public heath protocol for notification in Queensland accepts cases meeting the broader national case definitions for notification.

Influenza

Notifications

Only confirmed cases are notifiable. A confirmed case requires laboratory definitive evidence only. Laboratory definitive evidence:

- Isolation of influenza virus by culture from appropriate respiratory tract specimen, or
- Detection of influenza virus by nucleic acid testing from appropriate respiratory tract specimens, or
- Detection* of influenza virus antigen from appropriate respiratory tract specimen, or
- IgG seroconversion or a significant increase in antibody level or a 4-fold or greater rise in titre to influenza virus, or
- Single high titre* to influenza virus.

Laboratory-confirmed influenza only became notifiable in South Australia in May 2008.

Hospitalisations

The ICD-10-AM codes used to identify hospitalisations were J09 (influenza due to certain identified influenza viruses), J10 (influenza due to identified influenza virus) and J11 (influenza, virus not identified). In this report, we did not make the distinction between admissions where a virus was identified and those where it was not.

Deaths

The ICD-10 codes used to identify deaths were: J09 (influenza due to certain identified influenza viruses), J10 (influenza due to identified influenza virus) and J11 (influenza, virus not identified).

* The criteria of the national case definitions were revised and implemented in 2008. The revisions consisted of specifying that the influenza virus antigen detection has to be laboratory detection, and that the single high titre qualifying for notification has to be performed by complement fixation test (CFT) or by haemagglutination inhibition (HAI).

Measles

Notifications

Both confirmed cases and probable cases are notifiable. A confirmed case requires laboratory definitive evidence, or clinical evidence and an epidemiological link to a laboratory-confirmed case. A probable case requires laboratory suggestive evidence and clinical evidence.

Laboratory definitive evidence:

- Isolation of measles virus, or
- Detection of measles virus by nucleic acid testing, or
- Detection of measles virus antigen, or

Measles virus-specific IgG seroconversion or significant increase in IgG antibody level or a 4-fold or greater rise in antibody titre to measles virus, with paired sera tested in parallel and in the absence of receipt of measles-containing vaccine 8 days to 8 weeks prior to testing, or

Detection of measles virus-specific IgM antibody confirmed in an approved reference laboratory (in the absence of recent measles-containing vaccination).

Laboratory suggestive evidence:

- Detection of measles-specific IgM antibody other than by an approved reference laboratory (in the absence of recent measles-containing vaccination).

Clinical evidence:

- A clinical illness characterised by a generalised maculopapular rash lasting at least 3 days and fever of at least 38°C at the time of rash onset and cough, coryza, conjunctivitis or Koplik spots.

Hospitalisations and deaths

The ICD-10-AM/ICD-10 code B05 (measles) was used to identify hospitalisations and deaths. Subacute sclerosing panencephalitis (SSPE) was not included in these analyses.

Meningococcal disease

Notifications

Both confirmed cases and probable cases are notifiable. A confirmed case requires laboratory definitive evidence, or laboratory suggestive evidence and clinical evidence. A probable case requires specified clinical evidence only.

Laboratory definitive evidence:

- Isolation of *Neisseria meningitidis* from a normally sterile site, or
- Detection of specific meningococcal DNA sequences in a specimen from a normally sterile site by nucleic acid amplification testing.

Laboratory suggestive evidence:

- Detection of Gram-negative diplococci in Gram stain of specimen from a normally sterile site or from a suspicious skin lesion, or
- High titre IgM or significant rise in IgM or IgG titres to outer membrane protein antigens of *N. meningitidis*.

Clinical evidence for a confirmed case:

- Disease which in the opinion of the treating clinician is compatible with invasive meningococcal disease.

Clinical evidence for a probable case:

- The absence of evidence for other causes of clinical symptoms and either clinically compatible disease including haemorrhagic rash, or clinically compatible disease and close contact with a confirmed case within the previous 60 days.

Hospitalisations and deaths

The ICD-10-AM/ICD-10 code A39 (meningococcal infection) was used to identify hospitalisations and deaths. This includes meningococcal meningitis (A39.0), Waterhouse-Friderichsen syndrome (A39.1), acute meningococcaemia (A39.2), chronic meningococcaemia (A39.3), meningococcaemia unspecified (A39.4), meningococcal heart disease (A39.5), other meningococcal infections (A39.8), and meningococcal infection unspecified (A39.9).

Mumps

Notifications

Only confirmed cases are notifiable. A confirmed case requires laboratory definitive evidence, or laboratory suggestive evidence and clinical evidence, or clinical evidence and an epidemiological link to a laboratory-confirmed case.

Laboratory definitive evidence:

- Isolation of mumps virus, or
- Detection of mumps virus by nucleic acid testing, or
- IgG seroconversion or a significant increase in antibody level or a 4-fold or greater rise in titre to mumps virus (in the absence of recent mumps-containing vaccination).

Laboratory suggestive evidence:

- Detection of mumps-specific IgM antibody (in the absence of recent mumps-containing vaccination).

Clinical evidence:

- A clinically compatible illness characterised by swelling of the parotid or salivary glands lasting 2 days or more without other apparent cause.

Hospitalisations and deaths

The ICD-10-AM/ICD-10 code B26 (mumps) was used to identify hospitalisations and deaths.

Pertussis

Notifications

Both confirmed cases and probable cases are notifiable. A confirmed case requires laboratory definitive evidence, or laboratory suggestive evidence and clinical evidence, or clinical evidence and an established epidemiological link to a confirmed case with laboratory evidence. A probable case requires specified clinical evidence only.

Laboratory definitive evidence:

- Isolation of *Bordetella pertussis* from a clinical specimen, or
- Detection of *B. pertussis* by nucleic acid testing.

Laboratory suggestive evidence:

- Seroconversion or a significant increase in antibody level or a 4-fold or greater rise in titre to *B. pertussis* (in the absence of recent pertussis vaccination), or
- Single high IgA titre to whole cells, or
- Detection of *B. pertussis* antigen by immunofluorescence assay (IFA).

Clinical evidence for a confirmed case:

- A coughing illness lasting 2 or more weeks, or
- Paroxysms of coughing or inspiratory whoop or post-tussive vomiting.

Clinical evidence for a probable case:

- A coughing illness lasting 2 or more weeks, and
- Paroxysms of coughing or inspiratory whoop or post-tussive vomiting.

Hospitalisations and deaths

The ICD-10-AM/ICD-10 code A37 (whooping cough) was used to identify hospitalisations and deaths.

Pneumococcal disease (invasive)

Notifications

Only confirmed cases are notifiable. A confirmed case requires laboratory definitive evidence only.

Laboratory definitive evidence:

- Isolation of *Streptococcus pneumoniae* from a normally sterile site by culture, or
- Detection of *S. pneumoniae* from a normally sterile site by nucleic acid testing.

Hospitalisations

The ICD-10-AM codes used to identify hospitalisations were: G00.1 (pneumococcal meningitis); A40.3 (pneumococcal septicaemia) (together considered to be a proxy for invasive pneumococcal disease); and J13 (pneumococcal pneumonia). To avoid double counting, cases were identified in a hierarchical fashion. First, all those with code G00.1 were classified as meningitis, then those without G00.1 but with A40.3 were classified as septicaemia without meningitis, and then those with neither of these codes but with code J13 were counted as pneumococcal pneumonia.

Deaths

ICD-10 codes G00.1, A40.3 and J13 were used to select deaths attributed to IPD.

Poliomyelitis

Notifications

Both confirmed cases and probable cases are notifiable. A confirmed case requires laboratory definitive evidence and clinical evidence. A probable case requires clinical evidence and that the case is not discarded as non-polio paralytic illness by the Polio Expert Committee.

Laboratory definitive evidence:

- Isolation of wild poliovirus (or Sabin-like poliovirus for vaccine-associated paralytic poliomyelitis (VAPP) cases), confirmed in the WHO Western Pacific Regional Poliovirus Reference Laboratory, or
- Detection of wild poliovirus (or Sabin-like poliovirus for VAPP cases) by nucleic acid testing, confirmed in the WHO Western Pacific Regional Poliovirus Reference Laboratory.

Clinical evidence:

- Acute flaccid paralysis: acute onset of progressive weakness and flaccidity of one or more limbs with decreased or absent tendon reflexes in the affected limbs or bulbar palsy without other apparent cause, and without sensory or cognitive loss.

Hospitalisations and deaths

The ICD-10-AM/ICD-10 code A80 (acute poliomyelitis) was used to identify hospitalisations and deaths.

Note: This code includes VAPP and specific codes for Indigenous and imported wild-type poliovirus infection. Sequelae of poliomyelitis (ICD-10 code B91) were not included in these analyses.

Rotavirus

Hospitalisations and deaths

The ICD-10-AM/ICD-10 code A08.0 (rotaviral enteritis) was used to identify hospitalisations and deaths.

Rubella

Notifications

Both confirmed cases and probable cases are notifiable. A confirmed case requires laboratory definitive evidence only. A probable case requires clinical evidence and either laboratory suggestive evidence or an epidemiological link to a laboratory-confirmed case.

Laboratory definitive evidence:

- Isolation of rubella virus, or
- Detection of rubella virus by nucleic acid testing, or
- IgG seroconversion or a significant increase in antibody level or a 4-fold or greater rise in titre to rubella virus in the absence of recent rubella vaccination, in paired sera tested in parallel, or
- Detection of rubella-specific IgM antibody in the absence of recent rubella vaccination (in pregnant women, must be confirmed in a reference laboratory).

Laboratory suggestive evidence:

- In a pregnant patient, detection of rubella-specific IgM antibody that has not been confirmed in a reference laboratory (in the absence of recent rubella vaccination).

Clinical evidence:

- A generalised maculopapular rash and fever and one or more of arthralgia/arthritis or lymphadenopathy or conjunctivitis.

Hospitalisations and deaths

The ICD-10-AM/ICD-10 code B06 (rubella (German measles)) was used to identify hospitalisations and deaths.

Tetanus

Notifications

Only confirmed cases are notifiable. A confirmed case requires either laboratory definitive evidence or clinical evidence.

Laboratory definitive evidence:

- Isolation of *Clostridium tetani* from a wound in a compatible clinical setting and prevention of positive tetanospasm in mouse test from such an isolate using specific tetanus antitoxin.

Clinical evidence:

- A clinically compatible illness without apparent cause.

Hospitalisations and deaths

The ICD-10-AM/ICD-10 codes A34 (obstetrical tetanus) and A35 (other tetanus) were used to identify hospitalisations and deaths.

Varicella-zoster (Chickenpox)

Notifications

National definition from 2006

Both confirmed cases and probable cases are notifiable. A confirmed case requires laboratory definitive evidence and clinical evidence, or clinical evidence and an epidemiological link to a laboratory-confirmed case. A probable case requires clinical evidence only.

Laboratory definitive evidence:

- Isolation of varicella-zoster virus (VZV) from a skin or lesion swab, or
- Detection of VZV from a skin or lesion swab by nucleic acid testing, or
- Detection of VZV antigen from a skin or lesion swab by direct fluorescent antibody, or
- Detection VZV-specific IgM in an unvaccinated person.

If the case received varicella vaccine within 5 and 42 days prior to onset of rash, the virus must be confirmed to be a wild-type strain.

Clinical evidence:

- Acute onset of a diffuse maculopapular rash developing into vesicles within 24–48 hours and crusting over within 5 days.

Hospitalisations and deaths

The ICD-10-AM/ICD-10 code B01 (chickenpox) was used to identify varicella hospitalisations and deaths.

Varicella-zoster (Shingles)

Notifications

National definition from 2006

Both confirmed cases and probable cases are notifiable. A confirmed case requires laboratory definitive evidence and clinical evidence. A probable case requires clinical evidence only.

Laboratory definitive evidence:

- Isolation of varicella-zoster virus (VZV) from a skin or lesion swab, or
- Detection of VZV from a skin or lesion swab by nucleic acid testing, or
- Detection of VZV antigen from a skin or lesion swab by direct fluorescent antibody.

Clinical evidence:

- A vesicular skin rash with a dermatomal distribution that may be associated with pain in skin areas supplied by sensory nerves of the dorsal root ganglia.

Hospitalisations and deaths

The ICD-10-AM/ICD-10 code B02 (zoster (shingles)) was used to identify herpes zoster hospitalisations and deaths.

Appendix C: Summary of notifications in Australia, for vaccine preventable diseases, 2007 to 2010, by Indigenous status

Disease*	Indigenous status	Notifications† (2007–2010)		
		n	Rate‡	Rate ratio
Diphtheria	Indigenous	0	0.0	–
	Other	0	0.0	
Hib disease (invasive)	Indigenous	25	0.9	12.9
	Other	60	0.1	
Hepatitis A§	Indigenous	11	0.5	0.3
	Other	1,261	1.5	
Hepatitis B	Indigenous	72	3.5	3.1
	Other	951	1.1	
Measles§	Indigenous	3	0.1	0.5
	Other	248	0.3	
Meningococcal disease	Indigenous	104	3.2	2.7
	Other	975	1.2	
Invasive pneumococcal disease	Indigenous	698	42.0	3.6
	Other	5,606	11.5	
Poliomyelitis§	Indigenous	0	0.0	0.0
	Other	1	<0.01	
Rubella§	Indigenous	0	0.0	0.0
	Other	143	0.2	
Tetanus§	Indigenous	0	0.0	0.0
	Other	12	0.01	

* Varicella, mumps, pertussis, influenza excluded due to low Indigenous status completeness.

† Notifications (all jurisdictions) where the date of diagnosis was between 1 January 2007 and 31 December 2010.

‡ Rates are per 100,000 population for all ages combined, age-standardised to the Australian Bureau of Statistics Australian population estimates for 2006.

§ Rates not standardised due to low number of Indigenous cases as recommended by the Australian Institute of Health and Welfare.154

Appendix D: Summary of hospitalisations and deaths in Australia, for vaccine preventable diseases, 2005 to 2010, by Indigenous status

Disease*	Indigenous status	Hospitalisations† (July 2005–June 2010)			Deaths§ (2006–2010)
		n	Rate‡	Rate ratio	n
Diphtheria‖	Indigenous Other	47 55	1.9 0.1	33.5	0 1–4
Hepatitis A	Indigenous Other	48 1,031	1.6 1.0	1.6	0 6
Hepatitis B	Indigenous Other	31 680	1.6 0.7	2.2	6–9 23–26
Influenza	Indigenous Other	2,245 20,753	97.2 21.0	4.6	14 221
Influenza and pneumonia	Indigenous Other	29,734 532,439	1,604.2 527.2	3.0	183 7,696
Measles‖	Indigenous Other	6 133	0.2 0.1	1.8	0 1–4
Meningococcal disease	Indigenous Other	189 2,041	4.5 2.1	2.2	2–8 34–40
Mumps‖	Indigenous Other	43 329	1.7 0.3	5.1	0 1–4
Pertussis	Indigenous Other	362 3,410	10.0 3.5	2.9	1–4 6–9
Invasive pneumococcal disease	Indigenous Other	397 3,218	18.9 3.2	6.0	3–12 37–46
Poliomyelitis‖	Indigenous Other	1 22	0.04 0.06	0.7	0 0
Rotavirus	Indigenous Other	1,487 11,772	32.1 12.5	2.6	0 1–4
Rubella‖	Indigenous Other	6 134	0.2 0.1	1.8	0 0
Tetanus‖	Indigenous Other	2 85	0.08 0.09	0.9	0 1–4
Varicella	Indigenous Other	279 4,883	8.5 5.0	1.7	0 20
Zoster	Indigenous Other	376 25,231	26.0 23.8	1.1	1–4 71–74

* Hib disease is excluded because there is no type-specific code for hospitalisation. The code for *Haemophilus meningitis* was used as proxy to identify deaths recorded during the period between 2006 and 2010. There were no deaths due to *Haemophilus meningitis*.

† Hospitalisations (New South Wales, Northern Territory, Queensland, South Australia, Victoria, Western Australia only) where the date of separation was between 1 July 2005 and 30 June 2010.

‡ Rates are per 100,000 population for all ages combined, age-standardised to the Australian Bureau of Statistics Australian population estimates for 2006.

§ Underlying cause of death, recorded in New South Wales, Northern Territory, Queensland, South Australia, Western Australia only, from 2006 to 2010.

‖ Rates not standardised due to low number of Indigenous cases as recommended by the Australian Institute of Health and Welfare.[154]

Appendix E: Vaccine coverage, by state or territory and birth cohort

	ACT		NSW		Victoria		Queensland		South Australia		Western Australia		Tasmania		Northern Territory		Australia	
Coverage at 12 months of age (born January – December 2009)																		
	Indigenous	Others	Indigenous	Others	Indigenous	Others	Indigenous	Others	Indigenous	Others	Indigenous	Others	Indigenous	Others	Indigenous	Others	Indigenous	Others
Number of children	92	4,899	3,638	93,790	733	70,985	4,608	57,305	702	18,859	1,854	29,391	424	6,012	1,588	2,241	13,639	283,482
DTP 3 doses	87.0	94.9	87.4	92.6	84.5	93.0	86.3	92.8	79.5	92.7	81.1	91.3	88.4	92.6	88.4	92.7	85.7	92.6
Polio 3 doses	87.0	94.8	87.3	92.5	84.3	93.0	86.2	92.8	79.6	92.7	81.1	91.3	88.4	92.6	88.4	92.7	85.7	92.6
Hib (2 or 3 doses)	87.0	94.7	87.3	92.3	84.0	92.7	86.2	92.6	79.5	92.5	80.8	91.1	88.4	92.5	89.2	92.6	85.7	92.4
Hep B (2 or 3 doses)	87.0	93.7	87.3	92.1	83.9	92.3	86.2	92.3	79.8	92.1	81.0	90.6	88.4	92.4	88.4	92.2	85.6	92.1
7vPCV 3 doses	87.0	93.8	86.9	91.7	84.0	92.0	86.0	92.0	79.5	91.9	81.1	90.1	88.2	91.9	87.0	90.8	85.3	91.7
Rotavirus (2 or 3 doses)	77.2	88.0	80.7	86.6	68.1	83.5	66.6	82.9	63.5	84.4	74.2	86.0	83.3	86.4	72.1	88.2	71.7	85.1
'Fully vaccinated'*	87.0	93.6	87.2	91.9	83.9	92.1	86.2	92.1	79.1	92.0	80.6	90.4	88.4	92.3	88.0	91.7	85.5	91.9
'Fully vaccinated'† (inc. Rota and 7vPCV)	72.8	86.8	76.1	84.5	67.5	83.1	66.3	82.7	63.1	84.1	69.3	83.6	80.2	84.5	68.1	84.5	69.9	83.7
Coverage at 24 months of age (born January – December 2008)																		
	Indigenous	Others	Indigenous	Others	Indigenous	Others	Indigenous	Others	Indigenous	Others	Indigenous	Others	Indigenous	Others	Indigenous	Others	Indigenous	Others
Number of children	92	4,870	3,830	95,034	790	71,785	4,778	58,489	767	19,070	1,903	29,764	458	6,150	1,486	2,199	14,104	287,363
DTP 3 doses	97.8	96.0	94.3	94.7	94.4	95.3	94.5	94.4	92.8	94.4	92.0	93.3	94.3	95.2	95.2	94.5	94.1	94.7
Polio 3 doses	97.8	96.0	94.2	94.7	94.4	95.3	94.4	94.3	92.8	94.4	91.8	93.3	94.3	95.2	95.0	94.4	94.0	94.6
Hib (2 or 3 doses)	97.8	95.5	94.9	94.7	94.2	94.9	94.9	94.0	92.6	94.0	89.5	93.2	94.8	95.1	95.0	92.7	94.0	94.4
Hep B (2 or 3 doses)	97.8	95.1	94.2	94.0	94.2	94.5	94.4	93.7	93.0	93.8	91.6	92.4	94.1	94.9	95.0	93.8	94.0	93.9
MMR first dose	97.8	94.8	93.6	93.8	93.5	94.5	95.1	93.7	92.8	93.6	92.7	92.3	95.2	94.7	96.8	93.3	94.4	93.8
'Fully vaccinated'*	97.8	93.2	91.4	92.1	91.5	92.7	92.6	92.0	89.2	92.0	86.3	89.9	93.7	93.5	93.1	91.2	91.3	92.0
Men C 1 dose	96.7	94.1	93.3	93.3	93.4	94.1	94.9	93.2	92.6	93.4	91.3	91.6	94.1	94.7	96.5	93.2	93.9	93.3

Appendix E continued: Vaccine coverage, by state or territory and birth cohort

	ACT		NSW		Victoria		Queensland		South Australia		Western Australia		Tasmania		Northern Territory		Australia	
Varicella 1 dose	88.0	87.2	81.2	82.2	77.7	82.7	84.7	86.3	79.8	81.9	77.8	79.4	79.9	81.9	87.4	82.6	82.3	82.9
'Fully vaccinated'† (inc. Men C and Varicella)	88.0	85.3	78.6	80.1	76.0	80.9	82.3	84.8	76.5	80.3	72.4	77.3	77.7	80.5	84.4	80.6	79.4	81.1
Coverage at 60 months (born January – December 2005)																		
	Indigenous	Others	Indigenous	Others	Indigenous	Others	Indigenous	Others	Indigenous	Others	Indigenous	Others	Indigenous	Others	Indigenous	Others	Indigenous	Others
Number of children	81	4,575	3,140	90,703	777	67,545	3,970	56,878	625	18,144	1,812	27,971	342	5,923	1,493	2,074	12,240	273,813
MMR 2 doses	86.4	91.1	85.3	89.4	86.7	90.9	87.9	90.3	79.8	87.5	81.7	86.6	90.4	92.1	89.9	84.7	86.1	89.6
'Fully vaccinated'*	86.4	90.7	84.5	89.0	86.0	90.6	86.9	89.9	78.7	87.1	80.7	86.0	89.8	91.6	89.2	84.2	85.3	89.2
Vaccines specifically recommended for Indigenous children residing in jurisdictions of high disease incidence (Cohort born January – December 2008)																		
Hepatitis A 1 dose	-	-	-	-	-	-	68.1	-	57.4	-	74.4	-	-	-	88.4	-	71.9‡	-
Hepatitis A 2 doses	-	-	-	-	-	-	52.2	-	35.7	-	58.5	-	-	-	78.6	-	56.5‡	-
PPV 1 dose	-	-	-	-	-	-	57.8	-	39.6	-	55.2	-	-	-	81.8	-	59.7‡	-

* 'Fully vaccinated' definition from 1996 to June 2013: at 12 months of age – defined as receipt of 3 doses of diphtheria, tetanus, pertussis, Hib, hepatitis B and polio, but did not include rotavirus and pneumococcal vaccines, which are also due at the same schedule points; at 24 months of age – included 3 or 4 doses of Hib and hepatitis B, and 1 dose of measles, mumps, rubella, but did not include meningococcal C or varicella vaccines; at 5 years (60 months) – included a fourth dose of diphtheria, tetanus, pertussis, polio and a second dose of measles, mumps and rubella.

† 'Fully vaccinated' definition from July 2013: pneumococcal vaccine added at 12 months, varicella and meningococcal vaccines added at 24 months, unchanged at 60 months.

‡ Combined jurisdictions of high disease burden

Abbreviations

ABS	Australian Bureau of Statistics
ACIR	Australian Childhood Immunisation Register
ACT	Australian Capital Territory
AIHW	Australian Institute of Health and Welfare
ARIA	Accessibility/Remoteness Index of Australia
BCG	Bacille Calmette-Guérin
CRS	Congenital rubella syndrome
DTP	Diphtheria-tetanus-pertussis
DTPa	Diphtheria-tetanus-pertussis (acellular)
dTpa	Adolescent/adult diphtheria-tetanus-pertussis (acellular)
DTPw	Diphtheria-tetanus-pertussis (whole-cell)
Flu	Influenza
HAV	Hepatitis A virus
HbOC	*Haemophilus influenzae* type b polysaccharide conjugated to non-toxic diphtheria CRM197 protein
HBV	Hepatitis B virus
HepA	Hepatitis A (vaccine abbreviation)
HepB	Hepatitis B (vaccine abbreviation)
Hib	*Haemophilus influenzae* type b
HPV	Human papillomavirus
HZ	Herpes zoster
ICD	International Classificatizon of Diseases
ICD-10	International Classification of Diseases, 10th Revision
ICD-10-AM	International Statistical Classification of Diseases and Related Health Problems, 10th Revision, Australian Modification
ICU	Intensive care unit
IgA	Immunoglobulin A
IgG	Immunoglobulin G
IgM	Immunoglobulin M
IPD	Invasive pneumococcal disease
IPV	Inactivated poliomyelitis vaccine

MenCCV	Meningococcal C conjugate vaccine
MM	Measles-mumps
MMR	Measles-mumps-rubella
MMRV	Measles-mumps-rubella-varicella
NCIRS	National Centre for Immunisation Research and Surveillance of Vaccine Preventable Diseases
NHMRC	National Health and Medical Research Council
NIP	National Immunisation Program
NNDSS	National Notifiable Diseases Surveillance System
NSW	New South Wales
NT	Northern Territory
OPV	Oral poliomyelitis vaccine
7vPCV	7-valent pneumococcal conjugate vaccine
10vPCV	10-valent pneumococcal conjugate vaccine
13vPCV	13-valent pneumococcal conjugate vaccine
23vPPV	23-valent pneumococcal polysaccharide vaccine
PRP-D	Polyribosylribitol phosphate (*Haemophilus influenzae* type b polysaccharide) conjugated to diphtheria toxoid
PRP-OMP	Polyribosylribitol phosphate (*Haemophilus influenzae* type b polysaccharide) conjugated to the outer membrane protein of *Neisseria meningitidis*
PRP-T	Polyribosylribitol phosphate (*Haemophilus influenzae* type b polysaccharide) conjugated to tetanus toxoid
Qld	Queensland
SA	South Australia
SD	Statistical Division
Tas	Tasmania
Vic	Victoria
VPD	Vaccine preventable disease
VV	Varicella vaccine
VZV	Varicella-zoster virus
WA	Western Australia
WHO	World Health Organization

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