Australian Government
Department of Health

Communicable Diseases Intelligence

Volume 40 Number 1 Quarterly report March 2016

Original articles

Short reports

Surveillance summaries

Annual reports

Quarterly reports

Administration

ISSN 1445-4866 Online

Communicable Diseases Intelligence aims to diseminate information on the epidemiology and control of communicable diseases in Australia. *Communicable Diseases Intelligence* invites contributions dealing with any aspect of communicable disease epidemiology, surveillance or prevention and control in Australia. Submissions can be in the form of original articles, short reports, surveillance summaries, reviews or correspondence. Instructions for authors can be found in *Commun Dis Intell* 2015;39(1):E185–E189.

Communicable Diseases Intelligence contributes to the work of the Communicable Diseases Network Australia (http://www.health.gov.au/cdna)

Website

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

Contacts

Communicable Diseases Intelligence is produced every quarter by:
Health Protection Policy Branch
Office of Health Protection
Australian Government Department of Health
GPO Box 9848, (MDP 6)
CANBERRA ACT 2601;
Telephone: +61 2 6289 2717
Facsimile: +61 2 6289 2700
Email: cdi.editor@health.gov.au

This journal is indexed by *Index Medicus* and Medline

Disclaimer

Opinions expressed in *Communicable Diseases Intelligence* are those of the authors and not necessarily those of the Australian Government Department of Health or the Communicable Diseases Network Australia. Data may be subject to revision.

Original article

Clinical diagnosis and chemical confirmation of ciguatera fish poisoning in New South Wales, Australia

Hazel Farrell, Anthony Zammit, D Tim Harwood, Paul McNabb, Craig Shadbolt, Jennifer Manning, John A Turahui, Debra J van den Berg, Lisa Szabo

Abstract

Ciguatera fish poisoning is common in tropical and sub-tropical areas and larger fish (> 10 kg) are more susceptible to toxin accumulation with age. Although the coastal climate of northern New South Wales is considered sub-tropical, prior to 2014 there has only been 1 documented outbreak of ciguatera fish poisoning from fish caught in the region. During February and March 2014, 2 outbreaks of ciguatera fish poisoning involved 4 and 9 individuals, respectively, both following consumption of Spanish mackerel from northern New South Wales coastal waters (Evans Head and Scotts Head). Affected individuals suffered a combination of gastrointestinal and neurological symptoms requiring hospital treatment. At least 1 individual was symptomatic up to 7 months later. Liquid chromatography-tandem mass spectrometry detected the compound Pacific ciguatoxin-1B at levels up to 1.0 μg kg^{-1} in fish tissue from both outbreaks. During April 2015, another outbreak of ciguatera fish poisoning was reported in 4 individuals. The fish implicated in the outbreak was caught further south than the 2014 outbreaks (South West Rocks). Fish tissue was unavailable for analysis; however, symptoms were consistent with ciguatera fish poisoning. To our knowledge, these cases are the southernmost confirmed sources of ciguatera fish poisoning in Australia. Educational outreach to the fishing community, in particular recreational fishers was undertaken after the Evans Head outbreak. This highlighted the outbreak, species of fish involved and the range of symptoms associated with ciguatera fish poisoning. Further assessment of the potential for ciguatoxins to occur in previously unaffected locations need to be considered in terms of food safety. *Commun Dis Intell* 2016;40(1):E1–E6.

Keywords: ciguatera fish poisoning, New South Wales, Australia, Pacific ciguatoxin 1-B, liquid chromatography-tandem mass spectrometry

Introduction

Although it is a significantly under-reported foodborne illness,[1–4] ciguatera fish poisoning (CFP) is the most common non-bacterial seafood related illness worldwide.[5] Ciguatoxin and related chemical compounds (CTXs) are naturally occurring. The toxins are produced by marine micro-algal species within the dinoflagellate genus *Gambierdiscus*.[6] Accumulation of CTXs in the marine food chain typically result from herbivorous fish grazing on the toxin-producing micro algae, which are in turn preyed upon by larger carnivorous fish.[7,8] The toxins are metabolised to more toxic forms as they move up the food chain.[9,10]

As with other micro-algal biotoxins, the visual appearance, taste or odours of fish are not affected by the presence of CTXs.[11] There is no process that will remove CTXs from fish prior to consumption, and cooking or freezing the fish will not destroy the toxins. Fatalities from CFP are rare.[2,12] However, documented symptoms are wide-ranging and a combination of gastrointestinal, neurological and cardiovascular effects can occur.[1,13] Regional differences are apparent in reported CFP outbreaks and specific symptoms will depend on the portion size and what part of the fish was consumed, along with the age and wellbeing of the consumer.[3,10] Gastrointestinal indicators include but are not limited to nausea, vomiting, diarrhoea and stomach cramps while neurological features associated with CFP can involve tingling and numbness in fingers, toes, around lips, tongue, mouth and throat, burning sensation or skin pain on contact with cold water, joint and muscle pains with muscular weakness.[3,10] The most characteristic neurological symptom of CFP is that of temperature dysesthesia, although it is not reported in all cases.[3] The onset of the illness is usually within 24 hours of exposure and symptoms generally last 1–4 days but can persist for weeks or months, and in extreme cases years.[14–16] Symptoms can be exacerbated, or individuals may suffer a relapse, by drinking alcohol.[1,17,18] Such a wide array of symptoms, combined with a lack of awareness of CFP, contributes to the infrequent reporting of illness. There is consider-

able overlap of symptoms with other seafood illness (e.g. diarrhetic shellfish poisoning, paralytic shellfish poisoning caused by algal biotoxins or histamine contamination), while the chronic effects of CFP have similar clinical manifestations as chronic fatigue syndrome, brain tumours or multiple sclerosis.[3,19]

In Australia, the majority of CFP outbreaks have resulted from the consumption of fish caught in Queensland and the Northern Territory waters, with the majority of documented cases involving Spanish mackerel.[2,20,21] Cases reported from other Australian states have been linked to imported fish from warm-water tropical regions in Australia or internationally.[22,23] Prior to the 2014 outbreaks, a CFP outbreak was linked to 2 Spanish mackerel caught from northern New South Wales waters (Brunswick Heads) in 2002.[24] Investigations into suspected CFP outbreaks in New South Wales by the NSW Food Authority in 2005 and 2009 were linked to fish originating from Fiji and Queensland, respectively (NSW Food Authority, unpublished data).

Methods

Case investigations

The NSW Ministry of Health (NSW Health) and NSW Food Authority officers investigated all cases where individuals had consumed the suspected fish meals and suffered illness. The specifics of neurological and gastrointestinal symptoms were documented and fish samples were collected, where available.

These outbreak investigations were conducted under the *NSW Public Health Act 2010* and thus ethics approval was not required.

Toxin analysis

The 2 Spanish mackerel involved in the Evans Head case had a combined total weight of 27.8 kg. Although individual weights were not available, following consultation with the restaurant manager by New South Wales Food Authority officers, it was established that the 2 fish weighed approximately 17 kg and 10 kg. Three fish fillets (~1 kg each) were collected but it was not possible to distinguish which fillets were derived from the larger (17 kg) fish.

A single large flesh fillet (~2 kg) was collected following the reported illnesses at Scotts Head. Fish samples from South West Rocks (2015 outbreak) were unavailable for toxin analysis.

Toxin analysis was carried out at the Cawthron Institute, New Zealand. This analytical laboratory has ISO 17025 accreditation across a wide scope of disciplines including food chemistry, microbiology and natural toxins. Samples were frozen (–20°C) prior to shipping on ice. Samples arrived frozen and were stored at –20°C prior to analysis.

All available Spanish mackerel fish fillet samples were screened for the presence of Pacific ciguatoxin-1B (P-CTX-1B) using a liquid chromatography tandem mass spectrometry method developed in-house at the Cawthron Institute and purified P-CTX-1B standard gifted by Dr Mireille Chinain (Institut Louis Malardé, French Polynesia). A manuscript detailing the performance of the methodology is under preparation. Sample extraction followed the procedure described by Lewis et al.[25] The limit of detection of the analysis was 0.1 μg kg^{-1} P-CTX-1B. To assess toxin recovery, which may be adversely affected by the sample extraction procedure or by sample co-extractives, an aliquot of one of the fish extracts was fortified with a known quantity of P-CTX-1B at a level of 0.2 μg kg^{-1}.

Results

Description of outbreaks

Outbreak 1: Evans Head (~29° 06' S)

On 13 February 2014 the NSW Food Authority received notification from NSW Health of 4 possible CFP cases. The cases were recognised by a local general practitioner and were later reported by a local hospital. Each case had suffered symptoms of myalgia, headaches, tingles and a burning sensation when touching something cold. One individual also experienced vomiting, nausea, diarrhoea bradycardia and hypotension, and was admitted to hospital for 2 days. This individual continued to suffer from neurological symptoms approximately 7 months later.[26]

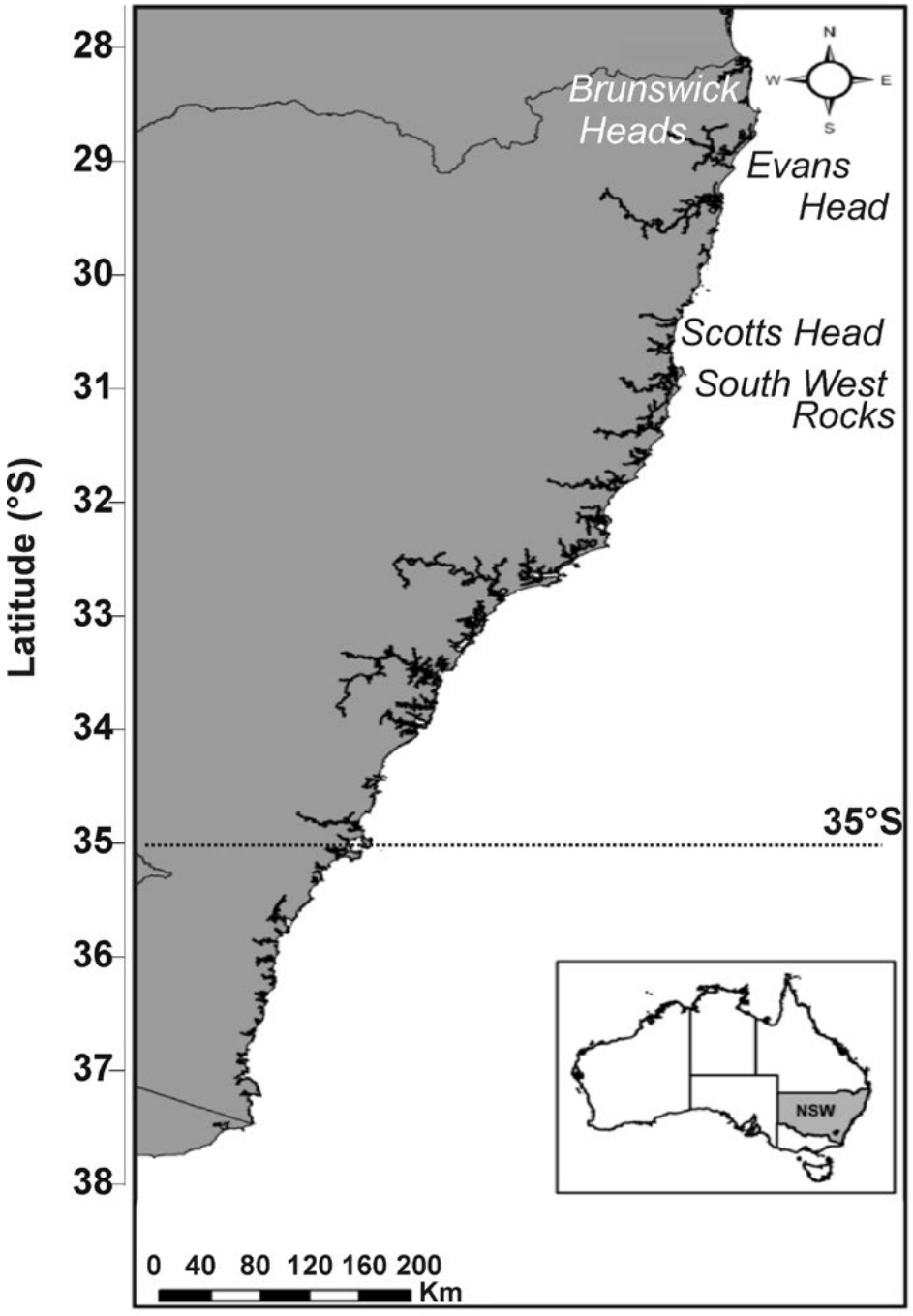
All cases were employees of a local restaurant and had reported eating Spanish mackerel, 3–4 hours prior to suffering symptoms. The restaurant owner was contacted immediately. The owner advised he had purchased 2 Spanish mackerel with individual weights of approximately 10 kg and 17 kg. The fish had been caught off Chaos Reef, near Evans Head (Figure). A chef prepared the fish into fillets for intended sale to bistro customers. However, none of the fish had been sold to customers. Some of the trimmings were used to make fish cakes for the staff, which were eaten for lunch on 13 February 2014 by 7 staff members including the chef. A third smaller (3.8 kg) Spanish mackerel

from the same catch, purchased by a different seafood retail outlet, was removed from sale and destroyed following the outbreak.

Outbreak 2: Scotts Head (~30° 45' S)

On 3 March 2014, NSW Health notified the NSW Food Authority of 9 people who had presented to local hospital emergency departments with suspected CFP. A recreational fisherman had caught a 25.7 kg Spanish mackerel off Scotts Head (Figure) on Sunday 2 March 2014. The fisherman shared the mackerel between himself and 3 family members and gave half of the mackerel to a friend. His friend did not eat the fish, but gave his share to 2 separate neighbours (resulting in 5 people being affected). All 9 people (ranging in age from 13 to 58) ate the fish for dinner on 2 March 2014. The fish fillets were barbequed prior to consumption and it was estimated that each steak was approximately 2 cm thick and weighed between 250–300 g. Adults ate up to 2 serves, while the younger cases ate smaller portions. The onset of symptoms varied from 1–4 hours after consumption, and cases suffered symptoms of abdominal cramps and paraesthesia of the hands and lips. Cardiac problems occurred

Figure: Map of New South Wales coastline showing locations of where the implicated Spanish mackeral were caught



in 1 case. All individuals were advised to avoid alcohol for at least 2–3 months. During follow up interviews, 1 and 2 months post-incident, 1 individual reported a persistent metallic taste for up to 2 months.

Outbreak 3: South West Rocks (~30° 53' S)

On 9 April 2015, the NSW Food Authority was notified of a suspected CFP in a group of 4 adults affected after eating a Spanish mackerel of approximately 10 kg, caught by a spearfisher off South West Rocks (Figure) on 3 April 2015. At least 1 person was hospitalised on 4 April 2015, and one of the doctors diagnosed the case with classic CFP symptoms.

Detection of ciguatoxins

P-CTX-1B was unambiguously detected in the fish sample from Scotts Head (0.4 μg kg^{-1}) and 2 of the 3 samples from Evans Head (0.6–1.0 μg kg^{-1}) (Table). Overspike recovery, where the fish tissue sample was spiked with a known concentration of toxin standard, was determined to be 41%. This was poor and suggested significant matrix suppression from natural compounds present in the fish tissue and likely means P-CTX-1B levels determined in the samples were underestimated. There was also evidence of other CTX analogues present in the P-CTX-1B positive samples. However, because no reference materials are available for the other CTX analogues they were not able to be accurately quantified as part of this analysis.

Table: Results of fish flesh analysis by liquid chromatography-tandem mass spectrometry

Sample	Location	P-CTX-1B	P-CTX-1B (µg kg^{-1})
V1207-A	Scotts Head	Detected	0.4
V1207-B	Evans Head	Detected	0.6
V1207-C	Evans Head	Detected	1.0
V1207-D	Evans Head	Not detected	Not detected

Discussion and outcomes

Following consumption of fish caught in New South Wales coastal waters during 2014, diagnosis of CFP in both cases was based on clinical symptoms and confirmed by chemical analysis and detection of CTXs in the fish tissues. The 2015 case was based on diagnosis of clinical symptoms only, which were consistent with that of CFP. The onset of symptoms was within 4 hours of consumption of the contaminated fish meals. Symptoms were similar in all cases and in 1 individual neurological symptoms were still apparent up to 7 months following the consumption of the contaminated fish.[26] In Pacific regions, P-CTX-1B has been documented in many fish species.[27,28] The US Food and Drug Administration has published suggested guidance levels of 0.01 μg kg^{-1} CTX equivalent for Pacific CTX,[29] which represents an extremely low level. Negative effects on human health from exposure to Pacific CTX have been reported at concentrations between 0.08 and 0.1 μg kg^{-1}.[10] The levels of P-CTX-1B detected in 3 of the 4 samples were up to 2 orders of magnitude higher than this value. In addition, matrix suppression from compounds present in the fish tissue likely resulted in an under-estimation of the toxin level in the samples. The variability in concentrations in samples from Evans Head may be partly explained by the difference in sizes of the 2 fish, which were not distinguishable following filleting. However, some variation within individual fish is also apparent. This may explain the lower attack rate in the Evans Head outbreak (4/7) compared with the Scotts Head outbreak (9/9).

To our knowledge, the locations where the contaminated fish implicated in the two 2014 outbreaks were caught were at that time the southernmost confirmed sources of CFP in Australia. The 2015 case further highlighted the potential for CFP incidence to extend further south along the eastern Australian coast. It is generally accepted that potentially ciguatoxic fish occur between the latitudes 35 °N and 35 °S globally.[29] However, a review by Tester et al.[30] demonstrated the paucity of data south of the Equator regarding CFP incidence for South America, West Africa, the southern Atlantic Ocean and Australia. For south-eastern Australia, an outbreak of CFP in Victoria in 1997 was linked to a consignment of fish from Queensland,[22] while another case in Victoria in 2005 was linked to fish imported from Fiji.[31] Although there have been investigations into CFP associated with imported fish in New South Wales, prior to 2014, only 1 other outbreak (2002, Brunswick Heads) was linked to fish caught in New South Wales waters.[24] At that time, chemical confirmatory testing was unavailable. However, clinical symptoms were consistent with CFP (A. Zammit, personal communication). The cases reported here occurred following the consumption of fish that were caught approximately 70 km (Evans Head), 250 km (Scotts Head) and 265 km (South West Rocks) further south. These cases of CFP occurred within what is considered as the southern CFP boundary (35 °S, Figure). However, the 2014 and 2015 outbreaks are consistent with global accounts of CFP, which are increasing in frequency. Additionally, there are increasing reports of CTXs or potentially CTX-producing microalgae in locations outside of tropic reefs regions that were previously con-

sidered not to be at risk.[4,32,33] It is likely that the Spanish mackerel implicated in each of the 2014 outbreaks in New South Wales originated from Queensland waters, particularly as another case of CFP was documented by Queensland authorities in the Gold Coast region during the same period (February 2014), and 67 cases of CFP were reported in Queensland during 2014.[21] While increasing ocean temperatures may be influencing the distribution and migration patterns of fish species, there is also scope for species of microalgae that produce CTXs to increase their geographic range.[34] For example, *Gambierdiscus* species have recently been reported from as far south as Merimbula in New South Wales (36° 53' S),[33] and in coastal waters of Northland New Zealand (35° 15' S).[35] Although it is difficult to ascertain if populations are extant or introduced, these locations are outside of what was originally regarded as the geographic boundary of the species.

There are still many unknowns surrounding the origins and dynamics of CFP from CTX in seafood. The implementation of regulatory criteria for CTXs has been hindered by the limited availability of reference material, which has also restricted the implementation of a commercial test procedure for routine analysis. In the absence of commercial testing, a precautionary approach is applied in Australia whereby certain species of fish are banned or have size restrictions and fish from specific ciguatera 'hotspots' are prohibited for sale.[36–38] Under-reporting of CFP cases can result from misdiagnosis,[19] a limited capability for chemical detection of CTXs and a lack of public awareness particularly in previously unaffected locations. In the wake of the 2014 New South Wales CFP illness outbreaks, material advising consumers to avoid eating large (> 10 kg) Spanish mackerel and fish head, roe, liver and viscera was distributed in the form of media releases and factsheets via the NSW Food Authority and NSW Health websites.[39,40] Educational outreach to 117,000 recreational fishers was facilitated through the NSW Department of Primary Industries (DPI) following the Evan's Head illnesses.[40] This highlighted the location of the outbreak, fish involved and the range of symptoms associated with ciguatera fish poisoning. Further, to protect consumer safety, the regional Fishermen's Co-op at Ballina New South Wales and Sydney Fish Markets updated their risk management strategy to implement a ban on the sale of all fish over 10 kg in the mackerel species. Additionally, a research project at the University of Technology Sydney, supported by NSW DPI and the Australian Government Fisheries Research and Development Corporation, is underway to investigate CTXs in Spanish mackerel from northern New South Wales and to establish testing capabilities.[41] Advancing our understanding of the risks of CFP, and working towards techniques to reliably and accurately detect if CFP toxins are present in Australian seafood, will reduce the risk to public health and may allow the fishing industry to re-enter the market with fish species that they currently cannot sell.

Acknowledgements

The authors wish to thank NSW Food Authority officers, Ms Dale Collier and Mr Glenn Loche for providing safe transport of fish samples to Sydney and Associate Professor Shauna Murray, University of Technology Sydney for facilitating sample transit to the Cawthron Institute. The expertise of technical staff at the Cawthron Institute is acknowledged. The authors also wish to thank those individuals affected by ciguatera fish poisoning who participated in the case investigations, and medical personal who provided notifications of the outbreaks.

Author details

Hazel Farrell, Shellfish Operations Officer[1]
Anthony Zammit, Manager NSW Shellfish Program[1]
Jennifer Manning, Recall Co-ordinator[1]
Craig Shadbolt, Manager, Food Incidents Response and Complaints[1]
Lisa Szabo, Chief Scientist, NSW Food Authority[1]
D Tim Harwood, Safe New Zealand Seafood Programme co-leader[2]
Paul McNabb, Technical Manager, Research and Method Development[2]
Mr John A Turahui, Communicable Disease Public Health Officer[3]
Ms Debra J van den Berg, Communicable Disease Public Health Officer[3]

1. NSW Food Authority, Newington, New South Wales
2. Cawthron Institute, Nelson, New Zealand
3. North Coast Public Health Unit, Lismore, New South Wales

Corresponding author: Dr Hazel Farrell, Shellfish Operations Officer, PO Box 6682 Silverwater NSW 1811. Telephone: +61 2 9741 4882. Mobile: 0427 290 367. Email: Hazel.Farrell@foodauthority.nsw.gov.au

References

1. Gillespie N, Lewis R, Pearn J, Bourke A, Holmes M, Bourke J, et al. Ciguatera in Australia. Occurrence, clinical features, pathophysiology and management. *Med J Aust* 1986;145(11-12):584-590.
2. Gillespie N, Lewis R, Holmes M. Ciguatera in Australia. South Pacific Commission Twentieth Regional Technical Meeting on Fisheries. Noumea, New Caledonia, 1–5 August 1988.
3. Friedman MA, Fleming LE, Fernandez M, Bienfang P, Schrank K, Dickey R, et al. Ciguatera fish poisoning: treatment, prevention and management. *Marine Drugs* 2008;6(3):456–479.
4. Skinner MP, Brewer TD, Johnstone R, Fleming LE, Lewis RJ. Ciguatera fish poisoning in the Pacific Islands (1998 to 2008). *PLoS Negl Trop Dis* 2011;5(12):e1416.

5. Litaker RW, Vandersea MW, Faust MA, Kibler SR, Nau AW, Holland WC, et al. Global distribution of ciguatera causing dinoflagellates in the genus *Gambierdiscus*. *Toxicon* 2010;56(5):711–730.
6. Adachi R, Fukuyo Y. The thecal structure of a marine toxic dinoflagellate *Gambierdiscus toxicus* gen. et sp. nov. collected in a ciguatera-endemic area. *Bulletin of the Japanese Society of Scientific Fisheries* 1979;45(1):67–71.
7. Randall JE. A review of ciguatera, tropical fish poisoning, with a tentative explanation of its cause. *Bulletin of Marine Science* 1958;8(3):236–267.
8. Cruz-Rivera E, Villareal TA. Macroalgal palatability and the flux of ciguatera toxins through marine food webs. *Harmful Algae* 2006;5(5):497–525.
9. Holmes MJ, Lewis RJ, Poli MA, Gillespie NC. Strain dependent production of ciguatoxin precursors (gambiertoxins) by *Gambierdiscus toxicus* (Dinophyceae) in culture. *Toxicon* 1991;29(6):761–775.
10. Lehane L, Lewis RJ. Ciguatera: recent advances but the risk remains. *Int J Food Microbiol* 2000;61(2):91–125.
11. Food and Agriculture Organization. Ciguatera Fish Poisoning (CFP). In: *Marine Biotoxins FAO Food and Nutrition Paper*. Rome, Italy; 2004. p. 185–278.
12. Hamilton B, Whittle N, Shaw G, Eaglesham G, Moore MR, Lewis RJ. Human fatality associated with Pacific ciguatoxin contaminated fish. *Toxicon* 2010;56(5):668–673.
13. Sims JK. A theoretical discourse on the pharmacology of toxic marine ingestions. *Ann Emerg Med* 1987;16(9):1006–1015.
14. Glaziou P, Martin PM. Study of factors that influence the clinical response to ciguatera fish poisoning. *Toxicon* 1993;31(9):1151–1154.
15. Barton ED, Tanner P, Turchen SG, Tunget CL, Manoguerra A, Clark RF. Ciguatera fish poisoning. A southern California epidemic. *West J Med* 1995;163(1):31.
16. Pearn J. Ciguatera-a potent cause of the chronic fatigue syndrome. *Journal of Immunology and ImmunoPharmacology* 1995;15:63–65.
17. Swift AE, Swift TR. Ciguatera. *Clin Toxicol* 1993;31(1):1–29.
18. Lewis RJ. Ciguatera management. *SPC Live Reef Fish Information Bulletin* 2000;7:11–13.
19. Lindsay JA. Chronic sequelae of foodborne disease. *Emerg Infect Dis* 1997;3(4):443.
20. OzFoodNet. OzFoodNet quarterly reports. 2001–2013. Available from: http://www.ozfoodnet.gov.au/internet/ozfoodnet/publishing.nsf/Content/reports-1.
21. Qld Health. Statewide weekly communicable diseases surveillance report 22 December 2014.
22. Ng S, Gregory J. An outbreak of ciguatera fish poisoning in Victoria. *Commun Dis Intell* 2000;24(11):344–346.
23. NSW Food Authority. Food safety risk assessment of NSW food safety schemes – Updated December 2012.
24. Tobin A, Mapleston A. Exploitation dynamics and biological characteristics of the Queensland east coast Spanish mackerel (*Scomberomorus commerson*) fishery. Townsville: CRC Reef Research Centre; 2004.
25. Lewis RJ, Yang A, Jones A. Rapid extraction combined with LC-tandem mass spectrometry (CREM-LC/MS/MS) for the determination of ciguatoxins in ciguateric fish flesh. *Toxicon* 2009;54(1):62–66.
26. Cheadle B. Ciguatera effects linger. *Rivertown Times, Evans Head NSW* 2014 24 September 2014;Sect. 1.
27. Yogi K, Oshiro N, Inafuku Y, Hirama M, Yasumoto T. Detailed LC-MS/MS analysis of ciguatoxins revealing distinct regional and species characteristics in fish and causative alga from the Pacific. *Anal Chem* 2011;83(23):8886–8891.
28. Yogi K, Sakugawa S, Oshiro N, Ikehara T, Sugiyama K, Yasumoto T. Determination of toxins involved in ciguatera fish poisoning in the Pacific by LC/MS. *J AOAC Int* 2014;97(2):398–402.
29. US Department of Health and Human Services Food and Drug Administration; Center for Food Safety and Applied Nutrition. *Fish and Fishery Products Hazards and Controls Guidance*. 4th edn. April 2011.
30. Tester PA, Feldman RL, Nau AW, Faust MA, Litaker RW. Ciguatera fish poisoning in the Carribean. *Proceedings of the Smithsonian Marine Science Symposium Smithsonian Contributions to the Marine Sciences* 2009:301–311.
31. OzFoodNet Working Group. Burden and causes of foodborne disease in Australia: Annual report of the OzFoodNet network, 2005; *Commun Dis Intell* 2006;30(3):278–300.
32. Llewellyn L, Tester P, Hallegraeff GM. Ciguatera – A neglected tropical disease. An international plan for improved research and management. *Intergovernmental Oceanographic Commission (of UNESCO) Eleventh session of the IOC Intergovernmental Panel on Harmful Algal Blooms UNESCO Headquarters, Paris, 28–30 April 2013* 2013:10.
33. Kohli GS, Murray SA, Neilan BA, Rhodes LL, Harwood DT, Smith KF, et al. High abundance of the potentially maitotoxic dinoflagellate *Gambierdiscus carpenteri* in temperate waters of New South Wales, Australia. *Harmful Algae* 2014;39:134–145.
34. Hallegraeff GM. Ocean climate change, phytoplankton community responses, and harmful algal blooms: a formidable predictive challenge. *Journal of Psychology* 2010;46(2):220–235.
35. Rhodes L, Giménez Papiol G, Smith K, Harwood T. *Gambierdiscus* cf. *yasumotoi* (Dinophyceae) isolated from New Zealand's sub-tropical northern coastal waters. *New Zealand Journal of Marine and Freshwater Research* 2014;48(2):303–310.
36. NSW Food Authority. Food safety program for seafood processing; 2012.
37. Qld Health. Communicable disease control guidance and information. 2015. Available from: https://www.health.qld.gov.au/foodsafety/documents/fs-37-sea-toxin.pdf
38. Sydney Fish Market Pty Ltd. Schedule of ciguatera high-risk areas and species size limits. 2005. Available from: http://sfm7.itworxconsulting.com/Portals/0/Ciguatera_Schedule.pdf
39. NSW Health Seafood Poisoning Factsheet. 2014. Available from: http://www.health.nsw.gov.au/Infectious/factsheets/Pages/seafood_poisoning.aspx
40. Advice to fishers on North, mid north and far north NSW Coast. 2014. Available from: http://www.foodauthority.nsw.gov.au/news/news-04-Mar-14-advice-to-fishers-nsw-coast
41. Martlew M. NSW fishing clubs get behind citizen science fish toxin project. 2015. Available from: http://newsroom.uts.edu.au/news/2015/02/nsw-fishing-clubs-get-behind-citizen-science-fish-toxin-project

Short report

Infectious and congenital syphilis notifications associated with an ongoing outbreak in northern Australia

Amy Bright, Johanna Dups

Introduction

In January 2011, an increase of infectious syphilis notifications among young Aboriginal and Torres Strait Islander people was identified in the North West region of Queensland. Subsequent increases in notifications were reported in the Northern Territory and Western Australia in July 2013 and June 2014 respectively, following sustained periods of low notification rates.[1] In 2012, in response to increased notifications, the Western Australian Department of Health led and funded, with in-kind contributions from the Northern Territory, Queensland and South Australia the development of the *Interim Guidelines for the Public Health Management of Syphilis in Remote Populations in Australia* (interim guidelines).

In April 2015, a Multijurisdictional Syphilis Outbreak Group (MJSO) of the Communicable Diseases Network Australia (CDNA) was formed in response to this on-going outbreak among young Aboriginal and Torres Strait Islander people living in remote areas of northern Australia. A subcommittee of the MJSO was formed in May 2015 to ensure Aboriginal communities were engaged in the outbreak response. The MJSO, with representatives from affected jurisdictions, sexual health physicians, experts in Aboriginal and Torres Strait Islander sexual health and the Australian Government Department of Health, meets monthly with the objective of co-ordinating the public health response for outbreak control and preventing transmission of syphilis from infected women to their babies, through rigorous antenatal testing and care.

All affected jurisdictions have responded to the outbreak in accordance with the *2015 National Guidelines for Syphilis*[2] and interim guidelines. The disease control interventions that have been implemented include: opportunistic and community screening/testing, particularly among young sexually active people aged less than 35 years; immediate treatment of people who are symptomatic (e.g. genital ulceration), have tested positive for syphilis or are sexual contacts of cases; and antenatal screening for syphilis. Public health alerts, health protection and education and campaigns, and active follow up of cases are also being conducted. This report provides a brief description of the epidemiology of the outbreak up to the end of 2015.

Methods

Cases of infectious and congenital syphilis, (as defined by the national CDNA surveillance case definitions[3,4]), were categorised as outbreak cases as defined by the MJSO outbreak case definition.

Any person newly diagnosed with confirmed or probable infectious syphilis according to the CDNA national surveillance case definition for infectious syphilis,

AND

1. is an Aboriginal or Torres Strait Islander person who resides in any of the following outbreak declared regions at or after the dates indicated:

Queensland

- Torres Cape Hospital and Health Service area (from 1 December 2012);
- Cairns and Hinterland Hospital and Health Service area (from 1 August 2013);
- North West Hospital and Health Service area (from 1 January 2011);
- Townsville Hospital and Health Service area (from 1 January 2014);

Northern Territory

- Alice Springs or Barkly district (from 1 July 2013);

Katherine district (from 1 May 2014);

- East Arnhem district (from 1 November 2015);

Western Australia

- Kimberley region (from 1 June 2014).

OR

2. is a sexual contact of a confirmed outbreak case.

Data on cases meeting the outbreak case definition were provided to the MJSO by Queensland, the Northern Territory and Western Australia. An area was declared an outbreak at the discretion of the local jurisdiction. When an area was declared an outbreak region, the same local jurisdiction applied retrospective analysis to determine an outbreak start date when increased numbers of notifications in the new outbreak area could be detected. Cases are reported from the outbreak start date for each outbreak area: these dates are captured in the case definition above.

The national rate of Aboriginal and Torres Strait Islander congenital syphilis cases per 100,000 live births was calculated using the Australian Bureau of Statistics (ABS) Births Australia, 2014 data cubes and past releases of these data. Congenital cases of syphilis were considered associated with the outbreak if the mother was an outbreak case. Live birth refers to the number of births registered within each calendar year and excludes stillbirths or foetal deaths. For a full list of caveats refer to the explanatory notes of the ABS Births Australia releases (catalogue number 3301.0).

Interpretation

It is important to note that changes in notifications over time may not solely reflect changes in disease incidence as changes in notifiable disease case definitions, screening programs,[5] the use of less invasive and more sensitive diagnostic tests[6] and periodic public awareness campaigns[7] may influence the number of notifications to health agencies. Rates for sexually transmissible infections, including infectious syphilis, are particularly susceptible to overall rates of testing.[8] As a priority 'at risk' populations and Aboriginal and Torres Strait Islander people are targeted for increased sexually transmissible infections screening and testing strategies.[9]

Results

The outbreak was first declared in September 2011 in the North West Hospital and Health Service region of Queensland, however, increased notifications associated with the start of the outbreak were first observed in January 2011. Similarly, the Northern Territory declared an outbreak in July 2014 but prior to that, a small linked cluster had been detected in July 2013 with a low number of new cases diagnosed in the ensuing months. In the Kimberley region of Western Australia, increased notifications and the declaration of the outbreak both occurred in the same month, June 2014. Data are reported from the start date indicated in the outbreak case definition to 31 December 2015.

Between 1 January 2011 and 31 December 2015, 790 outbreak associated cases of infectious syphilis (644 confirmed and 146 probable) were reported, in largely remote and rural geographic areas, in northern Australia (Figure 1). Of these, Queensland reported 482 cases (January 2011 to 31 December 2015); the Northern Territory reported 261 cases (July 2013 to 31 December 2015), and; Western Australia reported 47 cases of infectious syphilis (June 2014 to 31 December 2015) (Table).

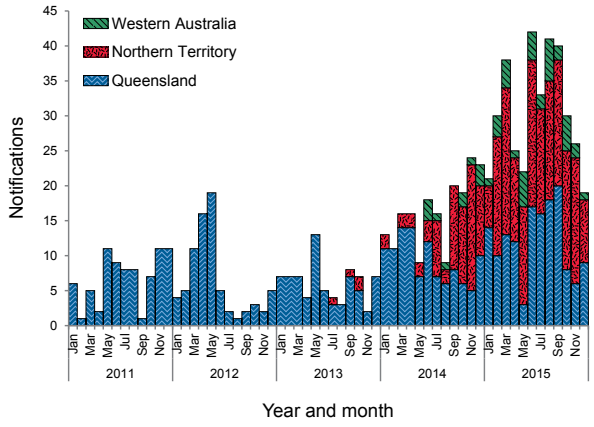
There was a similar distribution between males and females (45% male; 55% female) and cases were predominately reported in the 15–19 and 20–29 years age groups, representing 37% and 38% respectively.

Table: Outbreak associated infectious syphilis cases, 1 January 2011 to 31 December 2015

Affected regions	Confirmed cases	Probable cases	Total cases
Queensland			
Torres and Cape HHS	427	55	482
Cairns and Hinterland HHS			
North West HHS			
Townsville HHS			
Northern Territory			
Alice Springs	181	80	261
Barkly			
Katherine			
East Arnhem			
Western Australia			
Kimberley health region	36	11	47
Total outbreak cases	644	146	790

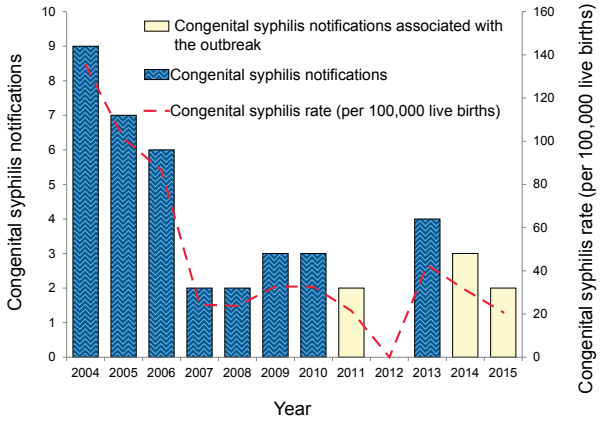
HHS – Hospital and Health Service

Figure 1: Infectious syphilis notifications associated with the outbreak in affected regions* of Queensland, the Northern Territory and Western Australia, 1 January 2011 to 31 December 2015



* Includes Torres and Cape, Cairns and Hinterland, North West and Townsville Hospital and Health service regions in Queensland; Alice Springs, Barkly, Katherine and East Arnhem districts in the Northern Territory, and; the Kimberley health region in Western Australia.

Figure 2: Number of congenital syphilis notifications and notification rate per 100,000 live births, in Aboriginal and Torres Strait Islanders people, Western Australia, the Northern Territory and Queensland, 2004 to 2015



Since 2011, a total of 7 outbreak associated congenital syphilis cases were reported in the Aboriginal and Torres Strait Islander population: 3 cases in the Northern Territory (1 confirmed and 2 probable) and 4 cases in Queensland (3 confirmed and 1 probable) (Figure 2). Of the Queensland cases, 2 were stillborn and 1 died in the neonatal period. All 3 cases in the Northern Territory returned negative serology results at or before the 18-month follow-up. Over the course of the outbreak (January 2011 to December 2015), the rate of congenital syphilis (including both confirmed and probable cases) in the Aboriginal and Torres Strait Islander populations for all of Queensland, the Northern Territory and Western Australia averaged 23.0 cases per 100,000 live births, which was lower than that recorded in the 5 years prior to the outbreak (2006 to 2010) at 38.2 cases per 100,000 live births (Figure 2).

Acknowledgement

Members of the Multijurisdictional Syphilis Outbreak Group include (in alphabetical order): Nathan Ryder (Chair, University of Newcastle), Sonya Bennett (Communicable Diseases Branch, Queensland Health), Amy Bright (Office of Health Protection, Australian Government Department of Health), Katy Crawford (Kimberley Population Health Unit), Johanna Dups (Communicable Disease Control Directorate, Western Australian Department of Health), Linda Garton (Centre for Disease Control, Northern Territory Department of Health), Carolien Giele (Communicable Disease Control Directorate, Western Australian Department of Health), Manoji Gunathilake (Centre for Disease Control, Northern Territory Department of Health), Alex Hope (Aboriginal Medical Services Alliance Northern Territory), Rae-Lin Huang (Nganampa Health Council), John Kaldor (The Kirby Institute, University of New South Wales), Vicki Krause (Centre for Disease Control, Northern Territory Department of Health), Donna Mak (Communicable Disease Control Directorate, Western Australian Department of Health), Arun Menon (Townsville Sexual Health Services, Queensland Health), Rhonda Owen (Office of Health Protection, Australian Government Department of Health), Annie Preston-Thomas (Tropical Public Health Services, Cairns), Amanda Sibosado (Kimberley Aboriginal Medical Services Council Inc), Jiunn-Yih Su (Centre for Disease Control, Northern Territory Department of Health, Matthew Thalanany (Centre for Disease Control, Northern Territory Department of Health), Ingrid Tribe (Communicable Disease Control Branch, Department of Human Services South Australia), Russel Waddell (Communicable Disease Control Branch, Department of Human Services South Australia) and James Ward (South Australian Health and Medical Research Institute).

Author details

Amy Bright, Australian Government Department of Health, Canberra, Australian Capital Territory

Johanna Dups, Western Australian Department of Health, on behalf of the Multijurisdictional Syphilis Outbreak Group, Western Australia

Corresponding author

Ms Amy Bright, Office of Health Protection, Australian Government Department of Health, MDP 14, GPO Box 9848, CANBERRA ACT 2601. Telephone: +61 2 6289 5844. Email: Amy.Bright@health.gov.au

References

1. Ward J, Guy R, Akre S, Middleton M, Giele C, Su J, et al. Epidemiology of syphilis in Australia: moving toward elimination of infectious syphilis from remote Aboriginal and Torres Strait Islander communities? *Med J Aust* 2011;194(10):525–529.
2. Communicable Diseases Network Australia. National guidelines for public health units: syphilis. 2015. Accessed on 21 December 2015. Available from: http://www.health.gov.au/internet/main/publishing.nsf/Content/cdna-song-syphilis.htm
3. Communicable Diseases Network Australia. Congenital syphilis case definition. 2015. Accessed on 25 February 2016. Available from: http://www.health.gov.au/internet/main/publishing.nsf/Content/cda-surveil-nndss-casedefs-cd_consyph.htm
4. Communicable Diseases Network Australia. Infectious syphilis case definition. 2015. Accessed on 25 February 2016. Available from: http://www.health.gov.au/internet/main/publishing.nsf/Content/cda-surveil-nndss-casedefs-cd_syphl2.htm
5. Hocking J, Fairley C, Counahan M, Crofts N. The pattern of notification and testing for genital *Chlamydia trachomatis* infection in Victoria, 1998–2000: an ecological analysis. *Aust N Z J Public Health* 2003;27(4):405–408.
6. Burckhardt F, Warner P, Young H. What is the impact of change in diagnostic test method on surveillance data trends in *Chlamydia trachomatis* infection? *Sex Transm Infect* 2006;82(1):24–30.
7. Chen M, Karvelas M, Sundararajan V, Hocking J, Fairley C. Evidence for the effectiveness of a chlamydia awareness campaign: increased population rates of chlamydia testing and detection. *Int J STD AIDS* 2007;18(4):239–243.
8. Ali H, Guy R, Fairley C, Wand H, Chen M, Dickson B, et al. Understanding trends in genital *Chlamydia trachomatis* can benefit from enhanced surveillance: findings from Australia. *Sex Transm Infect* 2012;88(7):552–557.
9. Australian Government Department of Health. Fourth National Aboriginal and Torres Strait Islander Blood-Borne Viruses and Sexually Transmissible Infections Strategy 2014–2017. 2014. [Online]. Accessed on 25 February 2016. Available from: http://www.health.gov.au/internet/main/publishing.nsf/Content/ohp-bbvs-atsi

Surveillance summaries

Surveillance systems reported in *Communicable Diseases Intelligence*, 2016

This article describes the surveillance schemes that are routinely reported on in *Communicable Diseases Intelligence* (CDI).

Communicable disease surveillance in Australia operates at the national, state and local levels. Primary responsibility for public health action lies with the state and territory health departments. The role of communicable disease surveillance at a national level includes:

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

State and territory health departments collect notifications of communicable diseases under their public health legislation. In September 2007, the *National Health Security Act 2007* (*National Health Security Act, No 174*) received royal assent. This Act provides the legislative basis for and authorises the exchange of health information, including personal information, between jurisdictions and the Commonwealth. The Act provides for the establishment of the National Notifiable Diseases List, which specifies the diseases about which personal information can be provided. The National Health Security Agreement, signed by Health Ministers in April 2008, establishes the operational arrangements to formalise and enhance existing surveillance and reporting systems, an important objective of the Act. States and territories voluntarily forward de-identified data on a nationally agreed group of communicable diseases to the Department of Health (Health) for the purposes of national communicable disease surveillance.

Surveillance has been defined by the World Health Organization as the 'continuing scrutiny of all aspects of the occurrence and spread of disease that are pertinent to effective control.' It is characterised by 'methods distinguished by their practicability, uniformity, and frequently by their rapidity, rather than complete accuracy.[1] Although some surveillance schemes aim for complete case ascertainment, others include only a proportion of all cases of the conditions under surveillance, and these samples are subject to systematic and other biases. Results generated from surveillance schemes must be interpreted with caution, particularly when comparing results between schemes, between different geographical areas or jurisdictions and over time. Surveillance data may also differ from data on communicable diseases gathered in other settings.

The major features of the surveillance schemes for which CDI publishes regular reports are described below.

Other surveillance schemes for which CDI publishes annual reports include tuberculosis notifications (*Commun Dis Intell* 2015;39(2):E217–E235), the Australian Mycobacterium Reference Laboratory Network (*Commun Dis Intell* 2014;38(4):E356–E368), and the Australian Rotavirus Surveillance Program (*Commun Dis Intell* 2015;39(3):E337–E346).

Arbovirus and malaria surveillance

The National Arbovirus and Malaria Advisory Committee (NAMAC) collates data and reports on the epidemiology of mosquito-borne diseases of public health importance in Australia by financial year (which represents the cycle of mosquito-borne disease activity in most parts of Australia). The reports include data from the National Notifiable Diseases Surveillance System (NNDSS) on notified cases of disease caused by the alphaviruses: Barmah Forest virus, chikungunya virus and Ross River virus; the flaviviruses: dengue virus, Murray Valley encephalitis virus (MVEV), the Kunjin strain of West Nile virus, Japanese encephalitis virus and yellow fever virus; and the protzoan infection, malaria. Both locally acquired and overseas acquired cases are described. Vector, climate and sentinel animal surveillance measures for arboviruses (in particular for MVEV) conducted

by states and territories, and also at the border are described. Sentinel chicken, mosquito surveillance, viral detection in mosquitoes and climate modelling are used to provide early warning of arboviral disease activity in Australia. Sentinel chicken programs for the detection of flavivirus activity are conducted in most states at risk of arboviral transmission. Other surveillance activities to detect the presence of arboviruses in mosquitoes or mosquito saliva or for surveying mosquito abundance included honey-baited trap surveillance, surveys of household containers that may provide suitable habitat for the dengue vector, *Aedes aegypti,* and carbon dioxide baited traps.

NAMAC provides expert technical advice on arboviruses and malaria to the Australian Health Protection Principal Committee through the Communicable Diseases Network Australia (CDNA). Members of the Committee have expertise in virus and disease surveillance, epidemiology, virology, vector ecology, vector control and quarantine, and represent agencies with a substantial interest in this area. NAMAC makes recommendations about surveillance and reporting systems, strategic approaches for disease and vector management and control, laboratory support, development of national guidelines and response plans and research priorities. NAMAC assists in the detection, management and control of real or potential outbreaks of arboviruses or malaria and provides advice on the risk of these diseases or exotic vectors being imported from overseas. NAMAC members participate in outbreak management teams as required.

Further details are provided in the NAMAC annual report (*Commun Dis Intell* 2016;40(1):E17–E47).

Australian Childhood Immunisation Register

Accurate information on the immunisation status of children is needed at the community level for program management and targeted immunisation efforts. A population-based immunisation register can fulfil this need. The Australian Childhood Immunisation Register (ACIR) commenced operation on 1 January 1996 and is now an important component of the Immunise Australia Program. It is administered and operated by Medicare Australia. The register was established by transferring data on all children under the age of 7 years enrolled with Medicare to the ACIR. This constitutes a nearly complete population register, as approximately 99% of children are registered with Medicare by 12 months of age. Children who are not enrolled in Medicare are added to the register when a recognised immunisation provider supplies details of an eligible immunisation. Immunisations are mostly notified to Medicare either by electronic means, the Internet or by paper ACIR notification forms. Immunisations recorded on the Register must have been given in accordance with the guidelines for immunisation determined by the National Health and Medical Research Council (NHMRC). From 1 January 2016, ACIR will record vaccinations for children up to 20 years of age. This change will support upcoming changes to immunisation requirements for child care payments and Family Tax Benefit Part A supplement.

From the data finally entered onto the ACIR, Medicare Australia provides regular rolling annualised quarterly coverage reports at the national and state level. Coverage for these reports is calculated using the cohort method previously described (*Commun Dis Intell* 1998;22:36–37). With this method, a cohort of children is defined by date of birth in 3-month groups. This birth cohort has the immunisation status of its members assessed at the 3 key milestones of 12 months, 24 months and 60 months of age. Analysis of coverage is undertaken 3 months after the due date for completion of each milestone, so that time is available for processing notifications and the impact on coverage estimates of delayed notification to the ACIR is minimised. Only children enrolled with Medicare are included, in order to minimise inaccuracies in coverage estimates due to duplicate records.

The ACIR coverage reports for the 3 milestones are published in CDI each quarter. Coverage estimates are provided for each state and territory and Australia as a whole and for each individual vaccine assessed at each milestone. A commentary on ACIR immunisation coverage estimates is included with the tables in each issue and a graph is used to provide trends in immunisation coverage.

An immunisation coverage report is also published in CDI on an annual basis and provides more detailed data on immunisation coverage for all recommended vaccines by age group that are funded by the Immunise Australia Program, timeliness of immunisation, small area coverage estimates and data on vaccination objection to immunisation. While vaccination is not compulsory in Australia, from 1 January 2016, objections on the basis of personal, philosophical or religious beliefs will no longer be a valid exemption from vaccination in order to receive some government family assistance payments.

Australian Gonococcal Surveillance Programme

The Australian Gonococcal Surveillance Programme (AGSP) is a continuing program to

monitor antimicrobial resistance in *Neisseria gonorrhoeae* and includes the reference laboratories in all states and territories. These laboratories report data on sensitivity to an agreed core group of antimicrobial agents on a quarterly basis and provide an expanded analysis as an annual report in CDI *(Commun Dis Intell* 2015;39(3):E347–E354). The antibiotics that are currently routinely surveyed are the penicillins, ceftriaxone, ciprofloxacin and spectinomycin, all of which are administered as single dose regimens. A major purpose of the AGSP is to help define standard protocols for antibiotic treatment of gonococcal infection. When *in vitro* resistance to a recommended agent is demonstrated in 5% or more of isolates, it is usual to reconsider the inclusion of that agent in current treatment schedules. Additional data are also provided on other antibiotics from time to time. At present, all laboratories also test isolates for the presence of high level resistance to the tetracyclines and intermittent surveys of azithromycin resistance are conducted. Comparability of data is achieved by means of a standardised system of minimal inhibitory concentration (MIC) testing and a program-specific quality assurance process.

Australian Meningococcal Surveillance Programme

The reference laboratories of the Australian Meningococcal Surveillance Programme report data on laboratory-confirmed cases confirmed either by culture or by non-culture techniques. Culture-positive cases where *Neisseria meningitidis* is grown from a normally sterile site or skin, and non-culture based diagnoses, derived from the results of nucleic acid amplification assays and serological techniques are defined as invasive meningococcal disease (IMD) according to the Public Health Laboratory Network definitions.

Data are reported annually and quarterly in CDI. Data in the quarterly reports are restricted to a description of the number of cases per jurisdiction, and serogroup where known. A full analysis of laboratory-confirmed cases of IMD, including phenotyping and antibiotic susceptibility data are published annually (*Commun Dis Intell* 2015;39(3):E347–E354).

Australian National Creutzfeldt-Jakob Disease Registry

Surveillance for Creutzfeldt-Jakob disease (CJD) in Australia is conducted through the Australian National Creutzfeldt-Jakob Disease Registry (ANCJDR). CJD is listed as a notifiable disease in all Australian states and territories. The ANCJDR is under contract to the Commonwealth to identify and investigate all suspect cases of transmissible spongiform encephalopathy in Australia. An annual update is published in CDI *(Commun Dis Intell* 2014;38(4):E348–E355).

Australian Paediatric Surveillance Unit

The Australian Paediatric Surveillance Unit (APSU) is an active surveillance mechanism for prospective, national identification and study of children aged <15 years, newly diagnosed with uncommon conditions including rare infectious and vaccine preventable diseases, genetic disorders, child mental health problems, rare injuries and other rare chronic childhood conditions. Up to 16 different conditions are studied simultaneously. The APSU relies on monthly reporting by ~1,400 paediatricians and other child health clinicians and over 85% of clinicians respond via e-mail. Clinicians reporting cases are asked to provide details about demographics, diagnosis, treatments and short-term outcomes. All negative and positive reports are logged into a database and the report card return rate has been maintained at over 90% for the last 20 years. The APSU, together with the National Centre for Immunisation Research and Surveillance jointly provide coordination for the Paediatric Active Enhanced Disease Surveillance (PAEDS). PAEDS is currently operational in 5 paediatric referral centres in 5 states and collects detailed information on relevant admitted cases (www.paeds.edu.au).

Communicable diseases currently under surveillance include: acute flaccid paralysis (to identify potential cases of poliovirus infection); congenital cytomegalovirus infection; congenital rubella; perinatal exposure to HIV, and HIV infection; neonatal herpes simplex virus infection; neonatal varicella, congenital varicella, severe complications of varicella and juvenile onset recurrent respiratory papillomatosis. After demonstrating feasibility in 2007, the APSU has conducted seasonal surveillance for severe complications of influenza each year. In 2009 APSU contributed to the national surveillance effort during the influenza A(H1N1) pdm09 pandemic.

The activities of the APSU are funded in part by the Australian Government Department of Health, and the NHMRC Practitioner Fellowship No: 1021480 (E Elliott). The Faculty of Medicine, The University of Sydney, and the Royal Australasian College of Physicians, Division of Paediatrics and Child Health, and the Kids Research Institute, Sydney Children's Hospitals Network provide in-kind support. APSU publishes an annual report *(Commun Dis Intell* 2014;38(4):E343–E347). For further information please contact the APSU Director, Professor Elizabeth Elliott on telephone:

+61 2 9845 3005, facsimile +61 2 9845 3082 or email: apsu@chw.edu.au; Internet: http://www.apsu.org.au

Australian Sentinel Practice Research Network

The Discipline of General Practice at the University of Adelaide operates the Australian Sentinel Practice Research Network (ASPREN). ASPREN is a national network of general practitioners who report presentations of defined medical conditions each week. The main aims of ASPREN are to provide an indicator of disease burden and distribution in the community and to be an early indicator of pandemic influenza.

The list of conditions is reviewed annually by the ASPREN management committee and an annual report is published. In 2016, 4 conditions are being monitored; all of which are related to communicable diseases. These are influenza like illness (ILI), gastroenteritis, chickenpox and shingles.

Laboratory testing of ILI cases was implemented in 2010, allowing for viral testing of 25% of ILI patients for a range of respiratory viruses including influenza A, influenza B and A(H1N1)pdm09.

There are currently 210 general practitioners registered with the network from all jurisdictions. Fifty-eight per cent of these are in metropolitan areas, 32% in rural and 10% in remote areas of Australia. Approximately 15,000 consultations are recorded by these general practitioners each week.

Data for communicable diseases are published in CDI each quarter. Data are presented in graphical format with the rate reported as the number of conditions per 1,000 consultations per week. The conditions are defined as:

Influenza-like illness – record once only per patient

Must have the following: fever, cough and fatigue.

Gastroenteritis – record once only per patient

Three or more loose stools, and/or 2 vomits in a 24 hour period excluding cases who have a known cause, for example bowel disease, alcohol, pregnancy.

Chickenpox – record once only per patient

An acute, generalised viral disease with a sudden onset of slight fever, mild constitutional symptoms and a skin eruption which is maculopapular for a few hours, vesicular for three to 4 days and leaves a granular scab.

Shingles – record once only per patient

Recurrence, recrudescence or re-activation of chickenpox infection. Vesicles with any erythematous base restricted to skin areas supplied by sensory nerves of a single or associated group of dorsal root ganglia. Lesions may appear in crops in irregular fashion along nerve pathways, are usually unilateral, deeper seated and more closely aggregated than those of chickenpox.

Note: Those conditions which show 'record once only per patient' are to have each occurrence of the condition recorded on 1 occasion no matter how many patient contacts are made for this episode of illness. If the condition recurs at a later date it can be recorded/counted again.

HIV surveillance

National surveillance for newly diagnosed HIV infection is coordinated by the Kirby Institute for Infection and Immunity in Society, in collaboration with state and territory health authorities, the Australian Government Department of Health, the Australian Institute of Health and Welfare and other collaborating networks in surveillance for HIV, viral hepatitis and sexually transmissible infections.

Cases of HIV infection are notified to the National HIV Registry on the first occasion of diagnosis in Australia, either by the diagnosing laboratory (Australian Capital Territory and Tasmania), by doctor notification (Western Australia) or by a combination of laboratory and doctor sources (New South Wales, Northern Territory, Queensland, South Australia and Victoria). Diagnoses of HIV infection are notified with the person's date of birth and name code, to minimise duplicate notifications while maintaining confidentiality.

Currently, 2 tables presenting the number of new diagnoses of HIV infection in Australia in the most recent quarter and cumulatively are published in CDI. The tabulations are based on data available 3 months after the end of the reporting period, to allow for reporting delay and to incorporate newly available information.

An annual surveillance report, *HIV, Viral Hepatitis and Sexually Transmissible Infections in Australia Annual Surveillance Report* has been published by the Kirby Institute since 1997. The Annual Surveillance Report, available through http://www.kirby.unsw.edu.au, provides a comprehen-

sive analysis and interpretation of surveillance data on HIV, viral hepatitis and sexually transmissible infections in Australia. The report *Bloodborne viral and sexually transmitted infections in Aboriginal and Torres Strait Islander people: Surveillance and Evaluation Report* has been published from 2007, as an accompanying document to the annual surveillance report. The Surveillance and Evaluation Report provides detailed analysis and interpretation of the occurrence of these infections in Aboriginal and Torres Strait Islander communities in Australia.

Invasive Pneumococcal Disease Surveillance Program

The Commonwealth has developed the Invasive Pneumococcal Disease (IPD) Surveillance Program as part of the NNDSS. The objectives and outcomes of the IPD Surveillance Program are to:

- record every case of IPD occurring in Australia;
- collect detailed information on each case of IPD as set out in the NNDSS Invasive Pneumococcal Infection Enhanced Surveillance Form;
- collate nationally this information in the NNDSS dataset for enhanced IPD surveillance;
- measure the impact of conjugate pneumococcal vaccination on the rates and types of pneumococcal disease, the prevalence of circulating pneumococcal serotypes and levels of antibiotic resistance; and
- assess whether cases or deaths in children under 5 years and adults over 65 years are due to IPD vaccine failure or antibiotic resistance.

The Commonwealth funds 4 laboratories to perform the laboratory component of enhanced surveillance of IPD, which consists of the serotyping all isolates of *Streptococcus pneumoniae* from cases of IPD.

IPD data are reported annually (*Commun Dis Intell* 2015;39(2):E265–E279) and quarterly in CDI. These reports include analysis notification and laboratory data collected through the NNDSS.

IPD surveillance is overseen by the Enhanced Invasive Pneumococcal Disease Surveillance Working Group (EIPDSWG), a subcommittee of the CDNA. The EIPDSWG assists in developing and implementing a nationally standardised approach to the enhanced surveillance of IPD in Australia.

National Influenza Surveillance Scheme

Australian influenza activity and severity in the community are monitored using a number of indicators and surveillance schemes:

- Notifications of laboratory-confirmed influenza are reported from all Australian states and territories and included in the NNDSS.
- Community level ILI is monitored through two sentinel systems, Flutracking, a weekly online survey integrating syndromic information with participant influenza immunity status; and data from the National Health Call Centre Network.
- Reports on general practice ILI consultations are provided through ASPREN and the Victorian Sentinel General Practice Scheme. Additionally, data on ILI presentations to hospital emergency departments are collected from sentinel hospital sites in Western Australia and New South Wales.
- Hospitalised cases of laboratory-confirmed influenza are reported through the Influenza Complications Alert Network (FluCAN); and severe complications in children are monitored by the APSU.
- Information on influenza subtypes and positivity are provided by sentinel laboratories, including the national influenza centre laboratories and some state public health laboratories. Additional virology and antiviral resistance data are also provided from the World Health Organization Collaborating Centre for Reference and Research on Influenza in Melbourne.

During the influenza season, data from each of these surveillance systems are compiled and published fortnightly in the Australian influenza surveillance report, which is generally available from May to October on the department's web site. These reports include the above data as well as additional mortality and international surveillance data.

Annual reports on the National Influenza Surveillance Scheme are published in the CDI each year (*Commun Dis Intell* 2010;34(1):8–22).

National Notifiable Diseases Surveillance System

National compilations of notifiable diseases have been published intermittently in a number of publications since 1917.[2] The NNDSS was established in 1990 under the auspices of CDNA.

More than 60 communicable diseases agreed upon nationally are reported to NNDSS, although not

all are notifiable in each jurisdiction. Data are sent electronically from states and territories daily (business days only in some jurisdictions). The system is complemented by other surveillance systems, which provide information on various diseases, including three that are not reported to NNDSS (HIV, and the classical and variant forms of CJD).

The NNDSS core dataset includes data fields for a unique record reference number; notifying state or territory, disease code, age, sex, Indigenous status, postcode of residence, date of onset of the disease, death, date of report to the state or territory health department and outbreak reference (to identify cases linked to an outbreak). Where relevant, information on the species, serogroups/subtypes and phage types of organisms isolated, and on the vaccination status of the case is collected. Data quality is monitored by Health and the National Surveillance Committee and there is a continual process of improving the national consistency of communicable disease surveillance.

While not included in the core national dataset, enhanced surveillance information for some diseases (hepatitis B [newly acquired], hepatitis C [newly acquired], invasive pneumococcal disease, donovanosis, gonococcal infection, syphilis < 2 years duration and tuberculosis) is obtained from states and territories.

Aggregated data are presented on the department's internet site under *Communicable Diseases Surveillance* and updated daily (http://www.health.gov.au/nndssdata). A summary report and data table are also published on the Internet each fortnight (http://www.health.gov.au/cdnareport).

Data are published in CDI each quarter and in an annual report. The reports include numbers of notifications for each disease by state and territory, and totals for Australia for the current period, the year to date, and for the corresponding period of the previous year. The national total for each disease is compared with the average number of notifications over the previous 5 years in the same period. A commentary on the notification data is included with the tables in each issue of CDI and graphs are used to illustrate important aspects of the data.

OzFoodNet: enhanced foodborne disease surveillance

The Australian Government Department of Health established the OzFoodNet network in 2000 with epidemiologists in every Australian State and Territory to collaborate nationally in the investigation of foodborne disease. OzFoodNet conducts studies on the burden of illness and coordinates national investigations into outbreaks of foodborne disease.

OzFoodNet reports quarterly on investigations of outbreaks and clusters of gastroenteritis potentially related to food. Annual reports have been produced and published in CDI since 2001 with the most recent being the 2010 annual report (*Commun Dis Intell* 2015;39(3):E236–E264). Data are reported from all Australian jurisdictions.

References

1. Last JM. A dictionary of epidemiology. New York: Oxford University Press, 1988.
2. Hall R. Notifiable diseases surveillance, 1917 to 1991. *Commun Dis Intell* 1993;17(11):226–236. Available from: http://www.health.gov.au/internet/main/publishing.nsf/Content/cda-pubs-annlrpt-oz_dis19_91.htm Accessed March 2015.

Annual report

Arboviral diseases and malaria in Australia, 2012–13: Annual report of the National Arbovirus and Malaria Advisory Committee

Katrina E Knope, Nina Kurucz, Stephen L Doggett, Mike Muller, Cheryl A Johansen, Rebecca Feldman, Michaela Hobby, Sonya Bennett, Angus Sly, Stacey Lynch, Bart J Currie, Jay Nicholson, and the National Arbovirus and Malaria Advisory Committee

Abstract

This report describes the epidemiology of mosquito-borne diseases of public health importance in Australia during the 2012–13 season (1 July 2012 to 30 June 2013) and includes data from human notifications, sentinel chicken, vector and virus surveillance programs. The National Notifiable Diseases Surveillance System received notifications for 9,726 cases of disease transmitted by mosquitoes during the 2012–13 season. The Australasian alphaviruses Barmah Forest virus and Ross River virus accounted for 7,776 (80%) of total notifications. However, over-diagnosis and possible false positive diagnostic test results for these 2 infections mean that the true burden of infection is likely overestimated, and as a consequence, the case definitions were revised, effective from 1 January 2016. There were 96 notifications of imported chikungunya virus infection. There were 212 notifications of dengue virus infection acquired in Australia and 1,202 cases acquired overseas, with an additional 16 cases for which the place of acquisition was unknown. Imported cases of dengue were most frequently acquired in Indonesia. No locally-acquired malaria was notified during the 2012–13 season, though there were 415 notifications of overseas-acquired malaria. There were no cases of Murray Valley encephalitis virus infection in 2012–13. In 2012–13, arbovirus and mosquito surveillance programs were conducted in most jurisdictions with a risk of vectorborne disease transmission. Surveillance for exotic mosquitoes at the border continues to be a vital part of preventing the spread of mosquito-borne diseases such as dengue to new areas of Australia, and in 2012–13, there were 7 detections of exotic mosquitoes at the border. *Commun Dis Intell* 2016;40(1):E17–E47.

Keywords: arbovirus; Barmah Forest virus, chikungunya, dengue, disease surveillance, epidemiology, flavivirus, Japanese encephalitis, Kunjin virus, malaria, mosquito-borne disease, Murray Valley encephalitis virus, Ross River virus, yellow fever, West Nile virus

Introduction

This report describes the epidemiology of mosquito-borne diseases of public health importance in Australia during the period 1 July 2012 to 30 June 2013. It includes a summary of notified cases of disease caused by the alphaviruses Barmah Forest virus (BFV), chikungunya virus (CHIKV) and Ross River virus (RRV); the flaviviruses dengue virus (DENV), Murray Valley encephalitis virus (MVEV), West Nile virus (WNV) and the Kunjin lineage of West Nile virus (KUNV), Japanese encephalitis virus (JEV) and yellow fever virus (YFV); and malaria. Both locally acquired and overseas acquired cases are described. Vector, climate and sentinel chicken surveillance measures for arboviruses conducted by states and territories, and also at the international first ports of entry are described.

The National Arbovirus and Malaria Advisory Committee (NAMAC) provides expert technical advice on arboviruses and malaria to the Australian Health Protection Principal Committee (AHPPC) through the Communicable Diseases Network Australia (CDNA). Members of NAMAC have expertise in virus and disease surveillance, epidemiology, virology, vector ecology, vector control and quarantine, and represent agencies with a substantial interest in this area. NAMAC makes recommendations about surveillance and reporting systems, strategic approaches for disease and vector management and control, and laboratory support outlines research priorities. NAMAC develops and provides input to national guidelines and response plans. NAMAC assists in the detection, management and control of real or potential outbreaks of arboviruses or malaria and provides advice on the risk of these diseases or exotic vectors being imported from overseas and the potential impacts on Australia. NAMAC members participate in outbreak management teams as required.

Methods

Human cases of arbovirus infection and malaria are monitored using the National Notifiable

Diseases Surveillance System (NNDSS). All Australian states and territories require doctors and/or pathology laboratories to notify cases of infectious diseases that are important to public health. The *National Health Security Act 2007* (NHS Act 2007) provides the legislative basis for the national notification of communicable diseases and authorises the exchange of health information between the Commonwealth and the states and territories. The NHS Act 2007 provides for the establishment of the National Notifiable Diseases List, which specifies the diseases about which personal information can be exchanged between the states and territories and the Commonwealth. State and territory health departments transfer these notifications regularly to the NNDSS. The primary responsibility for public health action resulting from a notification resides with state and territory health departments.

This report presents data from a snap-shot of NNDSS taken during July 2015 and analysed by date of diagnosis. This derived field is the onset date, or where the date of onset was not known, for vectorborne diseases, it is the earliest of the specimen collection date, the notification date, or the notification received date. Since the data are from a snap-shot, numbers in this report may vary slightly from those reported elsewhere due to changes in diagnostic validation or classification. Data were verified with state and territory public health surveillance managers. Detailed notes on the interpretation of NNDSS are available in the 2013 NNDSS annual report.[1] Case definitions for the diseases included in this report are available on the Australian Government Department of Health web site (http://www.health.gov.au/casedefinitions).

CHIKV infection became nationally notifiable in 2015, though a national case definition was implemented from 2010. Prior to this, CHIKV infections were notified under the disease category flavivirus (unspecified), and all notifications have now been included under CHIKV.

Data were analysed by financial year to reflect the seasonal cycle of arboviral activity in most areas of Australia. Crude notification rates or counts for the 2012–13 season were compared with those recorded over the previous 5 years. Notification rates were not calculated for diseases that are primarily acquired overseas because resident populations are not an appropriate denominator. Rates are not provided for rare diseases ($n < 20$) because these rates typically have large standard errors and therefore cannot be meaningfully compared across time or geographical location.

Notification rates were calculated using the Australian Bureau of Statistics estimated resident populations for Australia and each state or territory at June 2013.[2] Population data are supplied as an estimate for calendar years; for this report, the population for the second half of the financial year was applied (2013 population applied to the 2012–13 financial year). Analyses were conducted using Microsoft Excel® and Stata SE version 13.

Due to a limitation of surveillance systems, Queensland notifies mixed infections of malaria as a separate notification for each infecting organism. For the 2012–13 season, additional information was collected to enable these mixed infections to be reported as 1 case for the purpose of this report, resulting in 3 fewer notifications than if the adjustment was not made.

Additional information on the details of some notifications was obtained from state and territory public health surveillance managers. Data on sentinel chicken surveillance and vector (including detection of exotic mosquitoes at the border) and virus surveillance are also reported here.

Vertebrate, vector and climate surveillance in states and territories

Sentinel chicken flavivirus surveillance programs aim to provide early warning of the endemic arboviruses MVEV and KUNV and where relevant, exotic flaviviruses such as JEV.[3] Public health messaging or other response measures can be implemented in response to surveillance signals. Public health messaging may advise at-risk residents or target groups such as campers or fishermen of the need to take added precautions to avoid mosquito bites. Sentinel chicken flocks are an important component of the early warning system in several jurisdictions, and these are located geographically to detect flavivirus activity and provide a timely and accurate indication of the risk of transmission to people (Map).[4] Detailed descriptions of the sentinel chicken, vector and virus surveillance programs, as well as contact details for jurisdictional arbovirus reference/research laboratories are included in the Appendix.

Results

During the 2012–13 season, there were 9,726 notifications of mosquito-borne diseases in humans (Table 1). This represented a 16% increase from the mean of 8,404.2 notifications for the previous 5 years.

Table 1: Number of notified human cases, notification rate* and 5 year mean for mosquito-borne disease, Australia, 2012–13, by disease and state or territory

		ACT	NSW	NT	Qld	SA	Tas.	Vic.	WA	Aust. total
Barmah Forest virus infection	Cases 2012–13	5	456	353	2,025	84	2	70	926	3,921
	5 year mean cases	3.8	398.6	78.6	926.2	61.2	1.6	69.2	135.4	16,74.6
	Rate 2012–13	1.3	6.8	87.5	36.2	10.6	1.2	3.3	42.9	16.7
	5 year mean rate	3.8	12.7	129.9	48.3	26.8	6.8	8.2	37.9	23.4
Chikungunya virus infection	Cases 2012–13	0	12	1	11	7	1	26	38	96
	5 year mean cases	0.0	7.6	2.8	2.8	1.4	0.4	9.4	5.4	29.8
	Rate 2012–13	–	–	–	–	–	–	–	–	–
	5 year mean rate	–	–	–	–	–	–	–	–	–
Dengue virus infection	Cases 2012–13	16	263	38	425	49	8	305	326	1,430
	5 year mean cases	13.2	174.8	37.6	384.8	29.0	5.6	101.6	305.8	1,052.4
	Rate 2012–13	–	–	–	–	–	–	–	–	–
	5 year mean rate	–	–	–	–	–	–	–	–	–
Flavivirus (unspecified)†	Cases 2012–13	0	0	1	6	0	0	0	0	7
	5 year mean cases	0.0	0.2	0.0	4.0	0.2	0.0	6.4	0.0	10.8
	Rate 2012–13	–	–	–	–	–	–	–	–	–
	5 year mean rate	–	–	–	–	–	–	–	–	–
Japanese encephalitis virus infection	Cases 2012–13	0	0	0	0	1	0	0	1	2
	5 year mean cases	0.0	0.2	0.0	0.2	0.0	0.0	0.0	0.0	0.4
	Rate 2012–13	–	–	–	–	–	–	–	–	–
	5 year mean rate	–	–	–	–	–	–	–	–	–
West Nile virus/Kunjin virus infection	Cases 2012–13	0	0	0	0	0	0	0	0	0
	5 year mean cases	0.0	0.2	0.6	0.6	0.0	0.0	0.2	0.0	1.6
	Rate 2012–13	–	–	–	–	–	–	–	–	–
	5 year mean rate	–	–	–	–	–	–	–	–	–
Malaria	Cases 2012–13	16	81	23	105	10	9	89	82	415
	5 year mean cases	11.8	98.0	17.2	144.6	14.8	7.6	90.6	66.2	450.8
	Rate 2012–13	–	–	–	–	–	–	–	–	–
	5 year mean rate	–	–	–	–	–	–	–	–	–
Murray Valley encephalitis virus infection	Cases 2012–13	0	0	0	0	0	0	0	0	0
	5 year mean cases	0.0	0.8	0.6	0.4	0.4	0.0	0.0	2.4	4.6
	Rate 2012–13	–	–	–	–	–	–	–	–	–
	5 year mean rate	–	–	–	–	–	–	–	–	–
Ross River virus infection	Cases 2012–13	5	502	211	1,683	177	6	190	1,081	3,855
	5 year mean cases	13.6	909.6	295.4	2,149.4	440.4	34.0	458.6	877.8	5,178.8
	Rate 2012–13	1.3	6.8	87.5	36.2	10.6	1.2	3.3	42.9	16.7
	5 year mean rate	3.8	12.7	129.9	48.3	26.8	6.8	8.2	37.9	23.4
Yellow fever	Cases 2012–13	0	0	0	0	0	0	0	0	0
	5 year mean cases	0	0	0	0.3	0	0	0	0	0
	Rate 2012–13	–	–	–	–	–	–	–	–	–
	5 year mean rate	–	–	–	–	–	–	–	–	–
Total cases 2012–13		42	1,314	627	4,255	328	26	680	2,454	9,726

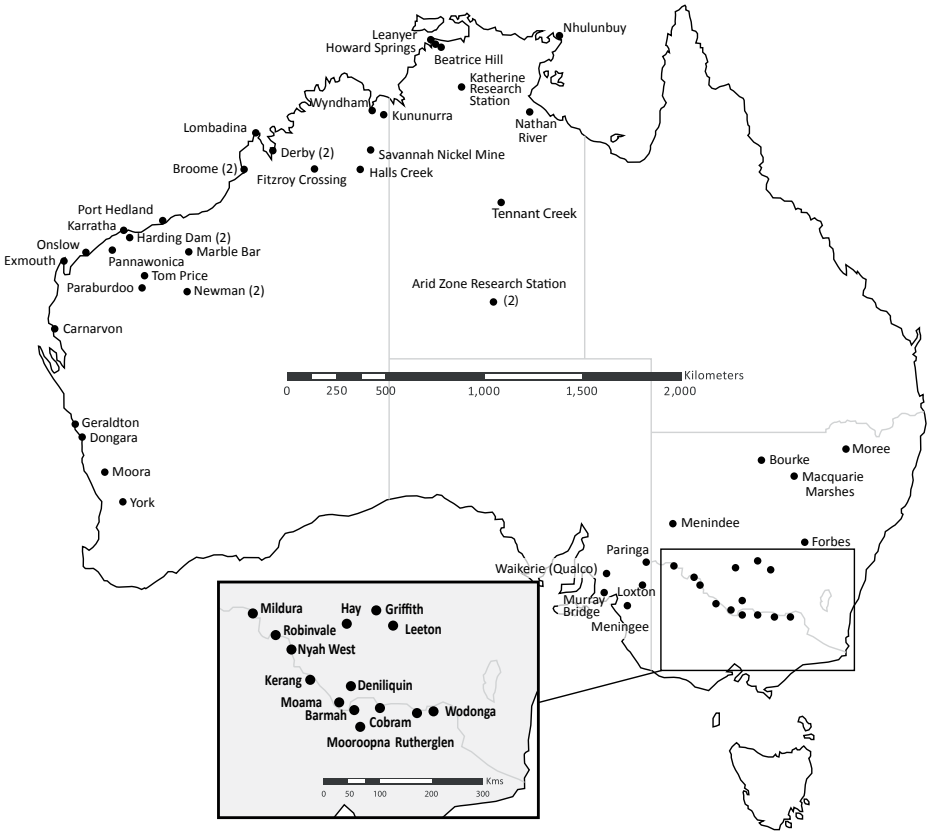
* Rates are not provided for diseases with less than 20 cases, or for diseases predominantly acquired overseas.

† Flavivirus (NEC) replaced Arbovirus (NEC) from 1 January 2004. Arbovirus (NEC) replaced Flavivirus (NEC) from 2008. Flavivirus (unspecified) replaced arbovirus (NEC) from 1 July 2015.

NEC Not elsewhere classified.

NN Not notifiable.

Map: Location of sentinel chicken sites, Australia, 2012–13



Alphaviruses

In Australia, the most frequently notified viruses in the genus alphavirus are RRV and BFV. RRV and BFV occur exclusively in the Australasian region.[5] Infection with RRV or BFV can cause illness characterised by fever, rash and polyarthritis. These viruses are transmitted by numerous species of mosquitoes that breed in diverse environments (freshwater habitats, coastal regions, salt marshes, floodwaters, established wetlands and urban areas).[6] However, there are known problems with the unreliability of serological tests that diagnose infection on the basis of IgM only and with the case definitions which allow for confirmation based on these tests, leading to over diagnosis particularly during the off-season.[7,8] Importantly, the case definitions have been reviewed by the case definitions working group (CDWG) and endorsed by CDNA. The revised case definitions were implemented on 1 January 2016.

Local transmission of the alphavirus CHIKV has not occurred in Australia, but the infection is regularly reported in travellers returning from overseas. Illness is characterised by an abrupt onset of fever, rash and severe joint pain. The acute disease lasts 1 to 10 days, but convalescence may include prolonged joint swelling and pain lasting months. Haemorrhagic manifestations may occur occasionally.[9] Humans are amplification hosts for CHIKV, and other vertebrates are not required for transmission to occur. Internationally, CHIKV is most commonly transmitted by *Aedes aegypti*, which occurs in northern Queensland and *Aedes albopictus,* which is found on Cocos Island, Christmas Island and in some areas of the Torres Strait Islands.[10] Other Australian mosquito species have been shown to be competent vectors of CHIKV in the laboratory,[11] but any role in field transmission is likely to be minor compared with either *Ae. aegypti* or *Ae. albopictus*.[12]

Barmah Forest virus infections

There were 3,921 notifications of BFV infections during the 2012–13 season, representing a rate of 16.7 per 100,000 population, a decrease from the mean of 23.4 per 100,000 for the previous 5 years (Table 1). Queensland reported the largest number of notifications of BFV infection (n=2,025) while the highest rate was reported in the Northern Territory (353 per 100,000 population). Rates in 2012–13 increased sharply for most state and territories compared to the past 5 years (Figure 1), and were greater than 2.0 times the 5 year mean for the Northern Territory, Queensland and Western Australia. Comparisons between regions are likely to be influenced by accuracy of case-ascertainment, which may vary between jurisdic-

tions due to differences in reporting criteria and diagnostic tests used (in particular, with IgM test kits).[13] It is important to note that seasonal trends vary between and within states and territories according to differences in mosquito vectors, hosts and climate. In addition, comparisons between regions are likely to be influenced by accuracy of case-ascertainment, which may vary between jurisdictions because of some differences in reporting criteria and the quality of diagnostic tests used, with false positive IgMs a long term issue.[8,14]

In 2012–13, infections were most frequently notified between February and May (Figure 2). BFV infections are more common during the warmer months when suitable mosquito vectors are abundant, and are unexpected outside of these warmer months. The higher than expected numbers of BFV notifications in the cooler months is likely to be an artefact, reflecting the likelihood of false positive IgM diagnoses. In October 2012, the number of BFV notifications began to increase dramatically, and marked the start of an 'epidemic' of notifications due to false positive IgM diagnoses, which continued to the end of 2012–13 and into 2013–14 (data not shown).

In 2012–13, BFV notifications were most commonly reported among middle aged adults, with notification rates peaking in the 40–64 year age range for females, and with a secondary peak of notifications among females in the 15–39 years age group females (Figure 3). Rates among younger and middle aged females were increased compared with the previous year, with rates of 12.2 and 13.0 per 100,000 among females aged 15–19 and 20–24 years respectively in 2012–13, compared with 5.6 and 6.7 per 100,000 in 2011–12. In 2012–13, 41% of cases were male, which was lower than in previous years (48% of cases during the previous 5 years were male).

Figure 1: Notification rate for Barmah Forest virus infection, Australia, July 2007 to June 2013, by year and state or territory

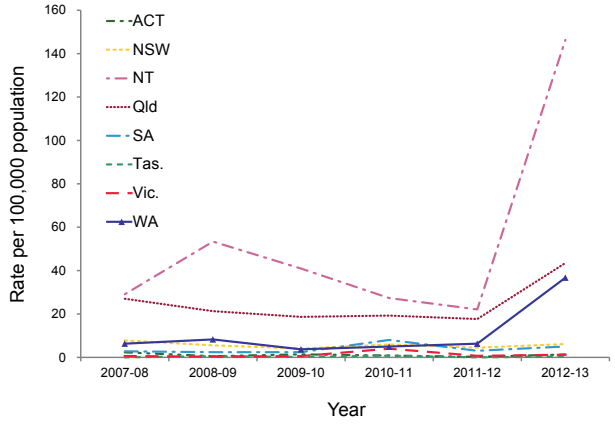
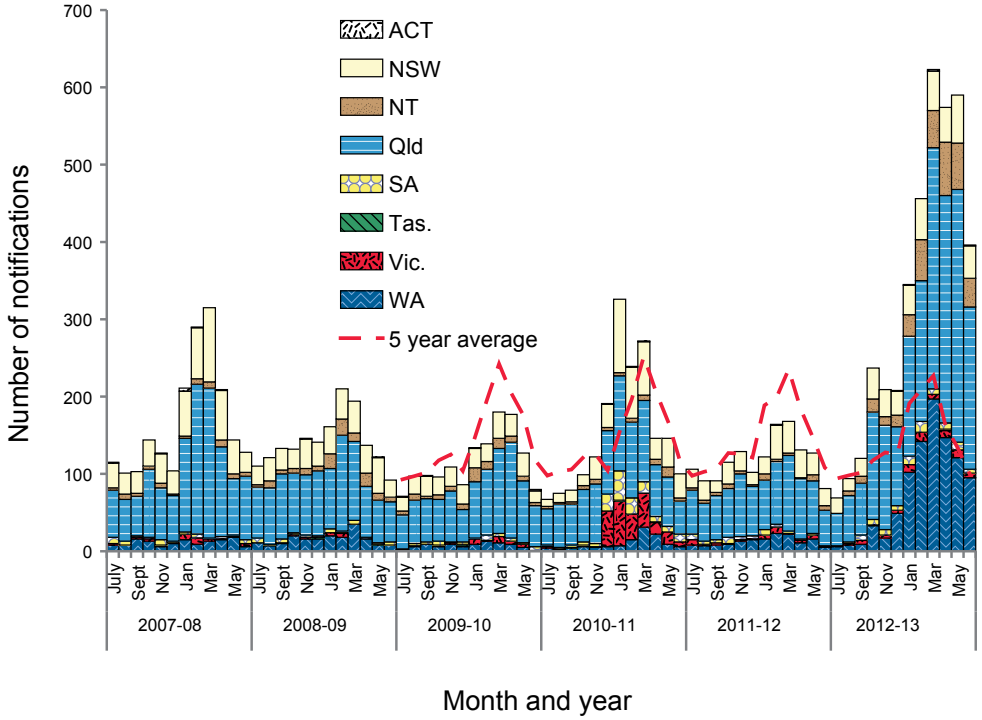


Figure 2: Notifications of Barmah Forest virus infection, Australia, July 2007 to June 2013, by month and year



Ross River virus infections

There were 3,855 notifications of RRV infection during the 2012–13 season, representing a rate of 16.7 per 100,000 population, compared with a 5-year mean of 5,178.8 notifications and a rate of 23.4 per 100,000 (Table 1, Figure 4). Queensland reported the largest number of cases (n=1,683), while the highest rate was in the Northern Territory (Figure 5).

Rates of RRV were similar to or below the 5-year mean in all jurisdictions (Table 1). RRV was most commonly reported among younger and middle-aged adults, with notification rates peaking in the 35–49 year age groups (Figure 6). In 2012–13, 47% of cases were male.

Figure 5: Notification rate for Ross River virus infection, Australia, 1 July 2007 to 30 June 2013, by year and state or territory

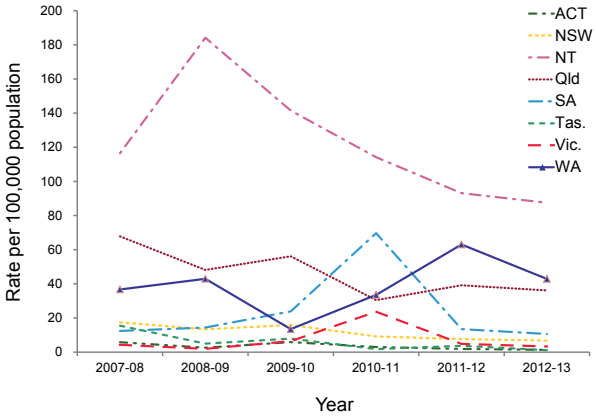


Figure 3: Notification rate for Barmah Forest virus infection, Australia, 2011–12 and 2012–13, by age group and sex (n=5,348)

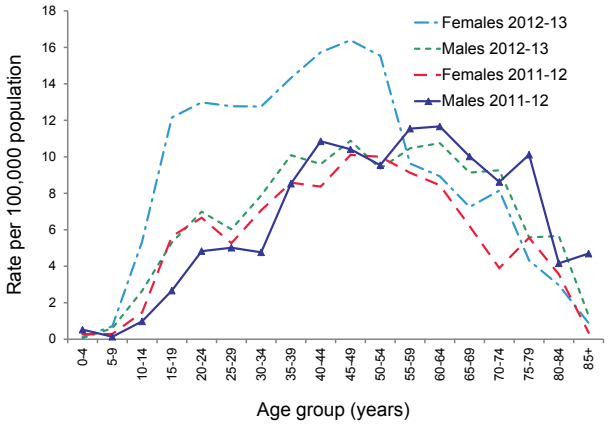


Figure 6: Notification rate for Ross River virus infection, Australia, 2012–13, by age group and sex (n=3,855)

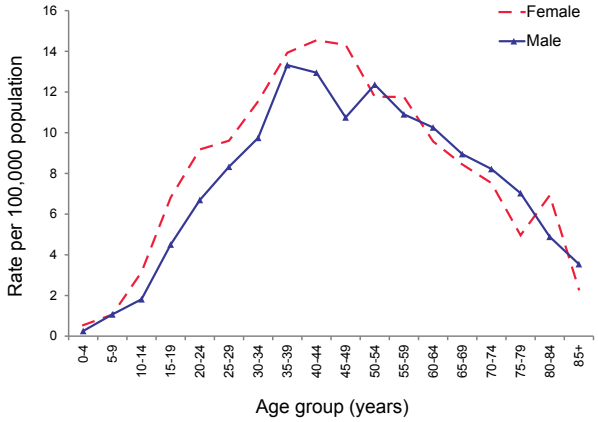
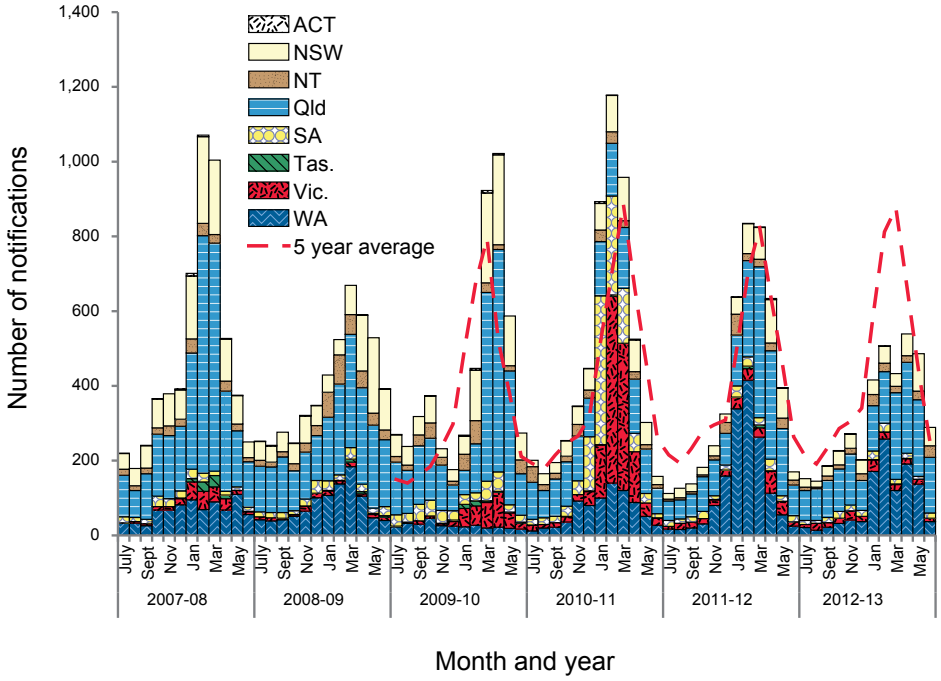


Figure 4: Notifications of Ross River virus infection, 1 July 2007 to 30 June 2013, by month



As in previous years, there was a marked seasonal trend in RRV notifications, with the largest number notified between January and April (Figure 4). It is important to note that as for BFV, seasonal trends vary between and within states and territories according to differences in mosquito vectors, hosts and climate. In addition, comparisons between regions are likely to be influenced by accuracy of case-ascertainment, which may vary between jurisdictions because of some differences in reporting criteria and the quality of diagnostic tests used, with false positive IgMs a long term issue.[8,14]

Chikungunya virus infection

There were 96 notifications of CHIKV infection during the 2012–13 season compared with a 5–year mean of 29.8 cases (Table 1, Figure 7). This was the largest number of cases ever reported in Australia. All cases were acquired overseas, with complete information supplied on the country of acquisition for 60% (58/96) of these (Table 2). The most frequently reported country of acquisition was Indonesia (34 cases). There were 13 importations from Papua New Guinea, where chikungunya was reported for the first time in June 2012 on PacNet,

Figure 7: Notifications of chikungunya virus infection, Australia, 2012–13, by month and year and state or territory

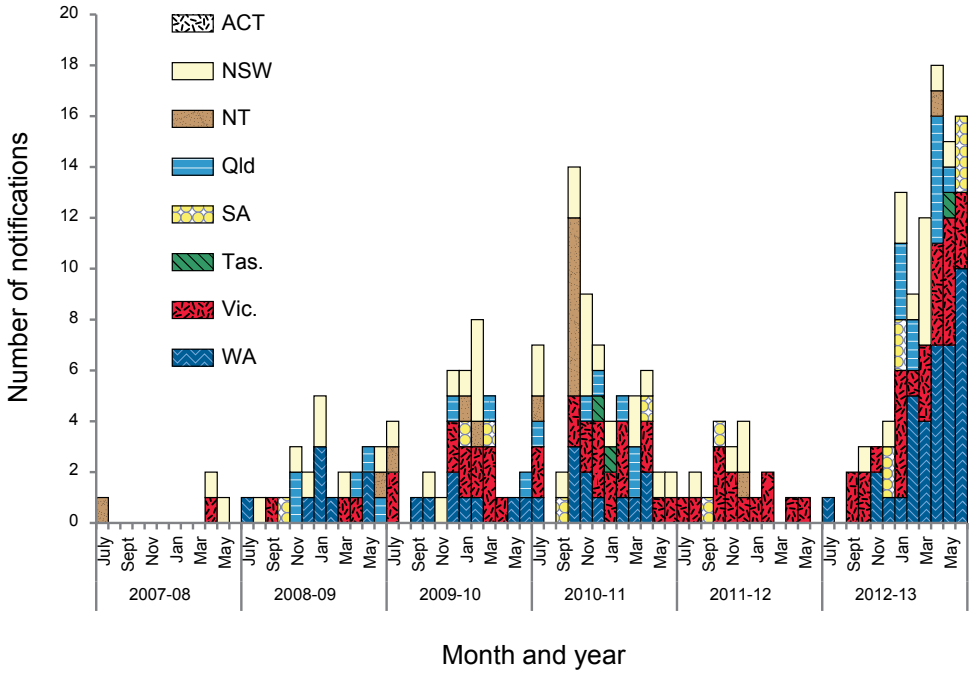


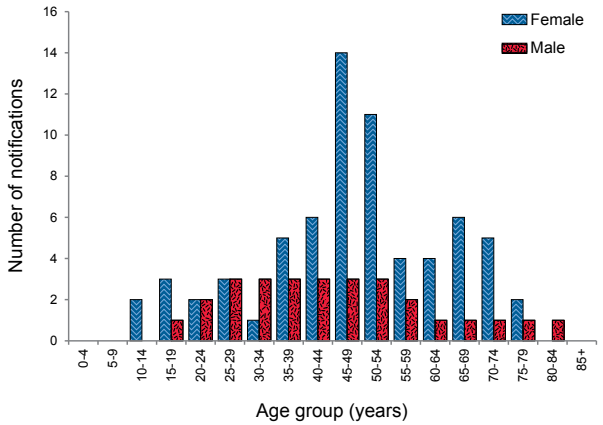
Table 2: Notifications of chikungunya virus infection, Australia, by year and country or region of acquisition

Country or region of acquisition	2008–09	2009–10	2010–11	2011–12	2012–13
Indonesia	2	7	32	2	34
Papua New Guinea	0	0	2	0	13
India	1	14	11	6	2
Philippines	0	1	0	2	2
Thailand	3	0	2	3	2
Malaysia	6	4	1	1	1
Cambodia	0	0	0	0	1
Vietnam	0	0	2	0	1
Kenya	0	0	0	0	1
Other countries or regions	2	10	11	4	0
Overseas – country unknown	11	1	2	2	39
Total	25	37	63	20	96

the Pacific Public Health Surveillance Network early warning system, with widespread outbreaks that continued into June 2013.[15,16]

CHIKV infection was most frequently notified among young and middle aged adults, particularly women aged between 45 and 54 years (Figure 8). The median age was 48 years and 71% of cases were female.

Figure 8: Notifications of chikungunya virus infection, Australia, 2012–13, by age and sex



Flaviviruses

This section provides information on several flaviviruses notified to NNDSS including DENV, MVEV, KUNV and JEV. Other flaviviruses may be notified under the flavivirus (unspecified) category.

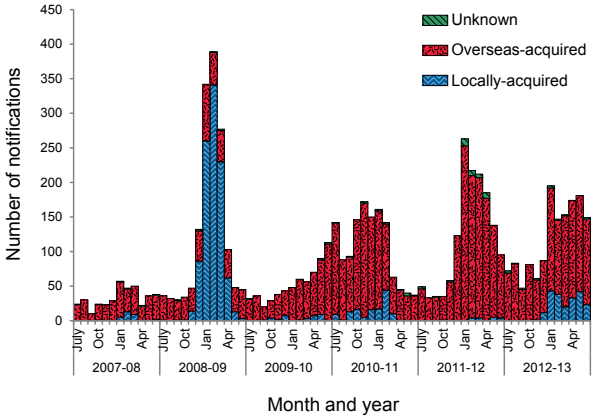
Four serotypes of dengue virus have been described and all 4 are reported in imported cases to varying degrees each year, some of which may result in local outbreaks. The clinical illness is characterised by mild to severe febrile illness with fever, headache, muscle/joint pain and sometimes a rash. A minority of cases progress to severe dengue with haemorrhage and shock, more commonly where, in a second or subsequent infection, a person is infected with a different DENV serotype to the first infection. Local transmission of dengue in Australia is normally restricted to areas of northern Queensland where the key mosquito vector, *Ae. aegypti,* is present in sufficient numbers and near human populations of sufficient size.[17] Dengue is not endemic in North Queensland, but local transmission can occur upon introduction of the virus to the mosquito vector by a viraemic tourist or a resident returning from a dengue-affected area overseas.[18]

Infection with MVEV, KUNV or JEV is usually asymptomatic or produces a non-specific illness, but a small percentage of cases progress to encephalomyelitis of variable severity. *Culex annulirostris* is the major vector of MVEV, KUNV and JEV. No specific treatment is available for these diseases and care is largely supportive. A vaccine is available to prevent JEV infection (available for residents in affected areas of Queensland and for long term travellers to endemic areas)[19] but there are no vaccines currently available for DENV, MVEV or KUNV. YFV does not occur in Australia, but travellers to affected areas overseas need to be aware of the risks and vaccination requirements, and there is a risk of transmission in the areas of North Queensland in which the vector *Ae. aegypti* is present.

Dengue virus infection

There were 1,430 notifications of DENV infection during the 2012–13 season. Of these, 212 cases were acquired in Australia, while the majority (1,202 cases) acquired the infection overseas (Table 3, Figure 9). For the remaining 16 cases, no information on place of acquisition was supplied. In 2012–13, the median age of cases was 39 years (range 0–86 years), and 51% (n=733) of cases were male.

Figure 9: Notifications of dengue virus infection, Australia, 1 July 2007 to 30 June 2013, by month, year and place of acquisition



Locally-acquired dengue virus infection

The 212 notified cases of DENV infection acquired in Australia during 2012–13 was a marked increase compared with the 18 locally-acquired cases in 2011–12, but was more consistent with previous years (Table 3). Of these, 206 were reported by Queensland, and 6 by other states.

In Queensland, a single case of locally-acquired dengue is considered an outbreak. Ten discrete dengue outbreaks were reported by Queensland Health in the 2012–2013 season, all located in the north of the state. A total of 203 notifications were

known to have been associated with these outbreaks, with the number of cases in each outbreak ranging from 1 to 138 (these numbers do not match exactly with the 189 reported from NNDSS due to differences in the dates used for data extraction). Seven of the 10 outbreaks, including the largest, were attributable to dengue serotype 1. One outbreak each was associated with dengue serotypes 2 and 3 and the serotype responsible for the remaining outbreak was unknown. The unknown outbreak consisted of only a single case where the serotyping could not be completed.

The 6 notifications of locally-acquired dengue from other states were listed in NNDSS as being acquired in Queensland.

Overseas-acquired dengue virus infection

There were 1,202 notifications of DENV infection acquired overseas during the 2012–13 season (Table 3), 1.5 times the 5-year mean of overseas-acquired infections (793.8). Almost all states and territories reported increased numbers of notified cases of overseas-acquired DENV infection compared with the long-term average. The ratio of notifications in 2012–13 compared with the 5-year mean ranged from 1.0 in the Northern Territory to 3.2 in Victoria.

A specific country or region of acquisition was supplied for 73% (879/1,202) of cases listed as overseas-acquired (Table 4). Indonesia was the country of acquisition for nearly a quarter of overseas acquired cases for which a specific country or region was available (24%, n=278), much lower than the 64% in 2011–12.[7] The infecting DENV serotype was determined for 42% (n=506) of overseas-acquired dengue cases (up from 23% in 2011–12 but similar to the 50% in 2010–11). DENV 1 (n=210) was the most frequently reported serotype in 2012–13 (Table 4).

Table 3: Notifications of dengue virus infection, Australia, 1 July 2007 to 30 June 2013, by year, state or territory and place of acquisition

Place of acquisition	Year	ACT	NSW	NT	Qld	SA	Tas.	Vic.	WA	Aust.
Locally-acquired*	2007–08	0	2	0	26	2	0	0	0	30
	2008–09	0	5	0	1,003	1	0	3	0	1,012
	2009–10	0	2	0	33	0	0	0	0	35
	2010–11	0	2	1	125	0	0	2	1	131
	2011–12	0	1	0	16	0	0	1	0	18
	2012–13	0	0	0	206	2	0	4	0	212
Overseas-acquired	2007–08	4	103	25	78	31	4	15	94	354
	2008–09	14	169	27	115	26	6	19	121	497
	2009–10	19	121	36	126	11	4	52	226	595
	2010–11	4	222	29	181	28	5	140	525	1,134
	2011–12	11	240	69	209	44	9	246	561	1,389
	2012–13	12	257	38	216	47	8	299	325	1,202
Unknown	2007–08	0	0	0	4	2	0	0	0	6
	2008–09	0	0	0	5	0	0	1	0	6
	2009–10	0	3	0	1	0	0	1	0	5
	2010–11	8	2	1	2	0	0	0	1	14
	2011–12	6	2	0	0	0	0	28	0	36
	2012–13	4	6	0	3	0	0	2	1	16
Total	2007–08	4	105	25	108	35	4	15	94	390
	2008–09	14	174	27	1,123	27	6	23	121	1,515
	2009–10	19	126	36	160	11	4	53	226	635
	2010–11	12	226	31	308	28	5	142	527	1,279
	2011–12	17	243	69	225	44	9	275	561	1,443
	2012–13	16	263	38	425	49	8	305	326	1,430

* Locally-acquired cases are acquired in Australia and not necessarily in the state or territory from which they are reported. Under the cross-border notification protocol, cases are notified by their state or territory of residence where this differs from the diagnosing state or territory.

Table 4: Overseas acquired cases of dengue virus infection, Australia, 2012–13, by serotype and country of acquisition

Country/ or region	Total number	Percentage of cases*	Serotype 1	Serotype 1 and 4	Serotype 2	Serotype 3	Serotype 4	Unknown/ untyped
Indonesia	278	24	43	0	20	22	2	191
Thailand	217	19	31	0	11	16	0	159
India	63	5	4	0	5	12	0	42
Philippines	46	4	9	1	3	0	2	31
Papua New Guinea	35	3	6	0	6	5	0	18
Cambodia	34	3	8	0	0	1	1	24
Sri Lanka	29	2	7	0	0	3	0	19
Malaysia	19	2	1	0	0	0	3	15
Timor-Leste	19	2	2	0	0	9	0	8
Vietnam	17	1	2	0	2	2	0	11
Fiji	17	1	5	0	3	0	0	9
Bangladesh	14	1	1	0	0	0	0	13
Solomon Islands	14	1	0	0	0	6	0	8
South-East Asia, nfd	9	1	0	0	0	1	0	8
Singapore	9	1	0	0	2	1	0	6
New Caledonia	7	1	3	0	0	0	0	4
Myanmar, The Republic of the Union of	4	0	2	0	0	0	0	2
Sudan	3	0	0	0	0	0	0	3
South America, nfd	3	0	1	0	0	0	0	2
China†	3	0	0	0	0	0	0	3
Maldives	3	0	1	0	0	0	0	2
Vanuatu	3	0	1	0	0	0	0	2
Other countries‡	33	3	6	0	2	2	2	21
Overseas – country unknown	323		77	0	96	44	11	95
Total	1,202		210	1	150	124	21	696

* The denominator excludes cases with place of acquisition "Overseas–country unknown". Percentages do not add up due to rounding.

† Excludes special administrative regions and Taiwan.

‡ Each country with less than 3 cases.

nfd Not further defined

Flavivirus infection (unspecified)

This disease category enables the capture and epidemiological analysis of emerging infections within this very broad disease group. Emerging diseases can be made nationally notifiable if required. An unspecified category is particularly important for the flaviviruses, because it is recognised that some infections cannot be attributed to a single flavivirus.

There were 7 notifications of flavivirus (unspecified) in 2012–13, similar to the 5-year average of 10.8 cases. All were confirmed infections as per the case definition. Five notifications relate to infections that were known to have been acquired overseas while for the remaining 2, the place of acquisition was unknown. In 2012–13, 2 notifications were for Kokobera, 1 was for Zika (acquired in Indonesia) and for the remainder, the infection could not be attributed to a specific flavivirus (Table 5).

The largest number of notifications were from Queensland (n=6). In Queensland, an extensive panel of flaviviruses is used for testing. Flaviviruses

Table 5: Notifications of flavivirus infection (unspecified), Australia, 2012–13

Virus species	Country of acquisition	State or territory	Month
Kokobera	Place of acquisition unknown	Qld	Jan
Kokobera	Place of acquisition unknown	Qld	Mar
Unspecified	Thailand	Qld	July
Unspecified	Philippines	Qld	Jan
Unspecified	Thailand	Qld	May
Unspecified	Indonesia	Qld	Jun
Zika	Indonesia	NT	Mar

may be more prevalent particularly in the north of the state, so patients may be more likely to be exposed to more than 1 flavivirus, and these factors could increase the probability of cross-reacting antibodies (Dr Sonya Bennett, Queensland Health, personal communication) resulting in more notifications of flavivirus (unspecified).

Japanese encephalitis virus infections

There were 2 notifications of JEV infection in Australia during 2012–13. The first was notified by Western Australia in a 41-year-old woman who acquired the infection in Indonesia, and the second was notified by South Australia in a 57-year-old man who had been resident in Thailand during the 3 years prior to onset and was transferred to Australia for treatment.

JEV infection is a rare disease in Australia, with an average of 0.4 cases per year during the past 5 years. The last locally-acquired case was in 1998.[20]

West Nile virus/Kunjin virus infection

This category includes all WNV infections, including KUNV, which is an Australian lineage and has not been isolated from anywhere except on the Australian mainland and Torres Strait, and other WNV infections that are acquired overseas. While infection with KUNV is probably not uncommon in northern Australia, clinical KUNV cases are rare in Australia.[21,22]

There were no notifications of WNV/KUNV infection in Australia in 2012–13. There was an average of 1.6 cases per year during the past 5 years.

Murray Valley encephalitis virus infection

There were no notifications of MVEV infection in Australia in 2012–13. MVEV infection is a rare disease in Australia, with an average of 4.6 cases per year during the past 5 years.

Yellow fever virus infection

There were no notifications of yellow fever virus infection in 2012–13. The only previous notifications of yellow fever were in 2011, and while the notifications met the surveillance case definition at the time, they were thought to be vaccine-associated. The surveillance case definition has since been revised to exclude vaccine associated cases.

Malaria

Malaria is a serious acute febrile illness that is transmitted from person to person through the bite of an infected mosquito of the genus *Anopheles*. It is caused by a protozoan parasite in the genus *Plasmodium* that includes 5 species that infect humans – *Plasmodium vivax*, *Plasmodium falciparum*, *Plasmodium malariae*, *Plasmodium ovale* and *Plasmodium knowlesi*.[23,24]

Australia is free of endemic malaria, but suitable vectors are present in northern Australia, and the area remains malaria-receptive. Malaria in Australia is therefore a disease associated with residing or travelling overseas in areas with endemic transmission. A case series in the Northern Territory showed that malaria cases were reported in travellers returning from endemic areas, but also reflected current events such as military operations and increased refugee arrivals from malaria endemic areas.[25] The last cases acquired on mainland Australia were during an outbreak in North Queensland in 2002.[26] Limited transmission occurs occasionally in the Torres Strait following importation. The most recent locally-acquired cases of malaria in Australia were a single case in 2013 acquired on Saibai Island in the Torres Strait and 7 locally-acquired cases in the Torres Strait in 2011.

There were 415 notifications of malaria during 2012–13 (Table 1), an 8% decrease compared with the mean of 450.8 notifications during the past 5 years, which is consistent with the steady decline in the number of notifications since 2004–05 (Figure 10). There was 1 locally-acquired case of malaria in Australia in 2012–13 (acquired in the

Torres Strait). Complete information on the overseas country or region of acquisition was supplied for 76% of overseas-acquired cases (316/414).

Malaria was most frequently reported among people aged 25–29 years, with 59 notified cases in this age group (Figure 11). Similar to previous years, the majority of cases were male (71%, n=294), and males predominated in every age group except those aged 70–74 years.

The infecting species was reported for 99% of notifications during 2012–13. *P. falciparum* and *P. vivax* were the predominant species (Table 6). No cases were infected with *P. knowlesi*. *P. vivax* infections were commonly associated with travel to Asia or Pacific nations while. *P. falciparum* infections were frequently associated with travel to the Middle East, Africa and Papua New Guinea.

Complete information about the country of acquisition was available for 77% (n=315) of malaria cases. Papua New Guinea was the most frequently reported place of acquisition (13%, 53/415), followed by India (12%, 50/415) (Table 6, Figure 12).

Figure 10: Notifications of malaria, Australia, 1 July 1991 to 30 June 2013

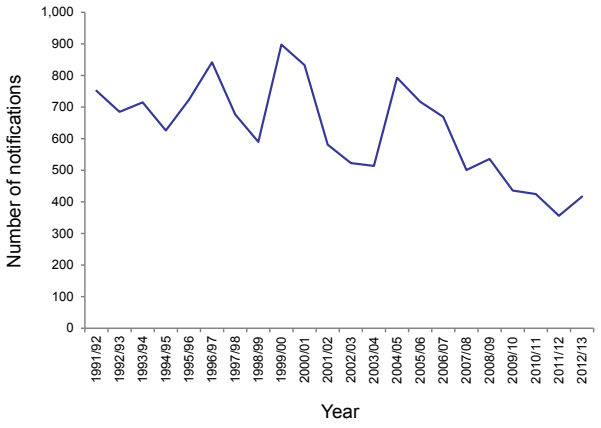
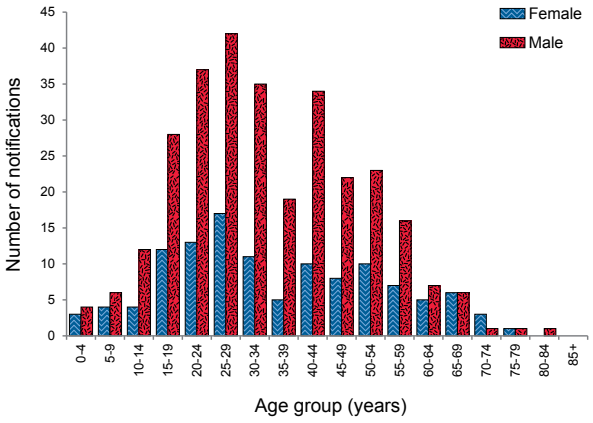
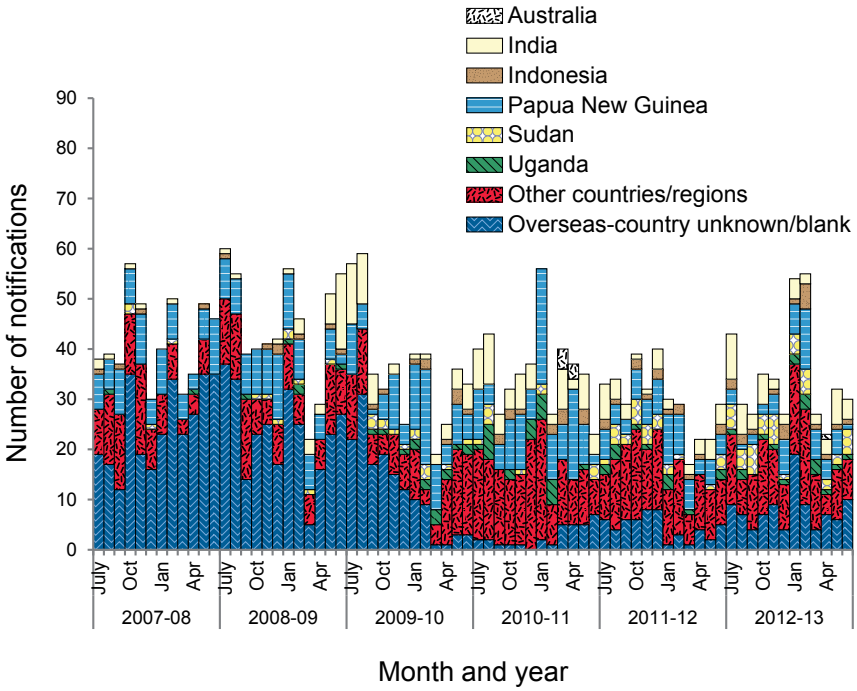


Figure 11: Notifications of malaria infection, Australia, 2012–13, by age group and sex*



* Cases were excluded where sex or age data were not available (n=2 cases).

Figure 12: Notifications of malaria, Australia, 1 July 2007 to 30 June 2013, by month, year and place of acquisition*



Note: Other countries/regions each had less than 17 cases in 2012–13.

Table 6: Cases of malaria, Australia, 2012–13, by *Plasmodium* species and country or region of acquisition

Country or region of acquisition	*Plasmodium falciparum*	*Plasmodium malariae*	*Plasmodium ovale*	*Plasmodium vivax*	Mixed species infections	*Plasmodium* spp	Total	% of all cases
Papua New Guinea	21	1	0	30	0	1	53	13
India	3	0	0	47	0	0	50	12
Sudan	40	0	3	1	0	0	44	11
Indonesia	6	0	1	12	0	0	19	5
Uganda	14	2	1	0	0	0	17	4
Pakistan	0	0	0	16	0	0	16	4
Tanzania	10	1	0	0	0	0	11	3
Kenya	7	0	1	0	1	0	9	2
Sub-Saharan Africa, nfd	8	1	0	0	0	0	9	2
Solomon Islands	0	0	0	8	0	0	8	2
Ghana	7	0	0	0	0	0	7	2
Nigeria	5	0	2	0	0	0	7	2
Sierra Leone	6	0	0	0	0	1	7	2
Guinea	6	0	0	0	0	0	6	1
Liberia	3	0	1	1	0	0	5	1
Southern and East Africa, nfd	3	0	1	0	0	0	4	1
Other countries or regions*	23	2	1	17	0	1	44	11
Australia	1	0	2	2	0	0	5	1
Overseas – country unknown	64	2	1	22	3	2	94	23
Total	227	9	14	156	4	5	415	100
% of cases	55	2	3	38	1	1	100	

* Each with less than 4 cases.

nfd Not further defined.

Sentinel chicken, arbovirus detections in mosquitoes and mosquito abundance monitoring

New South Wales

The season began with 150 pullets and a total of 2,929 samples were received from the 10 flocks in New South Wales over the 6-month period in 2012–2013. This represented 5,858 enzyme-linked immunosorbent assay (ELISA) tests (excluding controls and quality assurance samples), with each specimen being tested for MVEV and KUNV antibodies. There were no seroconversions to MVEV or KUNV recorded through the season.

For 2012–13 the climatic conditions leading up to the season for the inland were of well below average rainfall for the last quarter of 2012, whereas rainfall was average for the 1st quarter of 2013. Neither the Forbes nor the Nicholls hypotheses were suggestive of possible MVEV activity for the 2012–13 season. Despite the very dry spring months, moderate mosquito numbers were collected with close to 130,000 being trapped. However, arboviral activity was low; there were few isolates and no seroconversions in the sentinel chickens. Human notifications of arboviral infection were below normal; particularly from the inland where alphavirus notifications (RRV and BFV combined) were close to half the long term average.

For the coast, the climatic conditions were mostly similar to the inland (namely dry late 2012), however for the north coast, heavy precipitation fell during the first 3 months of 2013. With the ongoing wet conditions during the summer months, numbers of *Aedes vigilax* were quite low and only comprised around 10% of the overall mosquito collections. With reduced abundance of the major coastal vector, alphavirus activity was relatively minimal. Coastal disease notifications of RRV and BFV were 22% below the long-term average.

In a collaborative research project with colleagues in the United States of America, several new arboviruses were identified from New South Wales, including Liao Ning (previously only known from China), Beaumont, North Creek, Murrumbidgee, and Salt Ash viruses (all new). It is unknown if these have human health implications.

Further detail can be found in the New South Wales Arbovirus Surveillance Program annual reports (http://medent.usyd.edu.au/arbovirus/information/publications.htm).

Northern Territory

In 2012–13 there were 211 laboratory identified cases of RRV in the Northern Territory, which was similar to the 222 cases reported in 2011–12. Most cases were recorded in the Darwin region, and predominantly occurred in January and June, which coincided with a high number of *Ae. vigilax* in December and a high number of *Culex annulirostris* in June (Table 7).

A low number of RRV infections was recorded in the East Arnhem region, Katherine region and the Alice Springs region, with no cases recorded in the Barkly region in 2012–13.

As part of the investigation into the increased number of BFV disease cases (354) in the Northern Territory in 2012–13, mosquito trapping and virus isolations were carried out in liaison with the Department of Primary Industries and Fisheries (DPIF) in May 2013, to determine what levels of BFV were circulating in mosquito populations in the Darwin urban area. A total of 4,641 mosquitoes were tested for the presence of virus (Table 2). No BFV was isolated but 2 unidentified viruses were isolated from *Cx. annulirostris*. Most BFV cases in 2012–13 are believed to have been false positives.

In the 2012–13 season, no chickens seroconverted to MVEV or KUNV in the Northern Territory. One seroconversion to an unspecified flavivirus occurred at the Adelaide River Coastal Plains Research Station in May 2013 (Table 8).

However, between December 2012 and June 2013, honey bait traps were trialled at Leanyer (Darwin) and Beatrice Hill Research Farm near Fogg Dam to test the suitability of the new system for flavivirus surveillance. In April 2013, in a sugar-based surveillance system, the Flinders Technology Associates (FTA) cards from the trap set at Beatrice Hill Research Farm tested positive for MVEV (Table 8). This was the first detection of MVEV activity in Australia from FTA cards.[27]

Further details are available from the Northern Territory Medical Entomology annual reports (http://www.health.nt.gov.au/Medical_Entomology/index.aspx).

Table 7: Key mosquito vector abundance in selected regions, Australia, 2012–13, by species, state or territory, region and month

Species	State or territory	Region or locality	Month											
			July	Aug	Sept	Oct	Nov	Dec	Jan	Feb	Mar	Apr	May	Jun
Saltwater														
Aedes vigilax	NSW	North Coast	–	–	–	–	LOW	LOW	MED	MED	LOW	LOW	LOW	–
Ae. vigilax	NSW	Mid–North Coast	–	–	–	–	–	LOW	LOW	MED	LOW	LOW	LOW	–
Ae. vigilax	NSW	Central Coast	–	–	–	–	–	MED	HIGH	HIGH	LOW	LOW	LOW	–
Ae. vigilax	NSW	Sydney – Georges River	–	–	–	–	–		HIGH	HIGH	HIGH			–
Ae. vigilax	NSW	Sydney – Homebush	–	–	–	–	LOW	LOW	MED	HIGH	MED	HIGH		–
Ae. vigilax	NSW	Sydney – Western	–	–	–	–	–	LOW	LOW	LOW	LOW			–
Ae. vigilax	NT	Darwin region	LOW	LOW	LOW	MED	HIGH	HIGH	LOW	LOW	LOW	LOW	HIGH	MED
Ae. vigilax	NT	East Arnhem region	LOW	LOW	LOW	LOW	LOW	–	–	VERY HIGH	HIGH	HIGH	LOW	LOW
Ae. vigilax	Qld	Brisbane inland–Indooroopilly Island	–	LOW	LOW	LOW	LOW	MED	HIGH	HIGH	HIGH	LOW	LOW	–
Ae. vigilax	Qld	Brisbane coastal–Bracken Ridge	LOW	LOW	LOW	LOW	LOW	HIGH	VERY HIGH	VERY HIGH	HIGH	HIGH	LOW	LOW
Ae. vigilax	Qld	Brisbane coastal–Virginia	LOW	LOW	LOW	LOW	LOW	VERY HIGH	VERY HIGH	VERY HIGH	VERY HIGH	HIGH	LOW	LOW
Ae. vigilax	Qld	Brisbane coastal–Albion	–	–	–	–	LOW	LOW	HIGH	HIGH	MED	HIGH	LOW	–
Ae. vigilax	Qld	Brisbane coastal–Hemmant	LOW	LOW	LOW	–	LOW	HIGH	VERY HIGH	HIGH	HIGH	HIGH	LOW	LOW
Ae. vigilax	Qld	Brisbane coastal–Lota	–	–	–	LOW	LOW	LOW	HIGH	MED	HIGH	HIGH	LOW	LOW
Ae. camptorhynchus *Ae. vigilax*	SA	St. Kilda	–	–	LOW	MED	MED	MED	LOW	LOW	MED	LOW	–	–
Ae. camptorhynchus *Ae. vigilax*	SA	Globe Derby Park	–	–	HIGH	HIGH	LOW	LOW	LOW	MED	HIGH	LOW	–	–
Culex molestus	SA	Goolwa	–	–	LOW	LOW	LOW	LOW	LOW	LOW	LOW	LOW	–	–

Table 7 *continued*: Key mosquito vector abundance in selected regions, Australia, 2012–13, by species, state or territory, region and month

Species	State or territory	Region or locality	Month											
			July	Aug	Sept	Oct	Nov	Dec	Jan	Feb	Mar	Apr	May	Jun
Ae. camptorhynchus	Vic.	Gippsland / Lake Wellington	–	–	–	–	HIGH	VERY HIGH	HIGH	HIGH	MED	VERY HIGH	–	–
Ae. vigilax	WA	Broome region	–	–	–	–	–	–	–	–	HIGH	–	–	–
Ae. vigilax	WA	Derby/Willare region	–	–	–	–	–	–	–	–	HIGH	–	–	–
Ae. camptorhynchus	WA	Peel region	HIGH	HIGH	HIGH	HIGH	HIGH	HIGH	MED	LOW	LOW	LOW	MED	HIGH
Ae. vigilax	WA	Peel region	NIL	NIL	NIL	LOW	HIGH	HIGH	HIGH	HIGH	HIGH	MED	LOW	LOW
Ae. camptorhynchus	WA	Leschenault region	HIGH	HIGH	HIGH	HIGH	MED	HIGH	MED	LOW	MED	LOW	LOW	HIGH
Ae. vigilax	WA	Leschenault region	NIL	NIL	NIL	LOW	MED	MED	HIGH	HIGH	MED	LOW	LOW	NONE
Ae. camptorhynchus	WA	Capel-Busselton region	HIGH	HIGH	HIGH	HIGH	HIGH	HIGH	HIGH	LOW	LOW	LOW	LOW	HIGH
Freshwater														
Culex annulirostris	NSW	Inland – Riverina	–	–	–	–	HIGH	HIGH	VERY HIGH	VERY HIGH	HIGH	LOW	–	–
Cx. annulirostris	NSW	Inland – Murray region	–	–	–	–	LOW	LOW	LOW	LOW	LOW	LOW	–	–
Cx. annulirostris	NSW	Inland, West and North West	–	–	–	–	LOW	LOW	LOW	LOW	LOW	LOW	–	–
Cx. annulirostris	NT	Darwin region	MED	MED	LOW	LOW	LOW	LOW	LOW	MED	LOW	MED	HIGH	HIGH
Cx. annulirostris	NT	East Arnhem region	LOW	LOW	MED	MED	LOW	–	–	MED	HIGH	HIGH	HIGH	LOW
Cx. annulirostris	NT	Katherine region	–	–	–	–	LOW	LOW	LOW	LOW	–	–	–	–
Cx. annulirostris	NT	Barkly region	–	–	–	NIL	NIL	NIL	LOW	LOW	LOW	LOW	LOW	NIL
Cx. annulirostris	NT	Alice Springs region	LOW	LOW	LOW	LOW	LOW	LOW	LOW	LOW	LOW	LOW	LOW	LOW
	Qld	Brisbane inland–Oxley	LOW	LOW	LOW	LOW	LOW	LOW	LOW	HIGH	MED	MED	LOW	LOW
	Qld	Brisbane inland–Indooroopilly Island	–	LOW	LOW	LOW	LOW	LOW	LOW	LOW	LOW	MED	LOW	LOW
	Qld	Brisbane inland–The Gap	–	LOW	LOW	LOW	LOW	LOW	LOW	MED	HIGH	MED	LOW	LOW
Ae. camptorhynchus	SA	Wellington	–	–	HIGH	HIGH	HIGH	MED	LOW	LOW	LOW	LOW	–	–
Ae. camptorhynchus	SA	Tailem Bend	–	–	LOW	LOW	LOW	MED	LOW	LOW	LOW	LOW	–	–

Table 7 *continued*: Key mosquito vector abundance in selected regions, Australia, 2012–13, by species, state or territory, region and month

Species	State or territory	Region or locality	Month											
			July	Aug	Sept	Oct	Nov	Dec	Jan	Feb	Mar	Apr	May	Jun
Ae. camptorhynchus	SA	Murray Bridge	–	–	HIGH	LOW	LOW	LOW	LOW	LOW	LOW	LOW	–	–
Ae. camptorhynchus	SA	Mannum	–	–	HIGH	LOW	LOW	LOW	LOW	LOW	LOW	LOW	–	–
Ae. camptorhynchus	SA	Meningie	–	–	HIGH	LOW	LOW	LOW	LOW	LOW	LOW	MED	–	–
Culex molestus	SA	Swan Reach	–	–	LOW	LOW	LOW	LOW	LOW	MED	LOW	HIGH	–	–
Cx. annulirostris	SA	Blanchetown	–	–	LOW	LOW	LOW	LOW	LOW	LOW	LOW	LOW	–	–
Anopheles annulipes *Cx. annulirostris*	SA	Morgan	–	–	LOW	LOW	LOW	LOW	LOW	–	LOW	LOW	–	–
Cx. molestus	SA	Waikerie	–	–	LOW	LOW	LOW	LOW	MED	LOW	LOW	LOW	–	–
Cx. annulirostris	SA	Kingston on Murray	–	–	LOW	LOW	–	–	LOW	LOW	LOW	LOW	–	–
An. annulipes *Cx. annulirostris*	SA	Loxton	–	–	LOW	LOW	LOW	LOW	LOW	LOW	LOW	LOW	–	–
An. annulipes *Cx. annulirostris* *Cx. quinquefasciatus*	SA	Berri	–	–	MED	HIGH	LOW	LOW	LOW	LOW	LOW	LOW	–	–
An. annulipes *Cx. annulirostris*	SA	Renmark/Paringa	–	–	LOW	LOW	LOW	LOW	LOW	MED	LOW	LOW	–	–
Ae. camptorhynchus	SA	Wellington	–	–	HIGH	HIGH	HIGH	MED	LOW	LOW	LOW	LOW	–	–
Cx. annulirostris	Vic.	North West	–	–	–	–	LOW	MED	LOW	LOW	LOW	LOW		
Cx. annulirostris	Vic.	North East	–	–	–	–	LOW	LOW	LOW	LOW	MED	LOW	–	–
Cx. annulirostris	WA	Broome region	–	–	–	–	–	–	–	–	VERY HIGH	–	–	–
Aedes normanensis	WA	Broome region	–	–	–	–	–	–	–	–	LOW	–	–	–
Cx. annulirostris	WA	Derby/Willare region	–	–	–	–	–	–	–	–	MED	–	–	–
Ae. normanensis	WA	Derby/Willare region	–	–	–	–	–	–	–	–	HIGH	–	–	–

Calculated as an average for traps across the region and rated as:

LOW (<50)	MEDIUM (50–100),	HIGH (101–1,000)	VERY HIGH (1,001–10,000)	EXTREME (>10,000)

– No data because trapping not undertaken.

Table 8: Virus and sentinel chicken surveillance in selected regions, Australia, 2012–13, by surveillance method and virus genus

State or territory	Region	Flaviviruses			Alphaviruses		
		Number of positive or seroconverted/ number tested*	First positive date	Last positive date	Number positive or seroconverted/ number tested*	First positive date	Last positive date
Sentinel chickens							
NSW	Bourke	0/243			N/A		
NSW	Deniliquin	0/259			N/A		
NSW	Forbes	0/330			N/A		
NSW	Griffith	0/284			N/A		
NSW	Hay	0/315			N/A		
NSW	Leeton	0/299			N/A		
NSW	Macquarie Marshes	0/275			N/A		
NSW	Menindee	0/146			N/A		
NSW	Moama	0/132			N/A		
NSW	Moree	0/251			N/A		
NT	Darwin region	1/225 flavivirus unidentified	2 May 2013	2 May 2013	N/A		
NT	East Arnhem region	0/63			N/A		
NT	Katherine region	0/84			N/A		
NT	Barkly region	0/56			N/A		
NT	Alice Springs region	0/56			N/A		
SA	Paringa	0/15			N/A		
SA	Loxton	0/15			N/A		
SA	Waikerie (Qualco)	0/20			N/A		
SA	Murray Bridge	0/10			N/A		
SA	Meningie	0/5			N/A		
Vic.	Mooroopna	0/293			N/A		
Vic.	Mildura	0/315			N/A		
Vic.	Robinvale	0/238			N/A		
Vic.	Nyah West	0/358			N/A		
Vic.	Kerang	0/338			N/A		
Vic.	Barmah	0/315			N/A		
Vic.	Cobram	0/379			N/A		
Vic.	Wodonga	0/294			N/A		
Vic.	Rutherglen	0/382			N/A		

Table 8 *continued*: Virus and sentinel chicken surveillance in selected regions, Australia, 2012–13, by surveillance method and virus genus

State or territory	Region	Flaviviruses			Alphaviruses		
		Number of positive or seroconverted/ number tested*	First positive date	Last positive date	Number positive or seroconverted/ number tested*	First positive date	Last positive date
WA	Wyndham	0/24			N/A		
WA	Kununurra	1/30	17 Aug 2012	17 Aug 2012	N/A		
WA	Savannah Nickel mine	0/136			N/A		
WA	Halls Creek	0/180			N/A		
WA	Fitzroy Crossing	0/135			N/A		
WA	Derby	0/510			N/A		
WA	Lombadina	0/62			N/A		
WA	Beagle Bay	2/21	22 Jul 2012	30 Aug 2012	N/A		
WA	Broome	0/30			N/A		
WA	Roebuck Plains	2/127	9 Aug 2012	30 May 2013	N/A		
WA	Port Hedland	0/93			N/A		
WA	Karratha	0/270			N/A		
WA	Harding Dam	1/455	6 June 2013	6 June 2013	N/A		
WA	Marble Bar	0/77			N/A		
WA	Pannawonica	0/262			N/A		
WA	Tom Price	0/159			N/A		
WA	Paraburdoo	0/210			N/A		
WA	Onslow	0/163			N/A		
WA	Ophthalmia Dam	0/219			N/A		
WA	Newman	0/252			N/A		
WA	Exmouth	0/312			N/A		
WA	Carnarvon	0/206			N/A		
WA	Moora	0/102			N/A		
WA	Geraldton	0/160			N/A		
WA	Dongara	0/163			N/A		
WA	York	0/139			N/A		
WA	Leonora	0/11			N/A		

Table 8 *continued*: Virus and sentinel chicken surveillance in selected regions, Australia, 2012–13, by surveillance method and virus genus

State or territory	Region	Flaviviruses			Alphaviruses		
		Number of positive or seroconverted/ number tested*	First positive date	Last positive date	Number positive or seroconverted/ number tested*	First positive date	Last positive date
Flinders Technology Associates cards							
NSW	Leeton	1EHV/303 (Flinders Technology Associates cards not routinely used in 2012–13)					
NT	Darwin region	1/150 card – MVE	18 Apr 2013	18 Apr 2013	0/150		
Qld	Badu	NIL			1/8 cards RRV	18 Feb 2013	29 Feb 2013
Qld	Seisia	NIL			1/8 cards RRV	18 Feb 2013	31 May 2013
Qld	Rockhampton	NIL			2/7 cards RRV and BFV	5 May 2013	31 May 2013
Qld	Charleville	NIL			1/6 cards BFV	28 May 2013	1 May 2013
Qld	Longreach/ Emerald	NIL			4/6 cards RRV	21 May 2013	30 May 2013
Qld	Mareeba	NIL			1/6 cards RRV	19 April 2013	29 May 2013
Qld	Townsville	NIL			4/7 cards RRV and BFV	21 Feb 2013	21 May 2013
Virus isolation from mosquitoes							
NSW	Ballina	1 EHV, 1 STRV /9,801					
	Bankstown	2 EHV, 3 STRV /10,554					
	Blacktown	1 EHV, 4 STRV /3,892					
	Byron Bay	2 STRV /5,291			1 RRV/5,291		
	Georges River	2 EHV /12,914					
	Gosford	1 EHV/3,232					
	Hawkesbury	3 EHV, 2 STRV /2,299					
	Homebush	1 STRV/6,909					
	Lake Macquarie	2 STRV/1,622					
	Penrith	1 STRV/2,750					
	Port Macquarie	2 EHV/3,059					
	Wyong	1 EHV, 2 STRV/1,620					
NT	Darwin region	0/4,641			0/4,641		

Table 8 *continued*: Virus and sentinel chicken surveillance in selected regions, Australia, 2012–13, by surveillance method and virus genus

State or territory	Region	Flaviviruses			Alphaviruses		
		Number of positive or seroconverted/ number tested*	First positive date	Last positive date	Number positive or seroconverted/ number tested*	First positive date	Last positive date
Vic.	Inland North West	0/7,355			0/7,355		
Vic.	Inland North East	0/8,104			0/8,104		
Vic.	Gippsland – Lake Wellington	0/17,174			0/17,174		
Vic.	Melbourne	0/1,661			0/1,661		
Vic.	Geelong	0/4,040			0/4,040		
WA	Broome region	1 KOKV/10,979			10 RRV, 4 BFV/10,979		
WA	Derby/Willare region	1 KOKV/4,898			6 RRV, 4 BFV/4,898		
WA	Peel region	2 EHV/37,635			6 RRV, 1 BFV/37,635		
WA	Leschenault region	0/22,116			8 RRV, 2 BFV/22,116		
WA	Capel-Busselton region	0/13,399			9 BFV/13,399		

* The number tested is the number of individual mosquitoes or chickens tested, unless otherwise noted.

BFV Barmah Forest virus

EHV Edge Hill virus

KOKV Kokobera virus

RRV Ross River virus

STRV Stratford virus

Sentinel chickens are not screened for antibodies to alphaviruses.

Western Australia do not test all of the mosquitoes collected (sub-sample of to up to 500 per trap in northern Western Australia, and up to 350–500 depending on mosquito abundance in the south-west of Western Australia are tested). Therefore the number of mosquitoes shown as tested does not reflect the actual number of mosquitoes collected.

Queensland

The exotic Asian tiger mosquito, *Ae. albopictus* was found on the outer islands of Torres Strait in April 2005.[28] This mosquito is a competent vector for a number of arboviruses including DENVs and CHIKV, and represents a serious nuisance biting mosquito. Since 2005, the Australian Government has funded Queensland Health to conduct a mosquito elimination program in the Torres Strait. The initial aim of the program was to eliminate *Ae. albopictus* from the Torres Strait islands but this was revised in May 2008 to a cordon sanitaire approach (a barrier designed to prevent spread) focused on Thursday and Horn islands.

During the reporting period, surveillance and control activities were carried out on Thursday Island, Horn Island, and the Northern Peninsula Area (NPA), Cape York from July 2012 to May 2013. *Ae. albopictus* were detected in low numbers on several occasions on Thursday and Horn islands, but were not detected in the NPA.

The main focus of the program in 2012–13 was to suppress *Ae. albopictus* populations and possibly eliminate the species from Horn Island and Thursday Island, both regarded as the gateway to the mainland of Australia due to their strategic location and transport networks. Consistent monitoring of mosquito densities and distribution on the 2 islands showed up to a 60-fold decline in the number of adult *Ae. albopictus* after intensive intervention that included residual pyrethroid treatment of vegetated peri-domestic harbourage sites. The control operation also included repeated house-to-house yard inspections on at least 800 properties for removal or treatment of water-holding receptacles. At least 3,000 receptacles were inspected and treated on each field visit. The yard inspections also constituted part of the mosquito surveys and *Aedes* larval samples were collected from all positive receptacles for identification. A decline in the Breteau Index of up to 10-fold was recorded for *Ae. albopictus* on the 2 islands during the wet seasons of the reporting period, demonstrating a dramatic impact of the control program. Despite a reduction in container breeding sites as part of the Thursday Island control program, the numbers of *Ae. aegypti* remain relatively high, although some decline in population density was observed.

Murray Valley encephalitis virus surveillance trial

A trial of sugar-based virus surveillance conducted between February and May 2013 saw traps deployed at the following sites; Badu Island (Torres Strait), Bamaga and Seisia (Cape York), Mareeba and Townsville (north Qld), Rockhampton, Longreach and Emerald (central Qld), Charleville and St George (south west Qld). Sugar-baited FTA cards were collected and processed on 64 separate occasions over the trial period. Arboviruses were detected on 14 occasions: RRV on 11 occasions and BFV on 3 occasions. Neither MVEV nor KUNV were detected. The trial provided further evidence of the efficacy and efficiency of this system.

Container inhabiting mosquito surveillance

Ongoing weekly surveillance in Cairns via a network of traps across various suburbs did not detect *Ae. albopictus* during the reporting period. Surveillance for *Ae. aegypti* undertaken in Townsville using BG traps consistently detected *Ae. aegypti* across the season and city.

Ae. aegypti larvae were collected in May 2013 from Longreach and Alpha in the Central West, but repeated larval surveys in coastal Yeppoon throughout late 2012–early 2013 did not detect *Ae. aegypti*. Brief surveys were also conducted during July 2012 in Benaraby and Bororen with no *Ae. aegypti* detected in either location. Ovitraps and BG traps deployed in Biloela and Emerald between November 2012–May 2013 detected *Ae. aegypti* at both locations throughout the season. Ovitraps in Gladstone confirmed its presence in late 2012.

In the Bundaberg region, yard inspections conducted in April and May 2013 confirmed the presence of *Ae. aegypti* in the town of Gin Gin, but similar surveys did not detect *Ae. aegypti* in the towns of Wallaville, Sharon or South Kolan. Surveillance of 257 premises by local and state government in the North Burnett regional towns of Biggenden, Mt Perry, Monto, Eidsvold, Mundubbera and Gayndah detected *Ae. aegypti* in all towns except Mt Perry and Eidsvold (despite this species being detected in Eidsvold during surveys conducted in 2010–2011).

In January 2013, mosquito surveillance (house to house surveys) was conducted in South Burnett. Towns surveyed included Kingaroy (24 premises visited), Nanango (21 premises visited), Wondai (21 premises visited) and Murgon (18 premises visited). In total, 84 premises were surveyed and 157 samples were collected. BG traps were also placed in Kingaroy. *Ae. aegypti* was detected in Wondai and Murgon; this was the first time that *Ae. aegypti* has been detected in the South Burnett Regional Council region. In response, South Burnett Regional Council allocated additional funding towards the prevention of the spread of *Ae. aegypti* and engaged an independent consultant to assist with surveillance and control activities. Extensive survey and domestic treat-

ment of infested containers was conducted within Wondai and Murgon and Public Health Orders were issued, if required. The Council has also undertaken a number of community prevention campaigns, including media releases.

In the Brisbane region, property inspections were conducted in 75 commercial or Industrial premises across the Brisbane City area with no detections of *Ae. aegypti* or *Ae. albopictus*.

Fresh water and salt mash surveillance

In South-East Queensland, there was a dramatic contrast between the first half of the summer season and the second half. Rainfall from July to December was below average and most saltmarshes remained dry until the first major tide in mid-November. A king tide in mid-December saw the return of *Ae. vigilax*. From January to April, rainfall was above average, including the deluge and flooding associated with ex-tropical cyclone Oswald during late January. Both saltmarsh and freshwater species reacted accordingly, with some light traps in Brisbane collecting more than 2,000 *Ae. vigilax* per trap night in January, February and March. By mid-April, the saltmarshes were saturated and remained so wet that numbers of this species decreased; although freshwater *Culex annulirostris* and other species continued activity until May. The very high rainfall associated with Cyclone Oswald produced devastating flooding in the Burnett area and a number of councils in the south-east supported mosquito management efforts in Bundaberg and Mundubbera.

South Australia

Mosquito populations along the River Murray during the 2012–13 season north of Mannum in the Mid-Murray council were typified by a modest early season peak in *Cx. annulirostris* and *An. annulipes* in September and October, followed by another spike mid to late in the season during February to March. Mosquito catches (including peak catches) in the upper river (Renmark-Paringa, Loxton-Waikerie and Berri-Barmera Councils) were all well below average for the corresponding time of year, indicating a season of low mosquito abundance. Like the previous season, these areas also lacked any significant numbers of the spring mosquito *Ae. camptorhynchus* at any time of the season. In the northern Riverland councils, very small catches of some of the less common *Aedes* species were recorded, such as *Ae. eidsvoldensis, Ae. sagax and Ae. vittiger*.

In Adelaide's northern metropolitan areas of Globe Derby and St Kilda, *Ae. camptorhynchus* numbers were slightly higher than the previous season (2011–12) but still lower compared with the season of 2010–11. Adelaide experienced summer rainfall well below average, (receiving only 50% to 60% of the average rainfall). *Ae. vigilax* numbers in the late summer and autumn of 2013 continued to remain low, but numbers increased slightly towards the end of the season compared with the previous season. *Ae. vigilax* numbers peaked at approximately 68.2 per trap in mid-March 2013. This peak was possibly driven by high tides occurring in mid-March to April 2013.

Adelaide received 35.0 mm compared with the long-term average of 62.2 mm, making it the driest summer since 2009/2010. Both maximum and minimum temperatures were overall warmer than average throughout the summer.

The frequency of bleeds of the sentinel chickens varied from flock to flock this season due to varying set up dates. No seroconversions to MVEV or KUNV were recorded during the reporting period.

Tasmania

No viruses were isolated in 2012–13 from mosquitoes trapped during ad hoc collections undertaken in the Sorrell Council region.

Victoria

Through the standard sentinel chicken program, weekly blood samples were tested from the 9 flocks between November 2012 and March 2013. No seroconversions to flaviviruses were detected during the season, which involved testing of 3,122 samples.

Mosquito monitoring in Victoria was conducted through the Victorian Arbovirus Disease Control Program by 10 Local Government Areas. Across the standard mosquito monitoring program, 34,979 mosquitoes were collected between November and April and submitted for species identification and arbovirus detection. Mosquito abundance was low at all monitoring locations along the Murray River (except for Gannawarra) from November until February. High rainfall in February was associated with moderate to high numbers of mosquitoes through most of the monitoring sites along the Murray and inland rivers, except at Wodonga in the far east of the State. *Cx. annulirostris* was the dominant species at most inland sites, accounting for between 39% and 71% of collections. Other species that dominated catches included *Ae. notoscriptus*, *Anopheles annulipes*, *Coquillettidia linealis* and *Ae. bancroftianus*.

Coastal mosquito populations are monitored in the Gippsland and Bellarine Peninsula areas, with the Wellington Shire Council participating in the standardised mosquito monitoring program with weekly submissions. Mosquito abundance oscillated throughout the season, with high numbers *Ae. camptorhynchus* detected in Gippsland during all months from December through to March. Mosquito abundance peaked at the end of December 2012 and again at the end of April 2013. Mosquito abundance exceeded 4,000 mosquitoes per trap on multiple occasions at 1 site.

In the 2012–13 season, no arboviruses were isolated from the 38,334 mosquitoes processed for virus isolation.

Western Australia

Above average rainfall was observed in northern parts of Western Australia between October and December 2012. Between January and March 2013 conditions were average or drier than usual in the Kimberley region, with the exception of the West Kimberley. During this time the West Kimberley and parts of the east Pilbara experienced above average rainfall and in some parts, the highest on record rainfall. Tropical cyclones Narelle and Peta caused heavy rainfall in the western Gascoyne, Pilbara and northern Interior in January. Seasonal thunderstorms resulted in more rain in March. Between April and June above to very much above average rainfall was recorded in the Kimberley and most of the Pilbara regions, with highest on record rainfall being recorded in the East Pilbara and northern Interior. Above average rainfall continued into May and June 2013 in northern parts of Western Australia.

In the south-west of Western Australia, rainfall was generally below average, with the exception of November and December 2012, when rainfall was above average. Warm conditions prevailed for most of the year, and tides regularly inundated mosquito vector breeding saltmarsh during the warm spring and summer period.

A total of 4,508 serum samples from 28 sentinel chicken flocks were tested for antibodies to flaviviruses during 2012–13.[29] Seroconversions to flaviviruses were detected in just 6 (0.1%) samples. Seroconversions at Beagle Bay (1 MVEV) in July and Kununurra (1 MVEV), Beagle Bay (1 KUNV) and Roebuck Plains Station (1 flavivirus infection that was not due to infection with MVEV or KUNV) in August were associated with activity continuing from the 2011–12 season.[7,30] The first activity associated with the 2012–13 wet season occurred in late May 2013 when a KUNV seroconversion was detected in a sentinel chicken at Roebuck Plains Station. Shortly afterwards, 1 KUNV infection was detected in the Harding Dam flock in June. This was a very late start to the flavivirus season, and was also the lowest level of activity observed since 1995–96 when just 2 seroconversions to KUNV were detected in March–April 1996.[31] Predominantly, alphaviruses (RRV and BFV) were isolated from mosquitoes collected in the West Kimberley region of Western Australia (Table 8). This was probably because the timing of adult mosquito collections in that region was just a few weeks following heavy rainfall, when saltmarsh and floodwater mosquito populations were high (Table 7).

The first media release for northern Western Australia was issued by the Western Australian Department of Health on 28 March 2013. This was a general media release issued prior to the holiday Easter season, and warned residents and travellers to the north of Western Australia of the increased risk of mosquito-borne disease in northern Western Australia. A second media release was issued on 25 June 2013 in response to the detections of KUNV in sentinel chickens in the Kimberley and Pilbara regions and heavy unseasonal rain.

Vector abundance was high in the south-west of Western Australia, particularly *Ae. camptorhynchus* in winter and spring, and *Ae. vigilax* during summer in the Peel and Leschenault regions (Table 5).[29] The first arbovirus isolate for the season was BFV from *Ae. camptorhynchus* collected at Capel in September 2012, and a further 8 isolates of BFV were detected in Capel-Busselton through to December, prompting the Department of Health to issue a media release about the increased risk of vectorborne disease. The first arbovirus detections in the Peel and Leschenault regions were RRV and BFV respectively, both in early December 2012. In total, there were 14 RRV, 12 BFV and 2 Edge Hill virus (EHV) detections (Table 8) as well as 1 isolate that was not an alphavirus or flavivirus (identity yet to be determined). The majority of isolations were from *Ae. camptorhynchus* (18) and *Ae. vigilax* (10), with a single RRV isolate from *Cx. annulirostris*. In 2012, the infection rate for RRV peaked at 19.1 per 1,000 mosquitoes,[32] whilst the peak infection rate for BFV was 3.6 per 1,000 mosquitoes in November 2012. Further detail can be found in the Western Australian annual reports (http://ww2.health.wa.gov.au/~/media/Files/Corporate/general%20documents/Mosquitoes/PDF/Arbovirus-AnnRpt-2012-13.ashx)

Exotic mosquito detections at the border

Between July 2012 and June 2013 there were 7 exotic mosquito detections made by the Australian Government Department of Agriculture and

Water Resources at the Australian border (Table 9). This represented an increase from the 2011–12 period where there were 5 exotic mosquito detections. Two detections were via inspection of imported cargo and 5 detections resulted from routine vector monitoring activities performed at international ports. The detection of *Ae. albopictus* at a Post Entry Quarantine Facility in Melbourne in December 2012 highlights that imported consignments of Lucky Bamboo remain a significant risk for the introduction of exotic mosquitoes. No *Ae. albopictus* mosquitoes were detected beyond the infested quarantine glasshouses with surveillance still being maintained by Kingston City Council and the Victorian Department of Health. The detection of *Ae. aegypti* at Brisbane International Airport in December 2012 was the first detection of an exotic mosquito in a Department of Agriculture and Water Resources vector monitoring trap at an International Airport. DNA analysis of the mosquito larvae suggested a likely North Queensland origin (potentially via a domestic leg of an international flight) however, South Est Asia could not be ruled out as a possible origin (A Weeks, unpublished results). *Ae. aegypti* were detected on 3 occasions at Darwin Seaport in April / May 2013 with the second detection in May (*Ae. aegypti* larvae and pupae) suspected to have originated from the single adult female *Ae. aegypti* detected earlier in May. Enhanced surveillance in surrounding areas did not detect exotic mosquitoes further afar from the initial detection site. *Ae. aegypti* larvae were detected in a sentinel tyre trap located at the port of Mackay in May 2013. Despite the known presence of this species in the Mackay region, it was the first time it has been detected in a trap at the port. Mackay Regional Council conducted precautionary residual treatments in response to the detection.

Discussion

NAMAC contributes to a One-Health approach to the control of arboviral disease and malaria, by uniting experts from a range of fields to provide strategic advice on the epidemiology, surveillance and management of these diseases. This report describes the epidemiology of arboviral diseases and malaria for the season 1 July 2012 to 30 June 2013, activities undertaken by health authorities in response to human cases, and evidence of virus activity. Sentinel chicken and vector monitoring continue to be an important part of the early warning system for arboviruses in Australia.

In 2012–13, the number of notifications of BFV infection and the population rates increased markedly compared with the previous year, with an epidemic of false positive IgM diagnoses from October 2012 due to an IgM test kit that was later recalled, as reported previously.[7,33] In 2012–13, cases were younger and a higher proportion were female. Cases were also more numerous in metropolitan areas than in previous years.

On NAMAC's recommendation, the CDWG of the CDNA undertook a review of surveillance case definitions for BFV and RRV infection. Under the revised case definition, which has been endorsed by CDNA, a single IgM positive result no longer constitutes laboratory evidence for infection, and where a single result is IgM and IgG positive, it may be notified as a probable case. A confirmed case will require IgG seroconversion or a significant increase in IgG antibody level (e.g. 4-fold or greater rise in titre). There is currently no plan to undertake a retrospective revision of notifications to apply the new case definitions because there is insufficient information on the diagnosis method available in NNDSS. Therefore, the historical data prior to the upcoming change of case definition will continue to be considered unreliable. The new case definition was implemented on 1 January 2016.

The particularly wet conditions experienced in many locations on the east coast in early 2013, in combination with arrival of cyclone Oswald and widespread flooding, provided challenging conditions for mosquito management programs. Despite these conditions, notifications of RRV remained below the 5-year mean nation-wide.

The prevention of incursion of dengue vectors into densely populated areas of South-East Queensland where imported dengue cases are regularly notified, is a continuing priority in Queensland. Despite frequent outbreaks relating to transmission from imported cases, mosquito and infection control measures undertaken by public health authorities and by residents have ensured that dengue has not become endemic in north Queensland.

Continued vigilance and the involvement of all relevant sectors enable the rapid detection of and early response to the threat of arboviral disease and malaria in Australia. The expert advice provided by NAMAC to AHPPC, CDNA and health departments has a vital role in mitigating mosquito-borne disease threats. Into the future, NAMAC strives for a reduction in the number of arbovirus cases in Australia, a strengthened disease prediction capacity to allow planning for response, and to retain, build and disseminate expertise and knowledge pertaining to mosquito-borne diseases.

Table 9: Exotic mosquito detections at the border, Australia, 2012–13

Date	Species	Location	Method of detection	Source or origin	Action or mitigation	Surveillance results
Aug 2012	*Ae. albopictus* (larvae)	Townsville (Seaport)	Cargo Inspection	New oversize tyres from Papua New Guinea	Tyre treatment (chlorination, residual insecticide and fumigation) and increased trapping. Early intervention meant risk was low.	No further detections
Dec 2012	*Ae. albopictus* (larvae, pupae, adults)	Melbourne (Post Entry Quarantine Facility)	Cargo inspection	Lucky bamboo imported from China	Glasshouses fogged; sterilisation of water and grow out containers; destruction of plants; harbourage treatments; receptacle treatment surveys and increased trapping and surveillance. Audits of the on-arrival treatments for the infested consignments were also conducted.	No further detections
Dec 2012	*Ae. aegypti* (larvae, eggs)	Brisbane (International Airport)	Ovitrap	Unconfirmed	Ultra low volume fogging; residual harbourage treatment; receptacle treatment/ surveys; increased trapping	No further detections
Mar 2013	*Ae. albopictus* (1 adult)	Cairns (Seaport)	CO_2 baited BG trap	Unknown/unable to identify source.	Ultra low volume fogging; mosquito harbourage treatments; receptacle treatment/ surveys; increased trapping	No further detections
Apr 2013	*Ae. aegypti* (5 adults)	Darwin (Seaport)	CO_2 baited BG trap	Unknown. (North Queensland excluded based on genetic analysis)	Ultra low volume fogging; harbourage treatments; receptacle treatment surveys and increased trapping	Single adult *Ae. aegypti* detected in the same BG trap location 9 days later
May 2013	*Ae. aegypti* (1 adult)	Darwin (Seaport)	CO_2 baited BG trap	Unknown (North Queensland excluded based on genetic analysis)	Ultra low volume fogging; receptacle treatment/ surveys; increased trapping	*Ae. aegypti* larvae and pupae detected in a tyre trap 11 days later
May 2013	*Ae. aegypti* (larvae, pupae)	Darwin (Seaport)	Sentinel tyre trap	Unknown (North Queensland excluded based on genetic analysis)	Receptacle treatment/ surveys; increased trapping	No further detections

Appendix

Sentinel chicken, vector and climate surveillance programs in the states and territories

Australian Capital Territory

There were no vertebrate, vector and climate surveillance programs in the Australian Capital Territory.

New South Wales

Surveillance mechanisms include mosquito monitoring, virus isolation from mosquitoes and sentinel chicken surveillance. The New South Wales Arbovirus Surveillance and Vector Monitoring Program is funded and coordinated by the NSW Ministry of Health (NSW Health), and laboratory services are contracted to the Institute of Clinical Pathology and Medical Research, Pathology West at Westmead Hospital. Mosquito trapping occurs from mid-spring to mid-autumn (November to April), and mosquitoes are collected weekly for species identification and quantification, and processed for isolation of arboviruses. Data on the Southern Oscillation Index (SOI), rainfall and temperature obtained from the Bureau of Meteorology (BOM) are used by members of the program to predict mosquito-breeding capabilities and potential arboviral activity, while climatic data are used to predict MVEV outbreaks. Sentinel chickens are operated along with mosquito monitoring and isolation at inland locations of major population centres at risk of MVEV, while along the coast where MVEV does not occur, only mosquito monitoring and viral isolation are undertaken. FTA cards were not used routinely in 2012–13.

The 2012–13 season began on 29 October 2012 with the first bleed and ended on 30 April 2013 with the last. A total of 10 flocks each containing up to 15 Isa Brown pullets was deployed, with 1 flock each at Bourke, Deniliquin, Forbes, Griffith, Hay, Leeton, Macquarie Marshes, Menindee, Moama (near Mathoura), and Moree (Map).

The NSW Chicken Sentinel Program was approved by the Western Sydney Local Health Network Animal Ethics Committee. This approval requires that the chicken handlers undergo training to ensure the chickens are cared for appropriately and that blood sampling is conducted in a manner that minimises trauma to the chickens. The chickens are cared for and bled by local council staff and members of the public. Laboratory staff members are responsible for training the chicken handlers. A veterinarian (usually the Director of Animal Care at Westmead) must inspect all new flock locations prior to deployment to ensure animal housing is adequate. Existing flocks are inspected approximately every 2 years. The health of each flock is reported weekly, and is independently monitored by the Animal Ethics Committee via the Director of Animal Care. Full details of the bleeding method and laboratory testing regimen were detailed in the 2003–04 NSW Arbovirus Surveillance Program annual report.[34]

The results of chicken serology are disseminated via email to the relevant government groups as determined by NSW Health and are placed on the NSW Arbovirus Surveillance website. Confirmed positives are notified by telephone to NSW Health and CDNA.

Northern Territory

Sentinel chicken flocks in the Northern Territory are maintained, bled and analysed for MVEV and KUNV in a combined program between the Department of Health, the virology laboratories of the DPIF and volunteers.

Surveillance consists of monthly routine sentinel chicken surveillance during the high risk period for MVE, with flocks located in Leanyer (Darwin), Howard Springs, the Coastal Plains Research Station at Beatrice Hill (Darwin region), Katherine, Nhulunbuy, Nathan River, Tennant Creek and Alice Springs. When chickens from a flock show antibodies to MVEV during a prime risk period, a media warning is issued for the general region. These warnings advise Northern Territory residents and visitors of the need to take added precautions to avoid mosquito bites. In 2012–13, sentinel chickens were bled between November 2012 and June 2013.

In addition, *ad hoc* virus isolation from mosquitoes is carried out when MVEV or KUNV disease cases are reported. The NT Mosquito Borne Disease Control Program assists regional authorities with mosquito monitoring and provides some funding for direct mosquito control. In 2012–13, routine adult mosquito trapping consisted of 16 trapping sites throughout the Darwin urban area. In other Northern Territory regions, adult mosquito trapping is carried out in liaison with Environmental Health and mining companies, with 6 traps located in Nhulunbuy, 3 in Alyangula on Groote Eylandt, 4 in Katherine, 3 in Tennant Creek and 6 in Alice Springs. Climate information from the BOM is used in conjunction with sentinel chicken and vector surveillance. Rainfall patterns, daily rainfall records and rain threshold models are used to assist in predicting mosquito and virus activity.

Queensland

Queensland Health does not currently conduct state-wide surveillance for MVEV in vertebrate hosts, and does not maintain sentinel chicken flocks. However, Queensland Health recently undertook a second sugar-based arbovirus surveillance trial utilising passive box traps and FTA card technology. The trial evaluated the effectiveness of this system as a sustainable method for arbovirus surveillance in Queensland and was designed to ascertain the feasibility of field deployment, and determine the achievability of timely detection and reporting of virus activity. Passive box traps were deployed in 9 rural or remote locations between February and May 2013, comprising 64 total trap events. Traps were serviced by a combination of local government, public health unit and Australian Government Department of Agriculture staff, while laboratory analysis of FTA cards was performed at Queensland Health Forensic and Scientific Services using real-time TaqMan RT-PCR.[27] Cards were tested for the presence of MVEV, KUNV, RRV and BFV.

Mosquito monitoring using light traps is performed by some local councils, primarily for salt water and fresh water mosquitoes. Some councils and public health units perform surveillance for container inhabiting mosquitoes using various methods including larval surveys and trapping utilising ovitraps, BG traps and Gravid Aedes Traps in domestic and commercial premises as part of a joint Queensland Health and local government initiative. Cairns and Townsville Public Health Units conduct routine *Ae. aegypti* surveillance in urban locations. Cairns Public Health Unit undertakes the Commonwealth-funded *Ae. albopictus* prevention and control program in the Torres Strait and NPA in Cape York. The Technical Advisory Group continues to provide general strategic direction to the program and meets regularly to review progress.

South Australia

Across South Australia, mosquito management activities are conducted through a partnership between the South Australian Department of Health and Ageing (SA Health), the University of South Australia, and local government. The program is focused on the Riverland and Murraylands areas where arbovirus is endemic, and extends to a range of coastal areas in regional and metropolitan localities of the state. SA Health funds half of local government costs for mosquito surveillance and control on public land through the South Australian Mosquito Management Subsidy.

The Mosquitoes and Public Health Research Group at the University of South Australia provided mosquito surveillance and spot control services to 7 local governments along the Murray River in South Australia from September 2012 to April 2013, as well as to the City of Salisbury located in Adelaide's north west.

The establishment of South Australia's revised sentinel surveillance program was finalised during the 2012–13 mosquito season and consists of small dedicated sentinel flocks (5 chickens per flock) in Paringa, Loxton, Waikerie (Qualco), Murray Bridge and Meningie.

Tasmania

No state-wide systematic mosquito abundance, virus isolation or sentinel chicken surveillance activities are undertaken due to the relatively low risk of arbovirus transmission in the State. However, mosquito collections are undertaken in the Sorell Council region, (which includes mosquito breeding areas, is fairly populous, and is close to Hobart) during high risk periods over January to March, when tidal inundation floods salt marsh habitat, thereby leading to egg hatching and subsequent increased abundance of the main local vector, *Ae. camptorhynchus*. These are sent to Westmead Hospital for species identification and viral isolation.

Victoria

The Victorian Department of Health contracts the Victorian Department of Economic Development, Jobs, Transport and Resources (then Victorian Department of Primary Industries) to conduct sentinel chicken surveillance, mosquito species identification and arbovirus detection during the arbovirus season from November to April. The standard sentinel chicken monitoring program involves the weekly collection of blood samples from 20 chickens located at each of 9 sites in northern Victoria along the Murray River or in the surrounding region. This program has been in place in Victoria since the 1974 outbreak and acts as an early warning system for possible human infections with flaviviruses. Flocks are replaced annually. Seven councils undertake mosquito surveillance as part of the standard mosquito monitoring program, which involves the weekly trapping of mosquitoes at 4 sites within each area. Six councils are located along the Murray and Goulburn River and 1 is a coastal site in Gippsland. Collections are also received from 3 additional councils located on the Murray River, Bellarine Peninsula and Melbourne. Mosquitoes are sent in cold storage to the

Victorian Department of Economic Development, Jobs, Transport and Resources for identification, enumeration and virus isolation. The Victorian Arbovirus Taskforce examines the risk of outbreaks of MVEV using meteorological surveillance data such as the SOI and rainfall deciles, and Indian Ocean Dipole using the Forbes,[35] and Nicholls[36] and Bennett models, respectively.

Western Australia

The University of Western Australia Arbovirus Surveillance and Research Laboratory (ASRL) was funded in 2012–13 by the Western Australian Department of Health to coordinate the sentinel chicken program and mosquito surveillance and to provide confirmatory serological testing for other sentinel chicken programs in Australia as required.[37] The flavivirus sentinel chicken program in Western Australia was undertaken by the ASRL at The University of Western Australia, on behalf of the Western Australian Department of Health. The sentinel chicken surveillance program was approved by The University of Western Australia Animal Ethics Committee. Many state and local government authorities and community volunteers also took part in the program. Twenty-eight sentinel chicken flocks (of up to 12 chickens) were located at major towns and communities in the Kimberley, Pilbara, Gascoyne, Goldfields, Midwest and Wheatbelt regions of Western Australia (Map). Blood samples were collected from the chickens by environmental health officers or trained volunteers at fortnightly intervals during the peak MVEV risk season (December to June). At other times, monthly samples were collected unless prolonged flavivirus activity warranted continued fortnightly sampling. Samples were transported to ASRL where they were tested for antibodies to flaviviruses using an epitope blocking ELISA.[38] In addition, adult mosquitoes were collected from the West Kimberley region of northern Western Australia in March 2013. These mosquitoes were identified to species and processed for virus isolation to investigate vector species and virus infection rates. In the south-west of Western Australia, adult mosquitoes were collected by the ASRL at the University of Western Australia on a regular basis in the Peel, Leschenault and Capel-Busselton regions for surveillance of RRV and BFV. Full details of the 2012–13 season are available in the ASRL annual report.[37]

Arbovirus research and surveillance laboratories in Australia

Commonwealth Scientific and Industrial Research Organisation

CSIRO Australian Animal Health Laboratory
Private Bag 24 (5 Portarlington Road)
GEELONG VIC 3220
Telephone: +61 3 5227 5000

New South Wales

Institute of Clinical Pathology and Medical Research, Pathology West
Westmead Hospital
Locked Bag 9001
WESTMEAD NSW 2145
Telephone: +61 2 9845 7279

Northern Territory

Northern Territory Department of Primary Industries and Fisheries
Makagon Road,
BERRIMAH NT 0828
Telephone: +61 8 8999 9251

Queensland

Queensland Health Forensic and Scientific Services
39 Kessells Road
Coopers Plains
PO Box 594
ARCHERFIELD QLD 4108
Telephone: +61 7 3274 9151

Victoria

Victorian Infectious Diseases Reference Laboratory (Human)
10 Wrecklyn Street
NORTH MELBOURNE VIC 3051
Telephone: (03) 9342 2600

Victorian Department of Economic Development, Jobs, Transport & Resources (then Victorian Department of Primary Industries)
AgriBio, The Centre for AgriBioscience
5 Ring Road, Bundoora
BUNDOORA VIC 3083
Telephone: +61 3 9032 7515

Western Australia

PathWest Laboratory Medicine WA
Division of Microbiology and Infectious Diseases (Human)
Hospital Avenue
NEDLANDS WA 6009
Telephone: +61 8 9346 3122

Arbovirus Surveillance and Research Laboratory
Discipline of Microbiology and Immunology (animal/vector)
School of Pathology and Laboratory Medicine
The University of Western Australia
35 Stirling Highway
CRAWLEY WA 6009
Telephone: +61 8 9346 2212

Acknowledgements

NAMAC members during 2012–13 were (in alphabetical order): Bart Currie, Peter Daniels, Stephen Doggett, Debra El Saadi, Rebecca Feldman, Jenny Firman, Katrina Knope, Ann Koehler, Rogan Lee, Mike Lindsay, John Mackenzie, Mike Muller, Scott Ritchie, Richard Russell, Angus Sly, David Smith, Peter Whelan, Craig Williams. Jennifer Wall and Phil Wright (Secretariat).

The data on which this report is based is the work of many people. We thank public health laboratories, state and territory communicable disease control units and public health units and staff in state and territory arbovirus surveillance and monitoring programs. We thank the state health departments for surveillance program funding. We thank Cassie Jansen, Odwell Muzari and Kerryn Lodo from Queensland Health.

Author details

Katrina E Knope[1]
Nina Kurucz[2]
Stephen L Doggett[3]
Mike Muller[4]
Cheryl A Johansen[5]
Rebecca Feldman[6]
Michaela Hobby[7]
Sonya Bennett[8]
Angus Sly[9]
Stacey Lynch[10]
Bart J Currie[11]
Jay Nicholson[5]
The National Arbovirus and Malaria Advisory Committee (see acknowledgement).

1. Zoonoses, Foodborne and Emerging Infectious Diseases Section, Health Protection Policy Branch, Office of Health Protection, Department of Health, Canberra, Australian Capital Territory
2. Medical Entomology, Centre for Disease Control, Health Protection Division, Northern Territory Department of Health, Royal Darwin Hospital, Casuarina, Northern Territory
3. Department of Medical Entomology, Pathology West, Institute for Clinical Pathology and Medical Research, Westmead Hospital, Westmead, New South Wales
4. Medical Entomologist, Brisbane City Council, Fortitude Valley, Queensland
5. Arbovirus Surveillance and Research Laboratory, School of Pathology and Laboratory Medicine, Faculty of Medicine, Dentistry and Health Sciences, The University of Western Australia, Nedlands, Western Australia. As of July 2015: Division of Microbiology and Infectious Diseases, PathWest Laboratory Medicine WA, QEII Medical Centre, Western Australian Department of Health, Nedlands, Western Australia.
6. Communicable Disease Prevention and Control, Department of Health, Melbourne, Victoria
7. Health Protection, Public Health, South Australian Department of Health, Adelaide, South Australia
8. Communicable Diseases Unit, Queensland Health, Herston, Queensland
9. Operational Science Section, Department of Agriculture and Water Resources, Compliance Division, Eagle Farm, Queensland
10. Victorian Department of Economic Development, Jobs, Transport and Resources, AgriBio Centre, Victoria
11. Royal Darwin Hospital Northern Territory; Menzies School of Health Research, Darwin, Northern Territory

Corresponding author: Ms Katrina Knope, Zoonoses, Foodborne and Emerging Infectious Diseases Section, Health Emergency Management Branch, Office of Health Protection, Australian Government Department of Health, MDP 14, GPO Box 9848, CANBERRA ACT 2601. Telephone: +61 2 6289 2751. Email: Katrina.Knope@health.gov.au

References

1. NNDSS Annual Report Writing Group. Australia's notifiable disease status, 2012: Annual report of the National Notifiable Diseases Surveillance System. *Commun Dis Intell* 2015;39(1):E46–E136.
2. Australian Bureau of Statistics. 3101.0 Australian Demographic Statistics, June 2013. Available from: http://www.abs.gov.au/AUSSTATS/abs@.nsf/DetailsPage/3101.0Dec%202014?OpenDocument
3. Broom AK, Azuolas J, Hueston L, Mackenzie JS, Melville L, Smith DW, et al. Australian encephalitis: Sentinel Chicken Surveillance Programme. *Commun Dis Intell* 2001;25(3):157–160.
4. Broom AK. Sentinel Chicken Surveillance Program in Australia, July 2002 to June 2003. *Commun Dis Intell* 2003;27(3):367–369.
5. Mackenzie JS, Lindsay MD, Coelen RJ, Broom AK, Hall RA, Smith DW. Arboviruses causing human disease in the Australasian zoogeographic region. *Arch Virol* 1994;136(3–4):447–467.
6. Russell RC, Dwyer DE. Arboviruses associated with human disease in Australia. *Microbes Infect* 2000;2(14):1693–1704.
7. Knope K, Doggett S, Kurucz N, Feldman R, Johansen C, Nicholson J, et al. Arboviral diseases and malaria in Australia, 2011–12: annual report of the National Arbovirus and Malaria Advisory Committee. *Commun Dis Intell* 2014;38(2):E122–E142.
8. Selvey LA, Donnelly JA, Lindsay MD, Pottumarthy Boddu S, D'Abrera VC, Smith DW. Ross River Virus infection surveillance in the Greater Perth Metropolitan Area – has there been an increase in cases in the winter months? *Commun Dis Intell* 2014;38(2).

9. Parida MM, Santhosh SR, Dash PK, Lakshmana Rao PV. Rapid and real-time assays for detection and quantification of chikungunya virus. *Future Virol* 2008;3(2):179–192.
10. Harrington S, Lindsay M, Douglas A. Christmas Island and Cocos (Keeling) Islands, Indian Ocean: Mosquito fauna and mosquito-borne disease risk assessment and management recommendations. Final report of investigations undertaken in 2007–08: Public Health Division, Western Australian Department of Health; 2009.
11. Hall-Mendelin S, Ritchie SA, Johansen CA, Zborowski P, Cortis G, Dandridge S, et al. Exploiting mosquito sugar feeding to detect mosquito-borne pathogens. *Proc Natl Acad Sci U S A* 2010;107(25):11255–11259.
12. Jansen CC, Williams CR, van den Hurk AF. The usual suspects: Comparison of the relative roles of potential urban chikungunya virus vectors in Australia. *PLoS One* 2015;10(8):e0134975.
13. Cashman P, Hueston L, Durrheim D, Massey P, Doggett S, Russell RC. Barmah Forest virus serology; implications for diagnosis and public health action. *Commun Dis Intell* 2008;32(2):263–266.
14. Rich G MJ, McPhan I, Richards B. Laboratory diagnosis of Ross River virus infection. *Commun Dis Intell* 1993;17:208–209.
15. Horwood PF, Reimer LJ, Dagina R, Susapu M, Bande G, Katusele M, et al. Outbreak of chikungunya virus infection, Vanimo, Papua New Guinea. *Emerg Infect Dis* 2013;19(9):1535–1538.
16. Roth A, Hoy D, Horwood PF, Ropa B, Hancock T, Guillaumot L, et al. Preparedness for threat of chikungunya in the Pacific. *Emerg Infect Dis* 2014;20(8) doi: 10.3201/eid2008.
17. Hanna JN, Ritchie SA, Richards AR, Humphreys JL, Montgomery BL, Ehlers GJ, et al. Dengue in north Queensland, 2005–2008. *Commun Dis Intell* 2009;33(2):198–203.
18. Queensland Health. *Queensland Dengue Management Plan 2010–2015, 2011*. Brisbane: Queensland Health.
19. Australian Technical Advisory Group on Immunisation. *The Australian Immunisation Handbook* 10th edn. Canberra, Australia: Department of Health; 2013.
20. Hanna JN, Ritchie SA, Phillips DA, Lee JM, Hills SL, van den Hurk AF, et al. Japanese encephalitis in north Queensland, Australia, 1998. *Med J Aust* 1999;170(11):533–536.
21. Johansen CA, Nisbet DJ, Foley PN, Van Den Hurk AF, Hall RA, Mackenzie JS, et al. Flavivirus isolations from mosquitoes collected from Saibai Island in the Torres Strait, Australia, during an incursion of Japanese encephalitis virus. *Med Vet Entomol* 2004;18(3):281–287.
22. Gray TJ, Burrow JN, Markey PG, Whelan PI, Jackson J, Smith DW, et al. West Nile virus (Kunjin subtype) disease in the Northern Territory of Australia—A case of encephalitis and review of all reported cases. *Am J Trop Med Hyg* 2011;85(5):952–956.
23. Heymann DL. *Control of Communicable Diseases Manual*. 19 edn: American Public Health Association; 2008.
24. Cox-Singh J, Davis TM, Lee KS, Shamsul SS, Matusop A, Ratnam S, et al. *Plasmodium knowlesi* malaria in humans is widely distributed and potentially life threatening. *Clin Infect Dis* 2008;46(2):165–171.
25. Gray TJ, Trauer JM, Fairley M, Krause VL, Markey PG. Imported malaria in the Northern Territory, Australia—428 consecutive cases. *Commun Dis Intell* 2012;36(1):107–113.
26. Hanna JN, Ritchie SA, Eisen DP, Cooper RD, Brookes DL, Montgomery BL. An outbreak of *Plasmodium vivax* malaria in Far North Queensland, 2002. *Med J Aust* 2004;180(1):24–28.
27. van den Hurk AF, Hall-Mendelin S, Townsend M, Kurucz N, Edwards J, Ehlers G, et al. Applications of a sugar-based surveillance system to track arboviruses in wild mosquito populations. *Vector Borne Zoonotic Dis* 2014;14(1):66–73.
28. Ritchie SA, Moore P, Carruthers M, Williams C, Montgomery B, Foley P, et al. Discovery of a widespread infestation of *Aedes albopictus* in the Torres Strait, Australia. *J Am Mosq Control Assoc* 2006;22(3):358–365.
29. Johansen C, Nicholson J, Power S, Wong S, Burley M, Wallace M, et al. The University of Western Australia Arbovirus Surveillance and Research Laboratory annual report: 2012–13. 2013.
30. Nicholson J, Power S, Wong S, Wallace M, Burley M, Cashen C, et al. The University of Western Australia Arbovirus Surveillance and Research Laboratory annual report: 2011–2012. The University of Western Australia. 2012.
31. Broom A, Lindsay M, Van Heuzen B, Wright T, Mackenzie J, Smith D, et al. Contrasting patterns of flavivirus activity in the Kimberley region of Western Australia, 1992–1996. *Arbovirus Research in Australia* 1997;7(Article):25–30.
32. Chiang CL, Reeves WC. Statistical estimation of virus infection rates in mosquito vector populations. *Am J Hyg* 1962;75:377–391.
33. Therapeutic Goods Administration. Product recall, Panbio Barmah Forest Virus IgM ELISA. An *in vitro* diagnostic medical device (IVD). Recall no. RC-2013-RN-00967-1,13/09/2013. 2013. Accessed on 6 May 2014. Available from: http://www.tga.gov.au/SARA/arn-detail.aspx?k=RC-2013-RN-00967-1
34. Doggett S, Clancy J, Haniotis J, Russell RC, Hueston L, Marchetti M, et al. The New South Wales Arbovirus Surveillance and Mosquito Monitoring Program. 2003–2004 annual report. Department of Medical Entomology, Westmead; 2004.
35. Forbes JA. Murray Valley encephalitis 1974. also The epidemic variance since 1914 and predisposing rainfall patterns. Sydney; 1978.
36. Nicholls N. A method for predicting Murray Valley encephalitis in south-east Australia using the Southern Oscillation. *Aust J Exp Bioi Mod Sci* 1986;64:587–594.
37. Nicholson J, Power S, Wong, S, Burley M, Wallace M, Smith D, Shellem G. The University of Western Australia Arbovirus Surveillance and Research Laboratory annual report: 2012–2013: The University of Western Australia. 2013.
38. Hall RA, Broom AK, Hartnett AC, Howard MJ, Mackenzie JS. Immunodominant epitopes on the NS1 protein of MVE and KUN viruses serve as targets for a blocking ELISA to detect virus-specific antibodies in sentinel animal serum. *J Virol Methods* 1995;51(2–3):201–210.

Australia's notifiable disease status, 2014: Annual report of the National Notifiable Diseases Surveillance System

NNDSS Annual Report Working Group

Abstract

In 2014, 69 diseases and conditions were nationally notifiable in Australia. States and territories reported a total of 275,581 notifications of communicable diseases to the National Notifiable Diseases Surveillance System, an increase of 22% on the number of notifications in 2013. In 2014, the most frequently notified diseases were sexually transmissible infections (105,719 notifications, 38% of total notifications), vaccine preventable diseases (101,400 notifications, 37% of total notifications), and gastrointestinal diseases (40,367 notifications, 15% of total notifications). There were 17,411 notifications of bloodborne diseases; 8,125 notifications of vectorborne diseases; 1,942 notifications of other bacterial infections; 615 notifications of zoonoses and 2 notifications of quarantinable diseases. *Commun Dis Intell* 2016;40(1):E48–E145.

Keywords: Australia, communicable diseases, epidemiology, surveillance

Introduction

Australia's notifiable diseases status, 2014, is an annual surveillance report of nationally notifiable communicable diseases. Communicable disease surveillance in Australia operates at the national, jurisdictional and local levels. Primary responsibility for public health action lies with the state and territory health departments. The role of communicable disease surveillance at the national level includes:

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

Methods

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

State and territory health departments collect notifications of communicable diseases under their respective public health legislations. In September 2007, the *National Health Security Act 2007*[1] received royal assent. This Act provides a legislative basis for and authorises the exchange of health information, including personal information, between jurisdictions and the Australian Government. The Act provides for the establishment of the National Notifiable Diseases List,[2] which specifies the diseases about which personal information can be provided. The *National Health Security Agreement*,[3] which was signed by Health Ministers in April 2008, establishes the operational arrangements to formalise and enhance existing surveillance and reporting systems, an important objective of the Act. Under the Agreement, in 2014 states and territories forwarded de-identified notification data on 65 communicable diseases to the Australian Government Department of Health for the purposes of national communicable disease surveillance, although not all 65 diseases were notifiable in each jurisdiction. Data were electronically updated daily from states and territories. The system was complemented by other surveillance systems, which provided information on various diseases, including 4 that are not reported to the National Notifiable Diseases Surveillance System (NNDSS): human immunodeficiency virus (HIV), acquired immune deficiency syndrome (AIDS) and the classical and variant forms of Creutzfeldt-Jakob disease (CJD).

The NNDSS core dataset requires the following mandatory data fields: unique record reference number; notifying state or territory; disease code; confirmation status and the date when the jurisdictional health department was notified (notification received date). In addition, the following data fields were supplied where available: date of birth; age at onset; sex; Indigenous status; postcode of residence; disease onset date; date

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

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

Newly diagnosed HIV infection and AIDS were notifiable conditions in each state or territory health jurisdiction in 2014. These data were forwarded to the Kirby Institute for Infection and Immunity in Society (Kirby Institute). Further information can be found in the Kirby Institute's annual surveillance report.[4]

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

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

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

Notification rates for each notifiable disease were calculated using the estimated 2014 December resident population supplied by the Australian Bureau of Statistics (ABS) (Appendix 1 and Appendix 2).[7] Where diseases were not notifiable in a state or territory, national rates were adjusted by excluding the population of that jurisdiction from the denominator. For some diseases, age adjusted rates were calculated using the direct method of standardisation with 2011 census data as the standard population. All rates are represented as the rate per 100,000 population unless stated otherwise.

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

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

Notes on interpretation

This report is based on 2014 data from each state and territory, agreed upon in June 2015, and represents a snapshot of the year after duplicate records and incorrect or incomplete data were removed.

Totals in this report may vary slightly from the totals reported in CDI quarterly publications and state and territory reports.

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

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

A survey of jurisdictional public health departments was conducted in 2014 to ascertain the source of each notification (Table 1). Whilst most jurisdictions have data on laboratory notifications, the percentage of notifications attributed to doctor only and laboratory and doctor for each state and territory are based on estimates deduced from the data that are available, noting that fields for these data may be incomplete.

Methods of surveillance vary between states and territories, each having different requirements for notification by medical practitioners, laboratories and hospitals. Although the National Notifiable Diseases List[2] was established, some diseases are not notifiable in all 8 jurisdictions (Table 2).

Table 1: Percentage of notified cases from different sources in each jurisdiction, 2014*

State or territory	Source of notifications		
	Laboratory only	Doctor only	Laboratory and doctor
ACT	95	<1	4
NSW	99	<1	<1
NT	98	1	1
Qld	100	<1	<1
SA	6	2	92
Tas.	100	<1	<1
Vic.	64	7	29
WA	35	1	64

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

Figure 1: Communicable diseases notifiable fraction

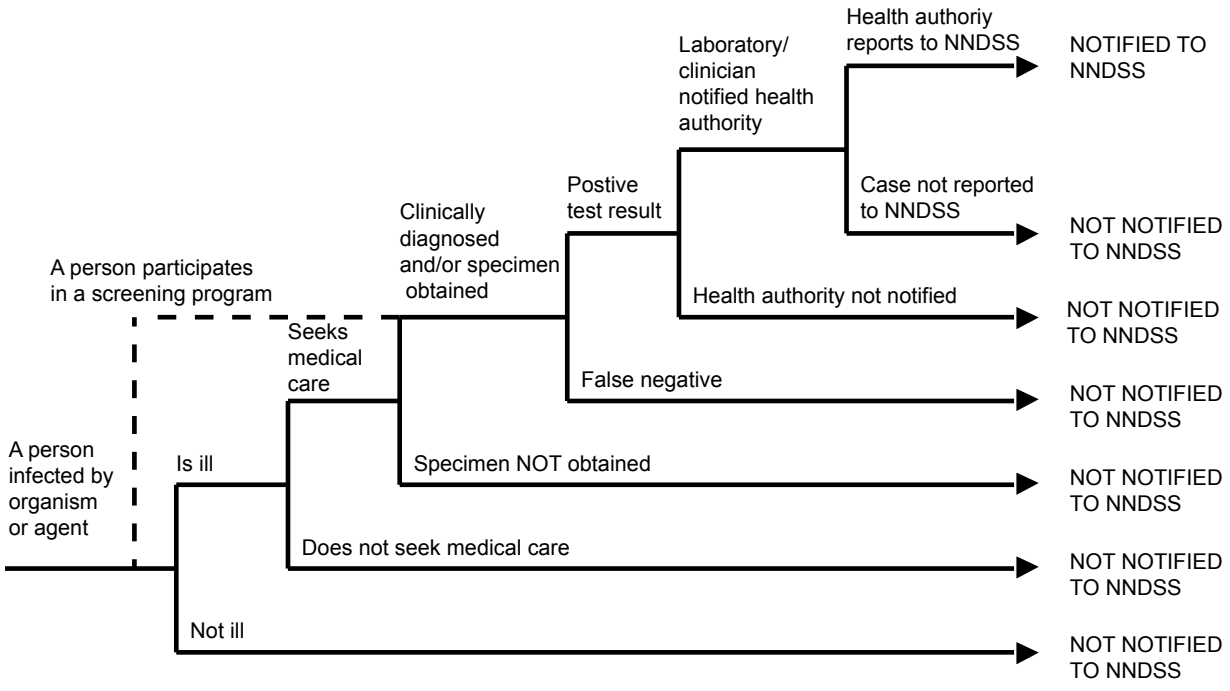


Table 2: Diseases notified to the National Notifiable Diseases Surveillance System, Australia 2014

Disease	Data received from
Bloodborne diseases	
Hepatitis B (newly acquired)	All jurisdictions
Hepatitis B (unspecified)	All jurisdictions
Hepatitis C (newly acquired)	All jurisdictions, except Queensland
Hepatitis C (unspecified)	All jurisdictions
Hepatitis D	All jurisdictions
Gastrointestinal diseases	
Botulism	All jurisdictions
Campylobacteriosis	All jurisdictions, except New South Wales
Cryptosporidiosis	All jurisdictions
Haemolytic uraemic syndrome	All jurisdictions
Hepatitis A	All jurisdictions
Hepatitis E	All jurisdictions
Listeriosis	All jurisdictions
Salmonellosis	All jurisdictions
Shigellosis	All jurisdictions
Shiga toxin producing *Escherichia coli*	All jurisdictions
Typhoid fever	All jurisdictions
Quarantinable diseases	
Cholera	All jurisdictions
Highly pathogenic avian influenza in humans	All jurisdictions
Plague	All jurisdictions
Rabies	All jurisdictions
Severe acute respiratory syndrome	All jurisdictions
Smallpox	All jurisdictions
Viral haemorrhagic fever	All jurisdictions
Yellow fever	All jurisdictions
Sexually transmissible infections	
Chlamydial infections	All jurisdictions
Donovanosis	All jurisdictions
Gonococcal infection	All jurisdictions
Syphilis < 2 years duration	All jurisdictions
Syphilis > 2 years or unspecified duration	All jurisdictions
Syphilis – congenital	All jurisdictions
Vaccine preventable diseases	
Diphtheria	All jurisdictions
Haemophilus influenzae type b	All jurisdictions
Influenza (laboratory confirmed)	All jurisdictions
Measles	All jurisdictions
Mumps	All jurisdictions
Pertussis	All jurisdictions
Pneumococcal disease (invasive)	All jurisdictions
Poliomyelitis	All jurisdictions
Rubella	All jurisdictions
Rubella – congenital	All jurisdictions
Tetanus	All jurisdictions

Table 2 *continued*: Diseases notified to the National Notifiable Diseases Surveillance System, Australia 2014

Disease	Data received from
Vaccine preventable diseases *cont'd*	
Varicella zoster (chickenpox)	All jurisdictions, except New South Wales
Varicella zoster (shingles)	All jurisdictions, except New South Wales
Varicella zoster (unspecified)	All jurisdictions, except New South Wales
Vectorborne diseases	
Arbovirus infection (NEC)	All jurisdictions
Barmah Forest virus infection	All jurisdictions
Dengue virus infection	All jurisdictions
Japanese encephalitis virus infection	All jurisdictions
Kunjin virus infection	All jurisdictions
Malaria	All jurisdictions
Murray Valley encephalitis virus infection	All jurisdictions
Ross River virus infection	All jurisdictions
Zoonoses	
Anthrax	All jurisdictions
Australian bat lyssavirus	All jurisdictions
Brucellosis	All jurisdictions
Leptospirosis	All jurisdictions
Lyssavirus (NEC)	All jurisdictions
Ornithosis	All jurisdictions
Q fever	All jurisdictions
Tularaemia	All jurisdictions
Other bacterial infections	
Legionellosis	All jurisdictions
Leprosy	All jurisdictions
Meningococcal disease (invasive)	All jurisdictions
Tuberculosis	All jurisdictions

NEC Not elsewhere classified.

Changes in surveillance practices may have been introduced in some jurisdictions and not in others, and must be taken into consideration when comparing data between jurisdictions. In this report, some additional information was obtained from states and territories to assist in the interpretation of the 2014 data. These include changes in surveillance practices, screening practices, laboratory practices, and major disease control or prevention initiatives.

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

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

The percentage of data completeness was defined as:

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

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

> 1=Indigenous – (Aboriginal but not Torres Strait Islander origin);
>
> 2=Indigenous – (Torres Strait Islander but not Aboriginal origin);
>
> 3=Indigenous – (Aboriginal and Torres Strait Islander origin);
>
> 4=Not Indigenous – (not Aboriginal or Torres Strait Islander origin);
>
> 9=Not stated.

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

Place of acquisition is where the disease is believed to have been acquired; either locally or overseas. The country of acquisition is determined by the Standard Australian Classification of Countries (SACC) from the ABS.[12] A notification is complete if a valid value from the SACC is entered.

For some diseases, changes in surveillance and testing practices should be taken into account when interpreting national trends.

In interpreting STI notification data it is important to note that changes in notifications over time may not solely reflect changes in disease prevalence as changes in screening programs,[13,14] the use of less invasive and more sensitive diagnostic tests[15] and periodic public awareness campaigns[16] may influence the number of notifications that occur over time. Rates for STIs are particularly susceptible to overall rates of testing, with low testing rates resulting in an underestimation of disease and increased testing potentially causing an increase in notifications.[17]

The differences in rates between females and males for STIs should be interpreted with caution, as rates of testing, symptom status, health care-seeking behaviours, and partner notification differ between the sexes.[18]

Notes on case definitions

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

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

Results

There were 275,581 communicable disease notifications received by NNDSS in 2014 (Table 3).

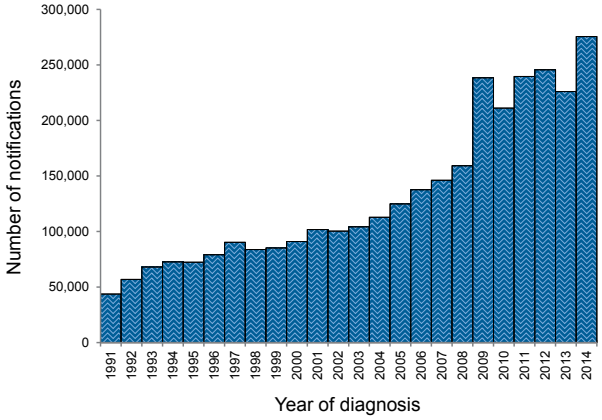
In 2014, the most frequently notified diseases were sexually transmissible infections (105,719 notifications, 38% of total notifications), vaccine preventable diseases (101,400 notifications, 37% of total notifications), and gastrointestinal diseases (40,367 notifications, 15% of total notifications).

Table 3: Notifications to the National Notifiable Diseases Surveillance System, Australia, 2014, by disease category rank order

Disease category	Number	%
Sexually transmitted infections	105,719	38
Vaccine preventable diseases	101,400	37
Gastrointestinal diseases	40,367	15
Bloodborne diseases	17,411	6
Vectorborne diseases	8,125	3
Other bacterial diseases	1,942	1
Zoonoses	615	0
Quarantinable diseases	2	0
Total	275,581	100

There was an increase of 22% compared with the total number of notifications in 2013 (226,041) (Figure 2). The increase can largely be attributed to the seasonal increase in influenza notifications for 2014, which reached a higher peak than in previous seasons.

Figure 2: Trends in notifications received by the National Notifiable Diseases Surveillance System, Australia, 1991 to 2014



Notifications and notification rates per 100,000 for each disease by state or territory in 2014, are shown in Table 4 and Table 5 respectively. Notifications and rates per 100,000 for the period 2009 to 2014 are shown in Table 6.

Table 4: Notified cases of communicable diseases, Australia, 2014, by state or territory

Disease	State or territory								
	ACT	NSW	NT	Qld	SA	Tas.	Vic.	WA	Aust.
Bloodborne diseases									
Hepatitis B (newly acquired)*	2	29	3	53	7	5	53	24	176
Hepatitis B (unspecified)†	95	2,514	150	988	323	55	1,741	628	6,494
Hepatitis C (newly acquired)*‡	11	26	2	NN	45	14	174	161	433
Hepatitis C (unspecified)†	164	3,555	178	2,648	449	217	2,048	990	10,249
Hepatitis D	0	19	1	13	9	0	14	3	59
Gastrointestinal diseases									
Botulism	0	0	0	1	0	0	0	0	1
Campylobacteriosis	505	NN	294	6,220	1,804	934	7,211	2,963	19,931
Cryptosporidiosis	30	418	87	668	225	30	637	310	2,405
Haemolytic uraemic syndrome	0	6	1	3	3	1	5	1	20
Hepatitis A	5	83	2	44	7	1	70	19	231
Hepatitis E	1	37	0	7	0	0	11	0	56
Listeriosis	1	23	2	17	6	4	22	5	80
Salmonellosis	225	4,314	457	4,937	1,220	249	3,695	1,261	16,358
Shiga toxin-producing *Escherichia coli*	0	30	0	28	45	0	10	2	115
Shigellosis	9	198	99	176	36	2	463	68	1,051
Typhoid fever	1	45	1	19	9	1	29	14	119
Quarantinable diseases									
Cholera	0	0	0	0	0	0	2	0	2
Highly pathogenic avian influenza in humans	0	0	0	0	0	0	0	0	0
Plague	0	0	0	0	0	0	0	0	0
Rabies	0	0	0	0	0	0	0	0	0
Severe acute respiratory syndrome	0	0	0	0	0	0	0	0	0
Smallpox	0	0	0	0	0	0	0	0	0
Viral haemorrhagic fever	0	0	0	0	0	0	0	0	0
Yellow fever	0	0	0	0	0	0	0	0	0
Sexually transmitted infections									
Chlamydial infection§‖	1,196	22,909	2,997	20,480	5,496	1,774	19,910	11,346	86,108
Donovanosis	0	0	0	0	0	0	0	1	1
Gonococcal infection‖	120	4,862	1,741	2,721	736	65	3,236	2,194	15,675
Syphilis – congenital‖	0	0	5	0	0	0	0	0	5
Syphilis < 2 years duration*‖¶	18	739	73	394	29	14	649	93	2,009
Syphilis > 2 years or unspecified duration†‖	26	536	73	279	123	19	801	64	1,921
Vaccine preventable diseases									
Diphtheria**	0	0	0	2	0	0	0	0	2
Haemophilus influenzae type b	0	6	1	9	1	0	3	1	21
Influenza (laboratory confirmed)	1,260	20,877	810	17,924	11,041	673	9,907	5,250	67,742
Measles	7	67	52	73	16	5	77	43	340
Mumps	2	79	1	49	19	5	12	23	190
Pertussis	233	3,131	83	1,392	503	68	4,702	1,751	11,863
Pneumococcal disease (invasive)	15	518	43	230	133	39	379	207	1,564
Poliomyelitis	0	0	0	0	0	0	0	0	0
Rubella	0	10	0	2	2	0	2	1	17
Rubella – congenital	0	0	0	0	0	0	0	0	0
Tetanus	0	1	0	1	0	0	0	1	3

Table 4 *continued*: Notified cases of communicable diseases, Australia, 2014, by state or territory

Disease	State or territory								
	ACT	NSW	NT	Qld	SA	Tas.	Vic.	WA	Aust.
Vaccine preventable diseases *cont'd*									
Varicella zoster (chickenpox)	63	NN	103	301	330	46	822	423	2,088
Varicella zoster (shingles)	92	NN	245	55	2,031	268	1,416	1,366	5,473
Varicella zoster (unspecified)	183	NN	8	5,544	146	141	4,810	1,265	12,097
Vectorborne diseases									
Arbovirus infection (NEC)	0	5	0	22	0	0	1	0	28
Barmah Forest virus infection	1	163	30	473	1	0	18	55	741
Dengue virus infection	16	377	62	393	72	17	329	450	1,716
Japanese encephalitis virus infection	0	0	0	0	1	0	0	0	1
Kunjin virus infection	0	0	0	0	0	0	1	0	1
Malaria	10	88	11	86	6	4	69	48	322
Murray Valley encephalitis virus infection	0	0	0	0	0	0	0	0	0
Ross River virus infection	5	682	412	2,344	75	18	208	1,572	5,316
Zoonoses									
Anthrax	0	0	0	0	0	0	0	0	0
Australia bat lyssavirus	0	0	0	0	0	0	0	0	0
Brucellosis	0	4	1	8	0	0	4	0	17
Leptospirosis	0	14	2	59	1	1	8	3	88
Lyssavirus (NEC)	0	0	0	0	0	0	0	0	0
Ornithosis	0	14	0	4	0	0	21	2	41
Q fever	1	179	1	240	10	0	33	5	469
Tularaemia	0	0	0	0	0	0	0	0	0
Other bacterial infections									
Legionellosis	2	70	7	94	40	8	87	116	424
Leprosy	0	1	0	1	1	0	1	5	9
Meningococcal infection††	2	38	3	40	34	2	33	18	170
Tuberculosis	30	472	28	165	48	9	448	139	1,339
Total	4,331	67,139	8,069	69,208	25,083	4,689	64,173	32,891	275,581

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

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

‡ In Queensland, includes newly acquired hepatitis C cases.

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

|| The national case definitions for chlamydial, gonococcal and syphilis diagnoses include infections that may be acquired through a non-sexual mode (especially in children – e.g. perinatal infections, epidemic gonococcal conjunctivitis).

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

** This number may underrepresent the number of diphtheria cases in Australia. For more details please see the 2014 summary of diphtheria in the Vaccine Preventable Diseases section.

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

NEC Not elsewhere classified.

NN Not notifiable.

Table 5: Notification rates per 100,000 of nationally notifiable communicable diseases, Australia, 2014, by state or territory

Disease	State or territory								Aust.
	ACT	NSW	NT	Qld	SA	Tas.	Vic.	WA	
Bloodborne diseases									
Hepatitis B (newly acquired)*	0.5	0.4	1.2	1.1	0.4	1.0	0.9	0.9	0.7
Hepatitis B (unspecified)†	24.6	33.4	61.3	20.9	19.2	10.7	29.8	24.5	27.7
Hepatitis C (newly acquired)*‡	2.9	0.3	0.8	NN	2.7	2.7	3.0	6.3	2.3
Hepatitis C (unspecified)†	42.5	47.3	72.8	56.1	26.6	42.2	35.1	38.6	43.7
Hepatitis D	–	0.3	0.4	0.3	0.5	–	0.2	0.1	0.3
Gastrointestinal diseases									
Botulism	–	–	–	<0.1	–	–	–	–	<0.1
Campylobacteriosis	131.0	NN	120.2	131.7	107.0	181.5	123.5	115.5	124.9
Cryptosporidiosis	7.8	5.6	35.6	14.1	13.3	5.8	10.9	12.1	10.2
Haemolytic uraemic syndrome	–	0.1	0.4	0.1	0.2	0.2	0.1	<0.1	0.1
Hepatitis A	1.3	1.1	0.8	0.9	0.4	0.2	1.2	0.7	1.0
Hepatitis E	0.3	0.5	–	0.1	–	–	0.2	–	0.2
Listeriosis	0.3	0.3	0.8	0.4	0.4	0.8	0.4	0.2	0.3
Salmonellosis	58.4	57.4	186.8	104.6	72.4	48.4	63.3	49.2	69.7
Shigellosis	2.3	2.6	40.5	3.7	2.1	0.4	7.9	2.7	4.5
Shiga toxin producing *Escherichia coli*	–	0.4	–	0.6	2.7	–	0.2	0.1	0.5
Typhoid fever	0.3	0.6	0.4	0.4	0.5	0.2	0.5	0.5	0.5
Quarantinable diseases									
Cholera	–	–	–	–	–	–	<0.1	–	<0.1
Highly pathogenic avian influenza in humans	–	–	–	–	–	–	–	–	–
Plague	–	–	–	–	–	–	–	–	–
Rabies	–	–	–	–	–	–	–	–	–
Severe acute respiratory syndrome	–	–	–	–	–	–	–	–	–
Smallpox	–	–	–	–	–	–	–	–	–
Viral haemorrhagic fever	–	–	–	–	–	–	–	–	–
Yellow fever	–	–	–	–	–	–	–	–	–
Sexually transmitted infections									
Chlamydial infection§‖	310.3	304.8	1225.2	433.8	326.1	344.7	341.0	442.3	366.8
Donovanosis	–	–	–	–	–	–	–	<0.1	<0.1
Gonococcal infection‖	31.1	64.7	711.7	57.6	43.7	12.6	55.4	85.5	66.8
Syphilis – congenital‖	–	–	2.0	–	–	–	–	–	<0.1
Syphilis < 2 years duration*‖¶	4.7	9.8	29.8	8.3	1.7	2.7	11.1	3.6	8.6
Syphilis > 2 years or unspecified duration†‖	6.7	7.1	29.8	5.9	7.3	3.7	13.7	2.5	8.2
Vaccine preventable diseases									
Diphtheria**	–	–	–	<0.1	–	–	–	–	<0.1
Haemophilus influenzae type b	–	0.1	0.4	0.2	0.1	–	0.1	<0.1	0.1
Influenza (laboratory confirmed)	326.9	277.8	331.1	379.6	655.1	130.8	169.7	204.6	288.6
Measles	1.8	0.9	21.3	1.5	0.9	1.0	1.3	1.7	1.4
Mumps	0.5	1.1	0.4	1.0	1.1	1.0	0.2	0.9	0.8
Pertussis	60.4	41.7	33.9	29.5	29.8	13.2	80.5	68.3	50.5
Pneumococcal disease (invasive)	3.9	6.9	17.6	4.9	7.9	7.6	6.5	8.1	6.7
Poliomyelitis	–	–	–	–	–	–	–	–	–
Rubella	–	0.1	–	<0.1	0.1	–	<0.1	<0.1	0.1
Rubella – congenital	–	–	–	–	–	–	–	–	–
Tetanus	–	<0.1	–	<0.1	–	–	–	<0.1	<0.1

Table 5 *continued*: Notification rates per 100,000 of nationally notifiable communicable diseases, Australia, 2014, by state or territory

Disease	State or territory								Aust.
	ACT	NSW	NT	Qld	SA	Tas.	Vic.	WA	
Vaccine preventable diseases *cont'd*									
Varicella zoster (chickenpox)	16.3	NN	42.1	6.4	19.6	8.9	14.1	16.5	13.1
Varicella zoster (shingles)	23.9	NN	100.2	1.2	120.5	52.1	24.2	53.2	34.3
Varicella zoster (unspecified)	47.5	NN	3.3	117.4	8.7	27.4	82.4	49.3	75.8
Vectorborne diseases									
Arbovirus infection (NEC)	–	0.1	–	0.5	–	–	<0.1	–	0.1
Barmah Forest virus infection	0.3	2.2	12.3	10.0	0.1	–	0.3	2.1	3.2
Dengue virus infection	4.2	5.0	25.3	8.3	4.3	3.3	5.7	17.5	7.3
Japanese encephalitis virus infection	–	–	–	–	0.1	–	–	–	<0.1
Kunjin virus infection	–	–	–	–	–	–	<0.1	–	<0.1
Malaria	2.6	1.2	4.5	1.8	0.4	0.8	1.2	1.9	1.4
Murray Valley encephalitis virus infection	–	–	–	–	–	–	–	–	–
Ross River virus infection	1.3	9.1	168.4	49.6	4.4	3.5	3.6	61.3	22.6
Zoonoses									
Anthrax	–	–	–	–	–	–	–	–	–
Australia bat lyssavirus	–	–	–	–	–	–	–	–	–
Brucellosis	–	0.1	0.4	0.2	–	–	0.1	–	0.1
Leptospirosis	–	0.2	0.8	1.2	0.1	0.2	0.1	0.1	0.4
Lyssavirus (NEC)	–	–	–	–	–	–	–	–	–
Ornithosis	–	0.2	–	0.1	–	–	0.4	0.1	0.2
Q fever	0.3	2.4	0.4	5.1	0.6	–	0.6	0.2	2.0
Tularaemia	–	–	–	–	–	–	–	–	–
Other bacterial infections									
Legionellosis	0.5	0.9	2.9	2.0	2.4	1.6	1.5	4.5	1.8
Leprosy	–	<0.1	–	<0.1	0.1	–	<0.1	0.2	<0.1
Meningococcal infection††	0.5	0.5	1.2	0.8	2.0	0.4	0.6	0.7	0.7
Tuberculosis	7.8	6.3	11.4	3.5	2.8	1.7	7.7	5.4	5.7

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

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

‡ In Queensland, includes newly acquired hepatitis C cases.

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

|| The national case definitions for chlamydial, gonococcal and syphilis diagnoses include infections that may be acquired through a non-sexual mode (especially in children – e.g. perinatal infections, epidemic gonococcal conjunctivitis).

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

** This number may underrepresent the number of diphtheria cases in Australia. For more details please see the 2014 summary of diphtheria in the Vaccine Preventable Diseases section.

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

NEC Not elsewhere classified.

NN Not notifiable.

Table 6: Notified cases and notification rate for communicable diseases, Australia, 2009 to 2014

Disease	Number of notified cases						5 year mean	Ratio (2014: 5 year mean)	Notification rate per 100,000					
	2009	2010	2011	2012	2013	2014			2009	2010	2011	2012	2013	2014
Bloodborne diseases														
Hepatitis B (newly acquired)*	253	231	192	196	175	176	209.4	0.8	1.2	1.0	0.9	0.9	0.8	0.7
Hepatitis B (unspecified)†	6,963	6,796	6,404	6,443	6,940	6,494	6,709.2	1.0	32.1	30.8	28.7	28.3	30.0	27.7
Hepatitis C (newly acquired)*‡	400	383	411	472	398	433	412.8	1.0	2.3	2.2	2.3	2.6	2.2	2.3
Hepatitis C (unspecified)†	11,066	11,062	9,912	9,662	10,339	10,249	10,408.2	1.0	51.0	50.2	44.4	42.5	44.7	43.7
Hepatitis D	51	44	47	36	61	59	47.8	1.2	0.2	0.2	0.2	0.2	0.3	0.3
Gastrointestinal diseases														
Botulism	1	0	2	0	4	1	1.4	0.7	<0.1	–	<0.1	–	<0.1	<0.1
Campylobacteriosis	16,104	16,993	17,726	15,668	14,692	19,931	16,236.6	1.2	110.0	114.1	117.2	101.6	93.5	124.9
Cryptosporidiosis	4,624	1,482	1,812	3,145	3,846	2,405	2,981.8	0.8	21.3	6.7	8.1	13.8	16.6	10.2
Haemolytic uraemic syndrome	13	9	13	20	15	20	14.0	1.4	0.1	<0.1	0.1	0.1	0.1	0.1
Hepatitis A	563	267	145	166	190	231	266.2	0.9	2.6	1.2	0.6	0.7	0.8	1.0
Hepatitis E	33	37	41	32	34	56	35.4	1.6	0.2	0.2	0.2	0.1	0.1	0.2
Listeriosis	92	71	70	93	76	80	80.4	1.0	0.4	0.3	0.3	0.4	0.3	0.3
Salmonellosis	9,501	11,912	12,275	11,251	12,785	16,358	11,544.8	1.4	43.8	54.1	54.9	49.5	55.3	69.7
Shigellosis	617	552	493	548	538	1,051	549.6	1.9	2.8	2.5	2.2	2.4	2.3	4.5
Shiga toxin-producing *Escherichia coli*	128	80	95	111	180	115	118.8	1.0	0.6	0.4	0.4	0.5	0.8	0.5
Typhoid fever	115	96	135	125	152	119	124.6	1.0	0.5	0.4	0.6	0.5	0.7	0.5
Quarantinable diseases														
Cholera	4	3	6	5	3	2	4.2	0.5	<0.1	<0.1	<0.1	<0.1	<0.1	<0.1
Highly pathogenic avian influenza in humans	0	0	0	0	0	0	0.0	–	–	–	–	–	–	–
Plague	0	0	0	0	0	0	0.0	–	–	–	–	–	–	–
Rabies	0	0	0	0	0	0	0.0	–	–	–	–	–	–	–
Severe acute respiratory syndrome	0	0	0	0	0	0	0.0	–	–	–	–	–	–	–
Smallpox	0	0	0	0	0	0	0.0	–	–	–	–	–	–	–
Viral haemorrhagic fever	0	0	0	0	0	0	0.0	–	–	–	–	–	–	–
Yellow fever	0	0	2	0	0	0	0.4	–	–	–	<0.1	–	–	–

Table 6 *continued*: Notified cases and notification rate for communicable diseases, Australia, 2009 to 2014

Disease	Number of notified cases						5 year mean	Ratio (2014: 5 year mean)	Notification rate per 100,000					
	2009	2010	2011	2012	2013	2014			2009	2010	2011	2012	2013	2014
Sexually transmissible infections														
Chlamydial infection§‖	63,200	74,418	81,099	83,121	82,974	86,108	76,962.4	1.1	291.4	337.8	363.0	365.7	358.9	366.8
Donovanosis	1	1	0	1	0	1	0.6	1.7	<0.1	<0.1	–	<0.1	–	<0.1
Gonococcal infection‖	8,274	10,320	12,095	13,880	14,902	15,675	11,894.2	1.3	38.1	46.8	54.1	61.1	64.5	66.8
Syphilis – congenital‖	3	3	7	1	7	5	4.2	1.2	<0.1	<0.1	<0.1	<0.1	<0.1	<0.1
Syphilis < 2 years duration*‖¶	1,293	1,118	1,280	1,556	1,768	2,009	1,403.0	1.4	6.0	5.1	5.7	6.8	7.6	8.6
Syphilis > 2 years or unspecified duration†‖	1,459	1,358	1,352	1,389	1,747	1,921	1,461.0	1.3	7.3	6.7	6.5	6.1	7.6	8.2
Vaccine preventable diseases														
Diphtheria**	0	0	4	0	2	2	1.2	1.7	–	–	<0.1	–	<0.1	<0.1
Haemophilus influenzae type b	19	24	13	16	20	21	18.4	1.1	0.1	0.1	0.1	0.1	0.1	0.1
Influenza (laboratory confirmed)	59,026	13,466	27,233	44,571	28,311	67,742	34,521.4	2.0	272.1	61.1	121.9	196.1	122.5	288.6
Measles	104	70	194	199	162	340	145.8	2.3	0.5	0.3	0.9	0.9	0.7	1.4
Mumps	166	98	155	200	218	190	167.4	1.1	0.8	0.4	0.7	0.9	0.9	0.8
Pertussis	30,192	34,845	38,750	24,101	12,362	11,863	28,050.0	0.4	139.2	158.2	173.5	106.0	53.5	50.5
Pneumococcal disease (invasive)	1,556	1,640	1,883	1,822	1,549	1,564	1,690.0	0.9	7.2	7.4	8.4	8.0	6.7	6.7
Poliomyelitis	0	0	0	0	0	0	0.0	–	–	–	–	–	–	–
Rubella	27	44	58	37	25	17	38.2	0.4	0.1	0.2	0.3	0.2	0.1	0.1
Rubella – congenital	0	0	0	1	2	0	0.6	–	–	–	–	<0.1	<0.1	–
Tetanus	3	2	3	7	4	3	3.8	0.8	<0.1	<0.1	<0.1	<0.1	<0.1	<0.1
Varicella zoster (chickenpox)	1,796	1,793	2,100	1,983	2,127	2,088	1,959.8	1.1	12.3	12.0	13.9	12.9	13.5	13.1
Varicella zoster (shingles)	2,779	3,045	4,022	4,506	5,038	5,473	3,878.0	1.4	19.0	20.5	26.6	29.2	32.1	34.3
Varicella zoster (unspecified)	7,425	8,155	8,608	9,421	10,983	12,097	8,918.4	1.4	50.7	54.8	56.9	61.1	69.9	75.8
Vectorborne diseases														
Arbovirus infection (NEC)	5	14	16	6	19	28	12.0	2.3	<0.1	0.1	0.1	<0.1	0.1	0.1
Barmah Forest virus infection	1,473	1,470	1,863	1,730	4,239	741	2,155.0	0.3	6.8	6.7	8.3	7.6	18.3	3.2
Dengue virus infection	1,402	1,228	821	1,541	1,840	1,716	1,366.6	1.3	6.5	5.6	3.7	6.8	8.0	7.3
Japanese encephalitis virus infection	0	0	0	1	4	1	1.0	1.0	–	–	–	<0.1	<0.1	<0.1
Kunjin virus infection	2	2	2	0	2	1	1.6	0.6	<0.1	<0.1	<0.1	–	<0.1	<0.1
Malaria	504	405	418	344	416	322	418.2	0.8	2.3	1.8	1.9	1.5	1.8	1.4
Murray Valley encephalitis virus infection	4	0	16	1	1	0	4.4	<0.1	<0.1	–	0.1	<0.1	<0.1	–
Ross River virus infection	4,741	5,129	5,137	4,682	4,316	5,316	4,801.0	1.1	21.9	23.3	23.0	20.6	18.7	22.6

Disease	Number of notified cases						5 year mean	Ratio (2014: 5 year mean)	Notification rate per 100,000					
	2009	2010	2011	2012	2013	2014			2009	2010	2011	2012	2013	2014
Zoonoses														
Anthrax	0	1	0	0	0	0	0.2	–	–	<0.1	–	–	–	–
Australian bat lyssavirus	0	0	0	0	1	0	0.2	–	–	–	–	–	<0.1	–
Brucellosis	32	21	37	31	14	17	27.0	0.6	0.1	0.1	0.2	0.1	0.1	0.1
Leptospirosis	141	131	215	114	88	88	137.8	0.6	0.7	0.6	1.0	0.5	0.4	0.4
Lyssavirus (NEC)	0	0	0	0	0	0	0.0	–	–	–	–	–	–	–
Ornithosis	63	58	89	76	47	41	66.6	0.6	0.3	0.3	0.4	0.3	0.2	0.2
Q fever	314	338	359	369	487	469	373.4	1.3	1.4	1.5	1.6	1.6	2.1	2.0
Tularaemia	0	0	2	0	0	0	0.4	–	–	–	<0.1	–	–	–
Other bacterial infections														
Legionellosis	297	307	358	383	508	424	370.6	1.1	1.4	1.4	1.6	1.7	2.2	1.8
Leprosy	5	10	10	8	14	9	9.4	1.0	<0.1	<0.1	<0.1	<0.1	0.1	<0.1
Meningococcal infection††	260	228	242	223	149	170	220.4	0.8	1.2	1.0	1.1	1.0	0.6	0.7
Tuberculosis	1,307	1,364	1,389	1,316	1,263	1,339	1,327.8	1.0	6.0	6.2	6.2	5.8	5.5	5.7
Total	238,401	211,124	239,611	245,610	226,037	275,581								

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

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

‡ In Queensland, includes newly acquired hepatitis C cases.

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

|| The national case definitions for chlamydial, gonococcal and syphilis diagnoses include infections that may be acquired through a non-sexual mode (especially in children – e.g. perinatal infections, epidemic gonococcal conjunctivitis).

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

** This number may underrepresent the number of diphtheria cases in Australia. For more details please see the 2014 summary of diphtheria in the Vaccine Preventable Diseases section.

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

NEC Not elsewhere classified.

NN Not notifiable.

Data completeness

Indigenous status

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

Indigenous status was complete in 45% of all notifications reported to NNDSS in 2014. Indigenous status was complete in 95% of data reported in the Northern Territory, 93% in Western Australia and 91% in South Australia. In the remaining jurisdictions, Indigenous status completeness ranged from 14% to 47% (Table 7).

Data completeness on Indigenous status also varied by disease as summarised in Appendix 3. In 2009, CDNA set target thresholds of 95% completeness for 18 priority diseases (17 notifiable to NNDSS and HIV, which are provided to the Kirby Institute) (Table 8) and 80% completeness for the remainder of the notifiable diseases as part of its 'Closing the Gap' strategy. Of all diseases notified to the NNDSS in 2014, 31 (62%) equalled or exceeded 80% completeness for Indigenous status and 15 (48%) were priority diseases.

In 2014, 11 of the 17 priority diseases notified to NNDSS had an Indigenous completeness that exceeded 95% (congenital syphilis, donovanosis, *Haemophilus influenzae* type b, hepatitis A, hepatitis C (newly acquired), measles, meningococcal infection, pneumococcal disease less than 5 years, pneumococcal disease ≥ 50 years, leprosy, and tuberculosis). This was an improvement on 2013 where 7 priority diseases exceeded 95% completeness. There has been a notable improvement in completeness of Indigenous status for hepatitis C (newly acquired) notifications from 88% in 2012 to 98% in 2014.

A review of the NNDSS priority diseases between 2004 and 2014 showed that meningococcal disease (invasive) and tuberculosis exceeded the 95% threshold for completeness of Indigenous status over the entire period. They ranged from 95%–97% and 98%–100% respectively.

Six of the priority diseases were consistently below the 95% threshold over the entire period (Figure 3):

- dengue virus infection (locally acquired)
- gonococcal infection
- hepatitis B (newly acquired)
- hepatitis C (newly acquired)
- pertussis less than 5 years
- shigellosis.

The completeness of the Indigenous status for 8 of the priority diseases has improved since 2004:

- congenital syphilis, increasing from 93% in 2004 to 100% in 2014;
- *Haemophilus influenzae* type b, increasing from 93% in 2004 to 100% in 2014
- hepatitis A, increasing from 90% in 2004 to 96% in 2014;
- hepatitis B (newly acquired), increasing from 81% in 2004 to 92% in 2014;
- hepatitis C (newly acquired), increasing from 85% in 2004 to 98% in 2014;
- pneumococcal disease less than 5 years, increasing from 89% in 2004 to 100% in 2014;
- pneumococcal disease 50 years or over, increasing from 90% in 2004 to 97% in 2014; and
- shigellosis, increasing from 72% in 2004 to 81% in 2014.

The completeness of Indigenous status for 2 diseases has not improved since 2004. Dengue virus infection (locally acquired), decreased from 86% in 2004 to 69% in 2014, and gonococcal infection, decreased from 69% in 2004 to 66% in 2014.

The completeness of Indigenous status for syphilis < 2 years was 92% in 2014 but has regularly exceeded the 95% threshold over the period.

Table 7: Indigenous status completeness of National Notifiable Diseases Surveillance System data, Australia, 2014, by state or territory

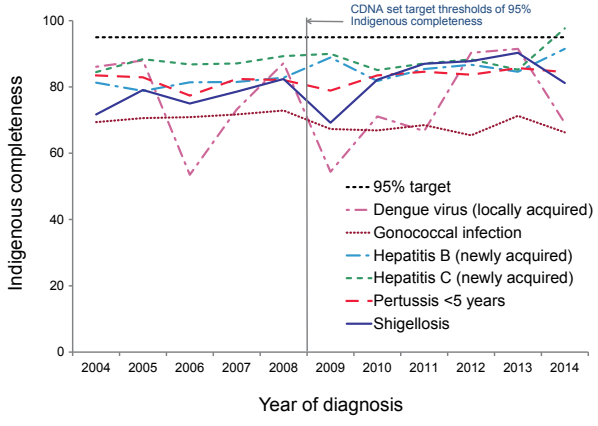
	State or territory								
	ACT	NSW	NT	Qld	SA	Tas.	Vic.	WA	Aust.
Total notifications	4,331	67,139	8,069	69,207	25,083	4,689	64,172	32,891	275,581
Indigenous status									
Unknown/ missing	2,284	57,507	437	39,027	2,191	3,066	43,940	2,232	150,684
Per cent complete	47	14	95	44	91	35	32	93	45

Table 8: Percentage completeness of priority diseases for Indigenous status completeness of National Notifiable Diseases Surveillance System data, Australia, 2014, by state or territory

Priority disease	ACT	NSW	NT	Qld	SA	Tas.	Vic.	WA	Aust.
Congenital syphilis	No cases	No cases	100	No cases	No cases	No cases	No cases	No cases	100
Dengue virus (locally acquired)	No cases	100	No cases	69	100	No cases	67	100	69
Donovanosis	No cases	No cases	No cases	No cases	No cases	No cases	No cases	100	100
Gonococcal infection	100	45	99	59	99	94	55	100	66
Haemophilus influenzae type b	No cases	100	100	100	100	No cases	100	100	100
Hepatitis A	100	100	100	80	100	100	100	100	96
Hepatitis B (newly acquired)	100	100	100	74	100	100	98	100	92
Hepatitis C (newly acquired)	100	100	100	NN	100	100	95	99	98
Leprosy	No cases	100	No cases	100	100	No cases	100	100	100
Measles	100	88	98	97	100	100	97	100	96
Meningococcal disease (invasive)	100	100	100	100	100	100	94	100	99
Pertussis <5 years	100	91	100	65	100	100	81	97	85
Pneumococcal disease <5 years	100	100	100	100	100	100	100	100	100
Pneumococcal disease ≥50 years	100	99	100	98	100	100	91	100	97
Shigellosis	100	83	98	61	100	100	80	100	81
Syphilis < 2 years	100	90	100	95	100	100	89	100	92
Tuberculosis	100	100	100	100	100	100	100	100	100

NN Not notifiable.

Figure 3: Priority diseases consistently below the 95% threshold for Indigenous completeness, 2004 to 2014



Place of acquisition

The place of acquisition is where the disease is known to have been acquired, either locally or overseas and is usually obtained through public health follow-up. Follow-up and thus completeness varies by disease and by jurisdiction. It is not possible to follow-up all cases for diseases with a large volume of notifications. Place of acquisition is not usually completed for diseases unless overseas travel is known to be a risk factor.

Through the NSC, jurisdictions have agreed that completeness for place of acquisition should be 100% for the following 24 priority diseases:

- arbovirus infection (NEC)
- brucellosis
- cholera
- dengue virus infection
- hepatitis A
- highly pathogenic avian influenza in humans
- Japanese encephalitis virus infection
- Kunjin virus infection
- legionellosis
- leprosy
- malaria
- measles
- Murray Valley encephalitis virus infection
- plague

- poliomyelitis
- Q fever
- rabies
- rubella
- severe acute respiratory syndrome
- smallpox
- tularaemia
- typhoid fever
- viral haemorrhagic fever (NEC)
- yellow fever.

In 2014, 14 of the 24 priority diseases had cases notified to NNDSS, the overall completeness for place of acquisition for these diseases was 96%. The completeness was 100% in 2014 for cholera, brucellosis, leprosy, Kunjin virus infection and Japanese encephalitis virus infection (Table 9).

Bloodborne diseases

In 2014, the bloodborne diseases reported to the NNDSS were hepatitis B, C, and D infections. Both hepatitis B and C infections were notified to the NNDSS as either 'newly acquired', where evidence was available that the infection was acquired in the 24 months prior to diagnosis; or 'greater than 2 years or unspecified' period of infection. These categories were reported from all states and territories except Queensland where all cases of hepatitis C infection, including newly acquired, were reported as being 'greater than 2 years or unspecified'.[19] Determination of a case as 'newly acquired' is reliant on public health follow-up, with the method and intensity of follow-up varying by jurisdiction and over time.

In interpreting these data it is important to note that changes in notified cases over time may not solely reflect changes in disease prevalence or incidence. National testing policies developed by the Australian Society for HIV Medicine and screening programs, including the preferential testing of high risk populations such as prisoners, injecting drug users and persons from countries with a high prevalence of hepatitis B or C infection, may contribute to these changes.[20,21]

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

Table 9: Percentage completeness of priority diseases* for place of acquisition completeness of National Notifiable Diseases Surveillance System data, Australia, 2014, by state or territory

Disease	2010	2011	2012	2013	2014
Cholera	100	100	100	100	100
Legionellosis	97	83	85	80	86
Murray Valley encephalitis virus infection	No cases	88	100	100	No cases
Tularaemia	No cases	100	No cases	No cases	No cases
Malaria	94	97	97	96	98
Dengue virus infection	>99	98	98	99	99
Yellow fever	No cases	100	No cases	No cases	No cases
Brucellosis	81	24	36	100	100
Hepatitis A	>99	100	94	95	99
Typhoid fever	100	98	94	98	97
Rubella	89	74	62	80	53
Leprosy	80	90	75	79	100
Measles	100	89	95	98	>99
Kunjin virus infection	50	100	No cases	100	100
Japanese encephalitis virus infection	No cases	No cases	100	100	100
Flavivirus unspecified	93	75	100	100	96
Q fever	81	57	78	89	89
Total	96	88	92	95	96

* Only includes priority diseases notified to the National Notifiable Diseases Surveillance System in 2010 to 2014 are included.

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

Hepatitis B

- In 2014, 6,670 cases of hepatitis B were notified to the NNDSS.
- Over the past 10 years, notifications of newly acquired hepatitis B have declined.

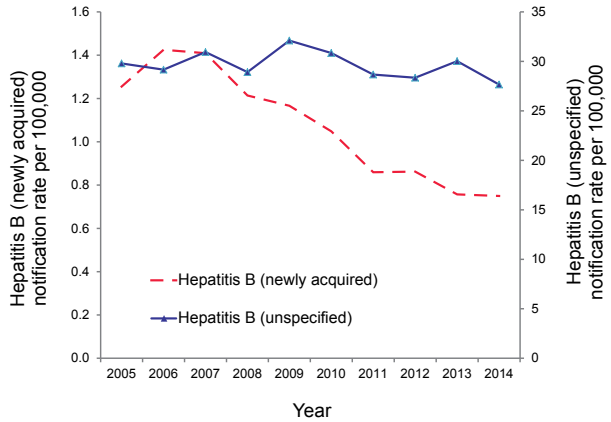
Hepatitis B is a potentially life-threatening liver infection caused by the hepatitis B virus. Major modes of transmission include unprotected sexual contact or needle sharing with an infected person, and perinatal transmission from mother to child. Symptoms of acute infection include abdominal pain, nausea and vomiting progressing to jaundice. Outcomes vary inversely with age; infected infants are more likely to progress to chronic infection whereas people who are infected as adults often clear the virus. Chronic infection can lead to a number of liver complications including cirrhosis, cancer and liver failure.[22]

Hepatitis B notifications are classified as being either 'newly acquired' (evidence that infection was acquired within the 24 months prior to diagnosis) or 'unspecified' (infection acquired more than 24 months prior to diagnosis or not able to be specified).

Epidemiological situation in 2014

In 2014, there were 6,670 notified cases of hepatitis B (both newly acquired and unspecified), representing a rate of 28.4 cases per 100,000 (Figure 4).

Figure 4: Notification rate for newly acquired hepatitis B and unspecified hepatitis B, Australia, 2005 to 2014, by year



Between 2005 and 2014, rates of newly acquired hepatitis B decreased by 40% from 1.3 to 0.7 per 100,000 (Figure 4). The decline in newly acquired hepatitis B notifications may be attributed to the hepatitis B vaccination program, which was introduced nationally for infants in 2000, and nationally funded adolescent hepatitis B vaccination programs, which were introduced from 1997 onwards, depending on the jurisdiction.[23] As at 30 June 2014, approximately 92% of children 12–15 months of age in Australia were assessed as being fully immunised for hepatitis B.[24] A 2007 study showed significant improvements in immunity to hepatitis B for the 12–17 years age group in jurisdictions with established school-based programs compared to those jurisdictions without such programs.[25]

From the 1980s, hepatitis B vaccination was also recommended for certain at-risk adults in Australia.[26] Some jurisdictions implemented vaccination programs to target identified at-risk adults in a variety of settings and at various times.[27] The full impact of Australian vaccination programs should be reflected in trends in chronic infection and reductions in hepatitis B related complications in the near future.[28]

Between 2005 and 2014, rates of unspecified hepatitis B have declined slightly by 7% from 29.8 to 27.7 per 100,000. It is important to note the significant impact of immigration on rates for unspecified hepatitis B. In 2014, Western Australia reported a decline in asylum seeker boat arrivals coinciding with a decline in unspecified hepatitis B notifications in the state, particularly in the Kimberley region (which includes the postcode for Christmas Island). In 2011, an Australian study estimated that more than 95% of new cases of chronic hepatitis B virus infection entered the population through migration.[29]

Newly acquired hepatitis B

- In 2014, 176 cases of newly acquired hepatitis B were notified to the NNDSS.
- The highest rate of notification was among males aged 35–39 years.

Epidemiological situation in 2014

In 2014, 176 cases of newly acquired hepatitis B infection were notified to the NNDSS, a rate of 0.7 per 100,000, which is similar to the 175 cases (0.8 per 100,000) reported in 2013 (Figure 4).

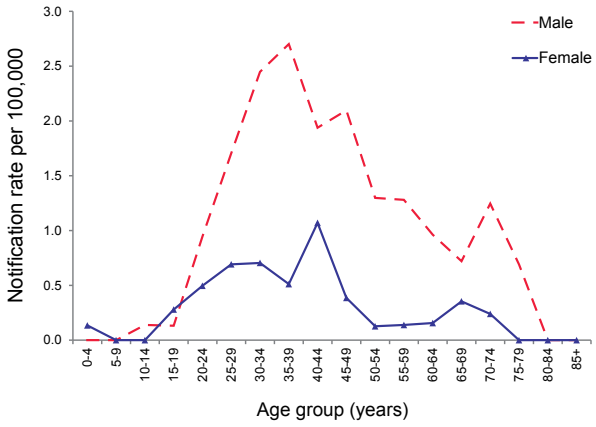
Geographical distribution

The highest rates were reported from the Northern Territory (1.2 per 100,000) and Queensland (1.1 per 100,000) (Table 5). This may be due to population differences between the jurisdictions, with hepatitis B disproportionately affecting a number of marginalised groups in Australia including migrant communities with origins in Asia, the Pacific and Africa; and Aboriginal and Torres Strait Islander people.[29–31]

Age and sex distribution

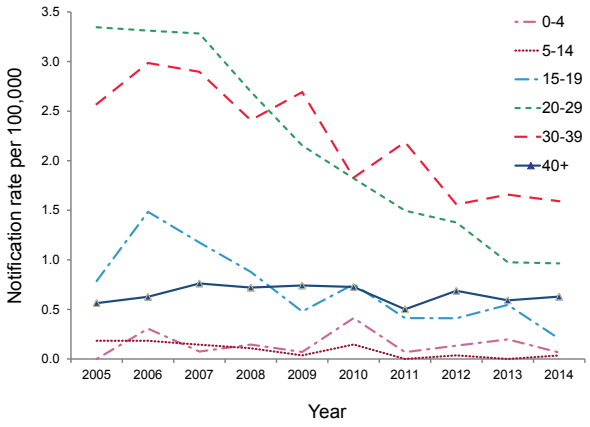
In 2014, males accounted for 77% of newly acquired hepatitis B notifications. In 2014, the highest rate of newly acquired hepatitis B infection was observed among males aged 35–39 years (2.7 per 100,000). For females, the highest rate was in those aged 40–44 years (1.1 per 100,000) (Figure 5). Exposure to hepatitis B may be more common in certain high risk groups, including immigrants from endemic regions; injecting drug users; prisoners; Aboriginal and Torres Strait Islander peoples; and men who have sex with men.[22,29] The greater representation of males in some of these groups may contribute to the higher notification rates among males.

Figure 5: Notification rate for newly acquired hepatitis B, Australia, 2014, by age group and sex



Between 2005 and 2014, most age group specific notification rates were low and remained stable or trended downwards. The most marked decreases occurred among those aged 15–39 years. During this period, notification rates declined by 74% for those aged 15–19 years (from 0.8 to 0.2 per 100,000), by 71% for those aged 20–29 years (from 3.3 to 1.0 per 100,000) and by 38% for those aged 30–39 years (from 2.6 to 1.6 per 100,000) (Figure 6). These declines are likely to be attributable to the hepatitis B vaccination program.[27]

Figure 6: Notification rate for newly acquired hepatitis B, Australia, 2005 to 2014, by year and selected age groups



Risk groups

Enhanced data on risk factors and country of birth were provided by the Australian Capital Territory, New South Wales, South Australia, Tasmania, Victoria and Western Australia* (Table 10). In 2014, 98 of 106 cases (92%) from these jurisdictions had at least 1 risk factor recorded, with a potential source of exposure not recorded or unable to be determined for the remainder. Sexual exposure was the most frequently reported potential source of infection (56/106, 53%), followed by injecting drug use (36/106, 34%). Of the 118 cases for which country of birth was reported, 91 were in Australian born persons (77%) and 27 cases were born overseas (10 from Asia, 8 from Europe, 4 from the Middle East, 4 from the Pacific, and 1 from South America).

Unspecified hepatitis B

- In 2014, 6,494 cases of unspecified hepatitis B were notified to the NNDSS.
- Notification rates peaked in females and males aged 30–34 years.

Epidemiological situation in 2014

In 2014, 6,494 cases of unspecified hepatitis B infection were notified to the NNDSS, representing a rate of 27.7 per 100,000, compared with 6,940 cases (30.0 per 100,000) reported in 2013 (Figure 4).

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

Table 10: Enhanced risk factor data on notifications of newly acquired hepatitis B cases in selected jurisdictions,* 2014, by sex and risk factors†‡

Exposure category	Number of exposure factors reported			Percentage of total cases* (n=106)
	Male	Female	Total	
Sexual exposure	45	11	56	53
Sexual contact (hepatitis B partner status unknown) – opposite sex	16	6	22	21
Sexual contact (hepatitis B positive partner) – opposite sex	6	4	10	9
Sexual contact – not further classified	9	1	10	9
Sexual contact (hepatitis B partner status unknown) – same sex	10	0	10	9
Sexual contact (hepatitis B positive partner) – same sex	4	0	4	4
Injecting drug use	26	10	36	34
Skin penetration procedure	5	2	7	7
Tattoos	4	1	5	5
Ear or body piercing	0	1	1	1
Acupuncture	1	0	1	1
Undetermined	3	3	6	6
Imprisonment	5	0	5	5
Healthcare exposure	3	0	3	3
Major dental surgery work	2	0	2	2
Surgical work	1	0	1	1
Other	11	7	18	17
Other risk not elsewhere classified (≤24 months prior to diagnosis)	9	3	12	11
Non-IDU remote risk (>24 months prior to diagnosis)	1	2	3	3
Needlestick/biohazardous injury	1	1	2	2
Household contact	0	1	1	1
Unknown (not recorded)	2	0	2	
Total exposure factors reported	98	33	131	
Total number of cases	80	26	106	

* Cases from the Australian Capital Territory, New South Wales, South Australia, Tasmania, Victoria and Western Australia. While these 6 jurisdictions provided enhanced data on risk factors, not all cases had this information recorded.

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

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

§ The denominator used to calculate the percentage is based on the cases with recorded enhanced data from the Australian Capital Territory, New South Wales, South Australia, Tasmania, Victoria and Western Australia. As more than 1 exposure category for each notification could be recorded, the total percentage does not equate to 100%.

Geographical distribution

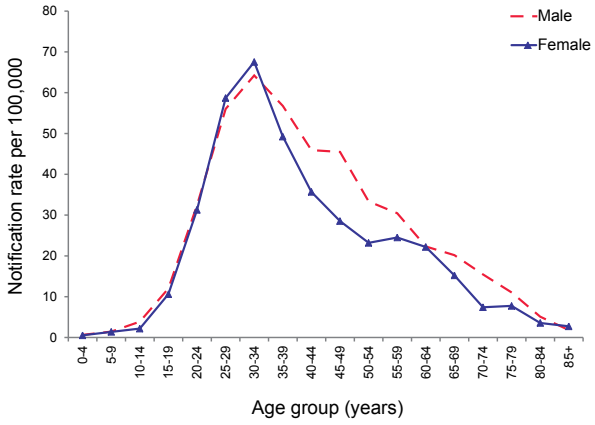
In 2014, the Northern Territory had the highest rate of unspecified hepatitis B infection (61.3 per 100,000) (Table 5).

Age and sex distribution

In 2014, males accounted for 53% (3,451/6,494) of unspecified hepatitis B notifications, with an overall rate of 29.5 per 100,000 for males and 25.5 per 100,000 for females. Notification rates were similar for males and females in most age groups. Notification rates in both males and females peaked in the 30–34 years age group (Figure 7).

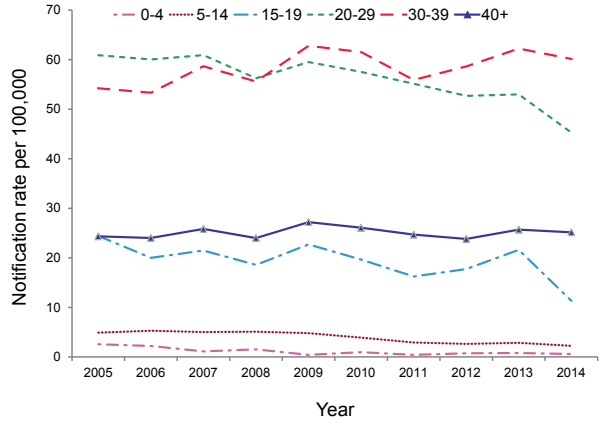
Between 2005 and 2014, notification rates for unspecified hepatitis B decreased overall in the age groups less than 30 years of age but slightly increased in those aged 30–39 years and remained relatively stable in those aged 40 years or over (Figure 8). The decrease in rates for the younger age groups is likely explained by the introduction of the infant and adolescent hepatitis B vaccination programs.[26] The adolescent vaccination program commenced in some jurisdictions from 1997 and the infant vaccination program commenced nationally from 2000.[32]

Figure 7: Notification rate for unspecified hepatitis B, Australia, 2014, by age group and sex*



* Excludes 36 cases where age and/or sex were not reported.

Figure 8: Notification rate for unspecified hepatitis B, Australia, 2005 and 2014, by year and selected age groups*



* Excludes 15 cases where age was not reported.

Hepatitis C

- In 2014, 10,682 cases of hepatitis C were notified to the NNDSS.
- Over the past 10 years, notifications of hepatitis C have declined by 12%.

Infection with hepatitis C virus causes inflammation of the liver. In more than 90% of cases, initial infection with hepatitis C virus is asymptomatic or mildly symptomatic. Approximately 50%–80% of cases go on to develop a chronic infection. Of those who develop a chronic infection, half will eventually develop cirrhosis or cancer of the liver.[22] There is no vaccine to prevent hepatitis C infection.

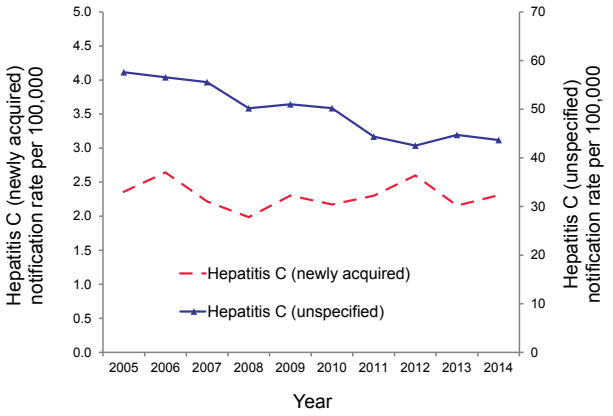
Hepatitis C notifications are classified as being either 'newly acquired' (evidence that infection was acquired within the 24 months prior to diagnosis) or 'unspecified' (infection acquired more than 24 months prior to diagnosis or not able to be specified).

Epidemiological situation in 2014

Of the 10,682 cases of hepatitis C notified in 2014, 4% (433/10,682) were identified as having been newly acquired infections. The proportion of hepatitis C notifications identified as newly acquired has remained reasonably stable since 2005 (range: 3%–5%).

Between 2005 and 2014, hepatitis C notifications (both newly acquired and unspecified) declined by 12% from 12,135 to 10,682. This was mainly due to a downward trend in unspecified hepatitis C notifications, while newly acquired hepatitis C notifications remained low and relatively stable (Figure 9).

Figure 9: Notification rate for hepatitis C (newly acquired* and unspecified†) infection, Australia, 2005 to 2014, by year



* Data from all states and territories except Queensland.

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

Newly acquired hepatitis C

- In 2014, 433 cases of newly acquired hepatitis C were notified to the NNDSS.
- The majority of newly acquired cases had a history of injecting drug use.
- The highest notification rate was among males in the 20–24 years age group.

Epidemiological situation in 2014

Cases of newly acquired hepatitis C infection were reported from all states and territories except Queensland, where all cases of hepatitis C infection are reported as unspecified. Nationally, the notification rate in 2014 was 2.3 per 100,000 (n=433) compared with 2.2 per 100,000 (n=398) in 2013 (Figure 9).

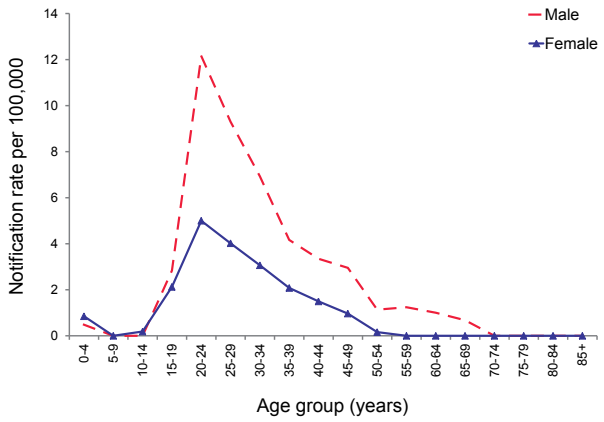
Geographical distribution

In 2014, Western Australia reported the highest jurisdiction-specific rate of newly acquired hepatitis C infection (6.3 per 100,000) (Table 5).

Age and sex distribution

In 2014, males accounted for 70% (304/433) of newly acquired hepatitis C notifications. In 2014, the highest notification rate for newly acquired hepatitis C infection was observed among males aged 20–24 years (12.2 per 100,000). For females, the highest notification rate was in those aged 20–24 years (5.0 per 100,000) (Figure 10).

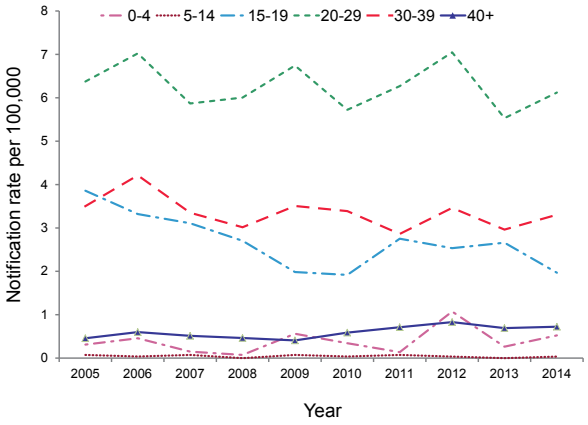
Figure 10: Notification rate for newly acquired hepatitis C* infection, Australia, 2014, by age group and sex



* Data from all states and territories except Queensland.

Between 2005 and 2014, notification rates for newly acquired hepatitis C infection have declined overall among those in the 15–39 years age groups. The largest decrease from 2005 to 2014 occurred in the 15–19 years age groups (from 3.9 to 2.1 per 100,000) (Figure 11). This may be partly explained by the findings of a recent survey, which suggested a decrease in the prevalence of injecting drug use among young people in Australia.[33]

Figure 11: Notification rate for newly acquired hepatitis C* infection, Australia, 2005 to 2014, by year and selected age groups†



* Data from all states and territories except Queensland.

† Excludes 1 case where age was not reported (2005).

Risk groups

Exposure histories for newly acquired hepatitis C cases reported in 2014 were analysed for all jurisdictions except Queensland (Table 11). In 2014, 99% (343/347) of cases with enhanced data had at least 1 risk factor recorded, with the potential source of exposure not recorded or unable to be determined for the remainder. Of the cases for which exposure history was reported, approximately 80% (279/347) had a history of injecting drug use and approximately 17% (59/347) reported possible sexual exposure.

Approximately 25% (n=86) of cases with exposure history had reported being imprisoned in the 24 months prior to diagnosis. Of these cases, approximately 87% (n=75) also reported a history of injecting drug use. However, it is important to note that screening rates are generally higher in the prison entry population than the general population. A screening survey of prison entrants conducted over a 2-week period found that the prevalence of hepatitis C infection, based on hepatitis C antibody detection, was 22% in 2012, a decrease from 35% in 2007.[34]

Unspecified hepatitis C

- In 2014, 10,249 cases of unspecified hepatitis C infection were notified to the NNDSS.
- The highest notification rates were among males in the 30–49 years age groups.

Table 11: Enhanced risk factor data on notifications of newly acquired hepatitis C infection in selected jurisdictions,* 2014, by sex and risk factors†‡

Exposure category	Number of exposure factors reported			Percentage of total cases§ (n=347)
	Male	Female	Total	
Injecting drug use	190	89	279	80
Imprisonment	77	9	86	25
Sexual contact	41	18	59	17
Sexual contact (hepatitis B positive partner) – opposite sex	19	17	36	10
Sexual contact (hepatitis B partner status unknown)	15	1	16	5
Sexual contact (hepatitis B positive partner) – same sex	7	0	7	2
Perinatal transmission	27	14	41	12
Other	21	15	36	11
Household contact	6	8	14	4
Other risk not elsewhere classified (≤24 months prior to diagnosis)	14	6	20	6
Needlestick/bio-hazardous injury	1	1	2	1
Skin penetration procedure	26	9	35	10
Tattoos	14	4	18	5
Ear or body piercing	5	4	9	3
Acupuncture	7	1	8	2
Healthcare exposure	7	3	10	3
Haemodialysis	4	2	6	2
Surgical work	2	1	3	1
Major dental surgery work	1	0	1	<1
Undetermined	3	6	9	3
Unknown (not recorded)	1	0	1	
Total exposure factors reported	389	157	546	
Total number of cases	233	114	347	

* Includes data from all states and territories except Queensland (not notified). While the 7 jurisdictions provided enhanced data on risk factors, not all cases had this information recorded.

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

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

§ The denominator used to calculate the percentage is based on the total number of notified cases with recorded enhanced data, from all jurisdictions except Queensland (notified as unspecified hepatitis C). As more than 1 exposure category for each case could be recorded, the total percentage does not equate to 100%.

Epidemiological situation in 2014

In 2014, 10,249 cases of unspecified hepatitis C infections were notified to the NNDSS (43.7 per 100,000) compared with 10,339 cases in 2013 (44.7 per 100,000). Apart from slight rises from 2008–2009 and 2012–2013, notification rates have decreased annually since 2005. There was an overall decline of 24% between 2005 (57.6 per 100,000) and 2014 (43.7 per 100,000) (Figure 9).

Several factors may account for the decrease including changes in surveillance practices, removal of duplicate notifications and a gradual decline in the prevalent group of hepatitis C cases accumulated prior to the introduction of hepatitis C testing in the early 1990s.[25,35] The continuing decline in the notification rate may also be attributable to an apparent decrease in the prevalence of injecting drug use among young people in Australia.[33]

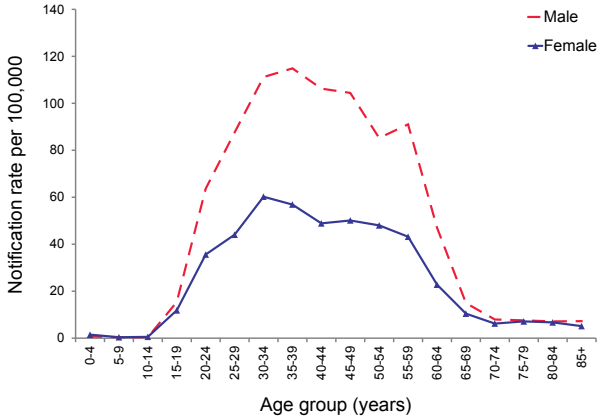
Geographical distribution

For the past 10 years, the Northern Territory has reported the highest jurisdiction-specific notification rate for unspecified hepatitis C. In 2014, the Northern Territory's notification rate was 72.8 per 100,000 (Table 5), which was 41% less than the 2005 rate of 123.6 per 100,000.

Age and sex distribution

Nationally in 2014, 66% (6,718/10,249) of unspecified hepatitis C notifications were in males (for cases where the sex was reported). The notification rate in males was 57.5 per 100,000 and in females 29.8 per 100,000; a male to female rate ratio of 1.9:1. Notification rates in males exceeded those in females across most age groups. The highest notification rates were among males in the 35–39 years (114.9 per 100,000) and 30–34 years (111.2 per 100,000) age groups. The highest notification rates among females were for those in the 30–34 years (60.2 per 100,000) and 35–39 years (56.9 per 100,000) age groups (Figure 12).

Figure 12: Notification rate for unspecified hepatitis C* infection, Australia, 2014, by age group and sex†



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

† Excludes 29 cases where age and/or sex were missing or unknown.

Between 2005 and 2014, notification rates for unspecified hepatitis C infection have declined overall across all age groups, except for the 0–4 years age group for which rates remained relatively stable at 1.1 per 100,000 due to the low number of notifications. The largest decreases have occurred in the 20–29 years (from 111.6 to 58.3 per 100,000), the 30–39 years (117.8 to 86.2 per 100,000) and the 15–19 years (24.3 to 13.7 per 100,000) age groups (Figure 13).

Figure 13: Notification rate for unspecified hepatitis C* infection, Australia, 2005 to 2014, by year and selected age groups†

* Data provided from Queensland (2005–2014) includes both newly acquired and unspecified hepatitis C cases.

† Excludes 54 cases where age was not reported (2005–2007 and 2009–2014).

Hepatitis D

- In 2014, 59 cases of hepatitis D were notified to the NNDSS.
- Hepatitis D is always associated with hepatitis B co-infection.

Hepatitis D is a defective single-stranded RNA virus that replicates in the presence of the hepatitis B virus. Hepatitis D infection can occur as either an acute co-infection with hepatitis B or as a super-infection with chronic hepatitis B infection. The modes of hepatitis D transmission are similar to those for hepatitis B.[22]

Epidemiological situation in 2014

In Australia, the notification rate for hepatitis D infection remains low. In 2014, there were 59 notified cases of hepatitis D, representing a rate of 0.3 per 100,000 (Table 5). Over the preceding 9 years, notifications of hepatitis D remained relatively low with an average of almost 46 cases notified per year (range: 34 to 61).

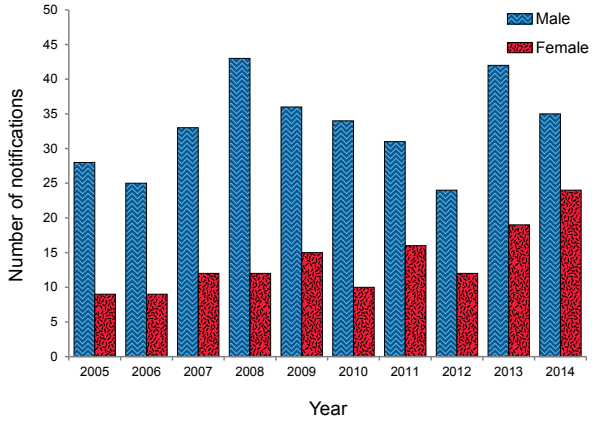
Geographical distribution

In 2014, New South Wales reported the highest number of cases (19) followed by Victoria (14), Queensland (13), South Australia (9), Western Australia (3) and the Northern Territory (1). No cases were reported from the Australian Capital Territory or Tasmania during this period.

Age and sex distribution

Hepatitis D notifications in males exceeded those in females each year from 2005 to 2014. In 2014, 59% (35/59) of notifications were in males. This represented a male to female notification ratio of 1.5:1. This was less than the average notification ratio of 2.7:1 over the preceding 9 years (Figure 14).

Figure 14: Notifications of hepatitis D infection, Australia, 2005 to 2014, by year and sex



Gastrointestinal diseases

Overview

In 2014, gastrointestinal diseases notified to NNDSS and discussed in this section were: botulism, campylobacteriosis, cryptosporidiosis, haemolytic uraemic syndrome (HUS), hepatitis A, hepatitis E, listeriosis, salmonellosis, shigellosis, Shiga toxin-producing *Escherichia coli* (STEC) infections and typhoid fever.

Overall notified cases of gastrointestinal diseases increased by 24%, from to 32,535 in 2013 to 40,367 in 2014. Notifications for campylobacteriosis, salmonellosis, and shigellosis were at the highest levels since NNDSS records began in 1991. It should be noted that nucleic acid-based testing methods were introduced by a number of diagnostic laboratories around the country from late 2013 onwards. Whilst these tests may have increased sensitivity compared with traditional techniques, such as culture, the effect on notifications has not been quantified.

Surveillance systems overview

The Australian Government established OzFoodNet—Australia's enhanced foodborne disease surveillance system—in 2000 as a collaborative network of epidemiologists and microbiologists who conduct enhanced surveillance, epidemiological outbreak investigations and applied research into foodborne disease across Australia. OzFoodNet's mission is to apply concentrated effort at the national level to investigate and understand foodborne disease, to describe its epidemiology more effectively and to identify ways to minimise foodborne illness in Australia. The data and results summarised in the following sections will be reported in more detail in the OzFoodNet annual report 2014.

Botulism

- In 2014, there was 1 case of botulism notified to NNDSS.

Botulism is a rare but extremely serious intoxication resulting from toxins produced by *Clostridium botulinum* (commonly toxin types A, B and E). Four forms of botulism are recognised; infant, foodborne, wound and adult intestinal toxaemia.[22]

Epidemiological situation in 2014

There was 1 case of infant botulism notified by Queensland in 2014. *C. botulinum* toxin type B gene was detected in the stools by polymerase chain reaction (PCR) and toxin type B was confirmed using a mouse bioassay. No source of infection was identified.

Campylobacteriosis

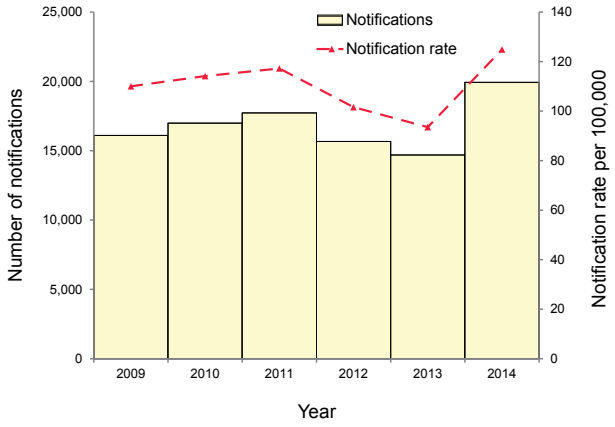
- In 2014, 19,931 cases of campylobacteriosis were notified to the NNDSS.
- Campylobacteriosis was the most frequently notified enteric infection in 2014.

The bacterium *Campylobacter* is a common cause of foodborne illness (campylobacteriosis) in humans. The severity of this illness varies and is characterised by diarrhoea (often bloody), abdominal pain, fever, nausea and or vomiting.[22] Campylobacteriosis is notifiable in all Australian states and territories except New South Wales.

Epidemiological situation in 2014

There were 19,931 notified cases of campylobacteriosis in 2014 making it the most frequently notified enteric infection (124.9 per 100,000 not including New South Wales). This was a 36% increase on the number of notifications received for 2013 (n=14,692) (Figure 15) and a 19% increase on the 5-year mean (n=16,237) (Table 6). The number of notified cases for 2014 was the highest recorded in NNDSS since 1991, and exceeded 2 standard deviations of the previous 5-year mean (2009 to 2013) by more than 1,300 notifications.

Figure 15: Notifications and notification rate for campylobacteriosis, Australia, 2009 to 2014, by year



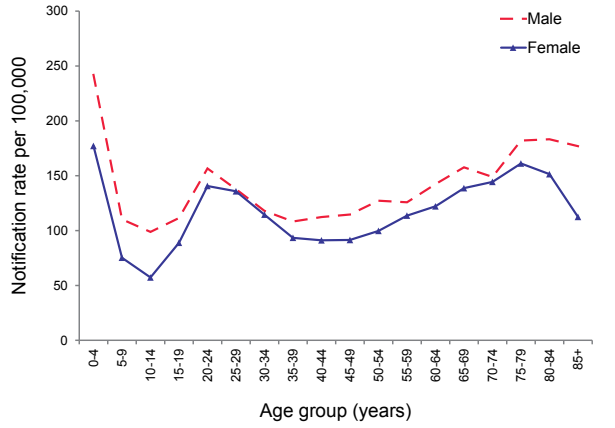
Geographical distribution

Notification rates ranged from 107.0 per 100,000 in South Australia to 181.5 per 100,000 in Tasmania; the Tasmanian rate was approximately 1.5 times higher than the national rate (124.9 per 100,000) (Table 5).

Age and sex distribution

Campylobacteriosis was most frequently notified among the 0–4 years age group for both males (241.9 per 100,000) and females (177.2 per 100,000). The median age of notified cases was 36 years (range 0 to 101 years) and 54% (10,811/19,896) where sex was known were male. Notification rates were highest among males in all age groups (Figure 16).

Figure 16: Notification rate for campylobacteriosis, Australia, 2014, by age group and sex (n=19.896)*



* Excludes notifications where age (n=12), sex (n=21) or both (n=2) were not reported.

Cryptosporidiosis

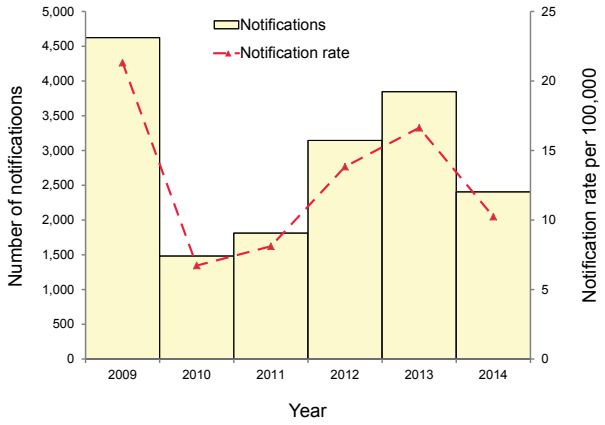
- In 2014, 2,405 cases of cryptosporidiosis were notified to the NNDSS.

Cryptosporidiosis is a parasitic infection characterised by abdominal cramping and usually large-volume watery diarrhoea. Ingesting contaminated water, typically from a recreational source like a community swimming pool or lake is a major risk factor for infection.[22]

Epidemiological situation in 2014

There were 2,405 notified cases of cryptosporidiosis in 2014 (10.2 per 100,000). This represents a 37% decrease on the number of notifications received for 2013 (n=3,846) and a 19% decrease on the 5-year mean (n=2,982) (Figure 17).

Figure 17: Notifications and notification rate for cryptosporidiosis, Australia, 2009 to 2014, by year



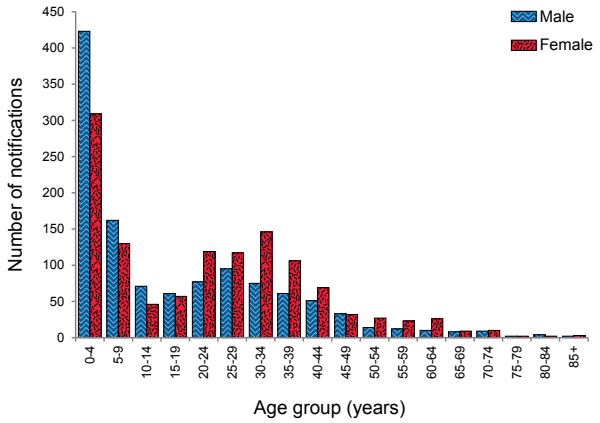
Geographical distribution

Notification rates ranged from 5.6 per 100,000 in New South Wales to 35.6 per 100,000 in the Northern Territory; the Northern Territory rate was 3.5 times higher than the national rate (10.2 per 100,000) (Table 5).

Age and sex distribution

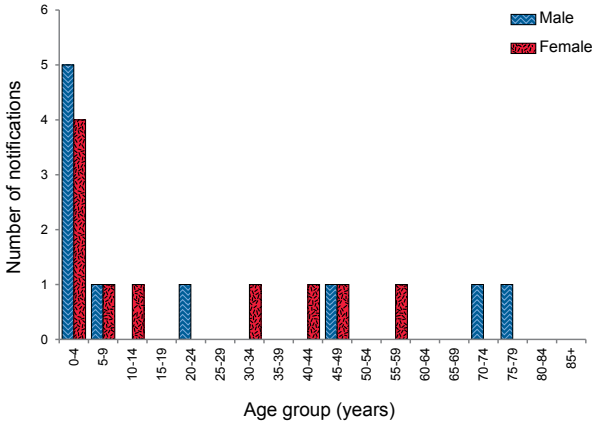
In 2014, notified cases for cryptosporidiosis, for which age was reported, were most frequent among the 0–4 years age group (31%, 732/2,403). The median age of notified cases was 18 years (range 0 to 92 years) and just over half (1,234/2,405) were female (Figure 18).

Figure 18: Notifications of cryptosporidiosis, Australia, 2014, by age group and sex



* Excludes notifications where age (n=2) was not reported.

Figure 19: Notifications of haemolytic uraemic syndrome, Australia, 2014, by age group and sex



Haemolytic uraemic syndrome

- In 2014, 20 cases of haemolytic uraemic syndrome notified to the NNDSS.
- Cases were most frequently notified among the 0–4 years age group.

HUS is a rare but serious illness that is characterised by acute renal impairment; with 50% of patients requiring dialysis and approximately 5% dying.[22] Not all diagnoses of HUS are related to enteric pathogens, but Australian cases are commonly associated with STEC infection.[36] In 2013, 68% (10/15) of notified HUS cases were positive for STEC.[37]

Epidemiological situation in 2014

There were 20 notified cases of HUS in 2014 compared with 15 in 2013 and a mean of 14 cases per year between 2009 and 2013.

Geographical distribution

Over half (55%, 11) of notifications were in residents from New South Wales (n=6) and Victoria (n=5).

Age and sex distribution

In 2014, HUS was most frequently notified among the 0–4 years age group (45%, 9) (Figure 19). Half of notified cases were in males (n=10).

Hepatitis A

- In 2014, 231 cases of hepatitis A infection notified to the NNDSS.
- Overseas travel was the primary risk factor for notified cases.

Hepatitis A is an acute viral infection primarily of the liver, characterised by fever, malaise, anorexia, nausea and abdominal discomfort followed by jaundice. The disease varies from a mild illness to a severely disabling disease lasting several months. Infection is usually spread from person to person via the faecal-oral route but can also be foodborne or waterborne.[22]

Epidemiological situation in 2014

There were 231 notified cases of hepatitis A infection in 2014 (1.0 per 100,000). This was a 22% increase on the number of notified cases in 2013 (n=190), and a 13% decrease on the 5-year mean (n=266). The historical mean reflects the impact of a 2009–2010 outbreak of hepatitis A associated with the consumption of semi-dried tomatoes (Figure 20).[38]

Geographical distribution

Two-thirds (66%, 153/231) of notifications were in residents from New South Wales (n=83) and Victoria (n=70).

Age and sex distribution

Hepatitis A infection was most frequently notified among the 5–9 years age group (14%, 32) in 2014 (Figure 21). The median age of notified cases was 23 years (range 1 to 76 years), and 58% (134) of all cases were male.

Indigenous status

Indigenous status was known for 96% (222) of notified cases of hepatitis A. Of these, 4 were identified as being Indigenous.

Figure 20: Notifications and notification rate for hepatitis A infection, Australia, 2009 to 2014, by year

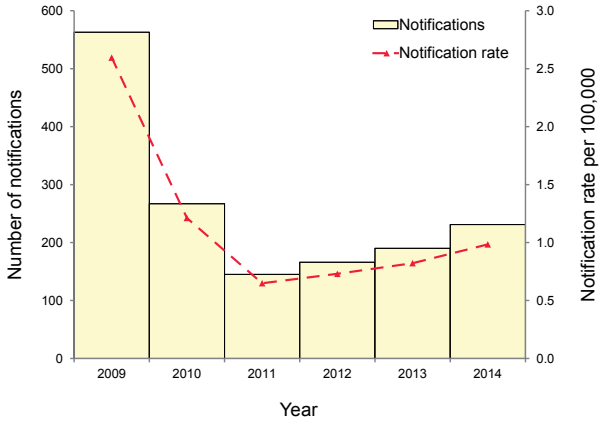
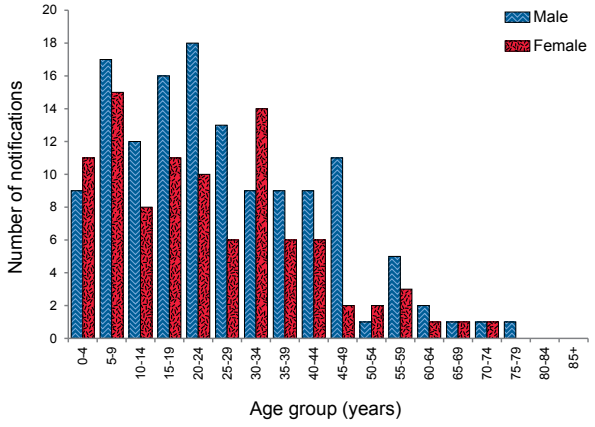


Figure 21: Notifications of hepatitis A infection, Australia, 2014, by age group and sex



Place of acquisition

Overseas travel was the primary risk factor for notified cases (Table 12). In 2014, 80% (184/231) reported overseas travel during their incubation period for hepatitis A infection and were considered to have been overseas acquired. The top 5 countries of acquisition were Fiji (n=30), the Philippines (n=24), India (n=22), Pakistan (n=18) and Indonesia (n=12).

In 2014, 19% (44) of notified cases were locally acquired. This was similar to 2012 where 24% (46/190) of notified cases were locally acquired (Table 12). A 2009–2010 outbreak associated with the consumption of semi-dried tomatoes contributed to an increase in locally acquired hepatitis A cases in those years.[38] Place of acquisition was unknown or not recorded for 3 notified cases.

Hepatitis E

- In 2014, 56 cases of hepatitis E infection notified to the NNDSS.

Hepatitis E infection is an acute viral infection primarily of the liver that is transmitted by the faecal-oral route, most often via food or water.[22] The infection is usually acquired overseas among travellers to endemic areas.

Epidemiological situation in 2014

There were 56 notified cases of hepatitis E infection in 2014 (0.2 per 100,000). This was a 65% increase on the number of notified cases in 2013 (n=34), and a 60% increase on the 5-year mean (n=35).

Geographical distribution

The majority of notifications were in residents from New South Wales (n=37).

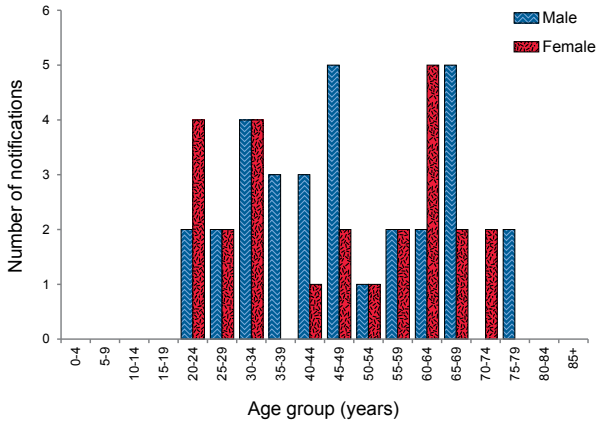
Table 12: Notifications of hepatitis A, Australia, 2009 to 2014, by place of acquisition

	Locally acquired		Overseas acquired		Unknown		
Year	n	%	n	%	n	%	Total
2009	373	66	139	25	51	9	563
2010	113	42	147	55	7	3	267
2011	41	28	103	71	1	1	145
2012	34	20	122	73	10	6	166
2013	46	24	134	71	10	5	190
2014	44	19	184	80	3	1	231

Age and sex distribution

Hepatitis E infection was most frequently notified among the 30–34 years age group (14%, 8) (Figure 22). In 2014, the median age of notified cases was 47 years (range 21 to 77 years), and 55% (31) were male.

Figure 22: Notifications of hepatitis E infection, Australia, 2014, by age group and sex



Place of acquisition

Hepatitis E in Australia has traditionally been associated with overseas travel. In 2014, 52% of cases (29) reported overseas travel during their incubation period and were considered to have been acquired overseas. Of these, 38% (11/29) reported travel to India. The place of acquisition was unknown for 6 notified cases.

In 2014, 38% (21) of cases were locally acquired, with the majority of these reported in New South Wales residents (n=20). The large number of notified cases among residents from New South Wales can be attributed to a cluster of hepatitis E infection associated with consumption of pork liver pâté at a specific restaurant in that state.[39] This was the first documented locally acquired outbreak of hepatitis E in Australia.

Listeriosis

- In 2014, 80 cases of listeriosis notified to the NNDSS.
- Notifications were highest in the 80+ year age group.

Invasive listeriosis is caused by a bacterial infection that commonly affects the elderly or immunocompromised, and typically occurs among people with serious underlying illnesses. Listeriosis can also affect pregnant women and infect their unborn baby.[40] Laboratory-confirmed infections in a mother and her unborn child or neonate are notified separately in the NNDSS.

Epidemiological situation in 2014

There were 80 notified cases of listeriosis in 2014 (0.3 per 100,000), which was a slight increase on the number of notified cases in 2013 (n=76) and the same as the 5-year mean (n=80).

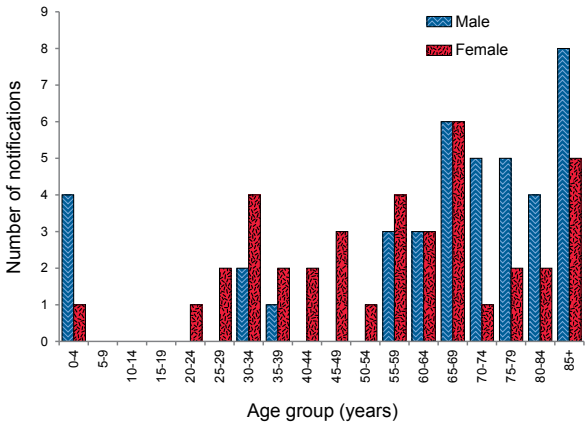
Geographical distribution

Over half (56%, 45) of notifications were in residents from New South Wales (n=23) and Victoria (n=22).

Age and sex distribution

Notifications of listeriosis were highest in the 80 years or over age group (16%, 13/80) (Figure 23), with just over half (51%, 41) of all notified cases being male.

Figure 23: Notifications of listeriosis, Australia, 2014, by age group and sex



Enhanced surveillance datasets

In 2010, OzFoodNet started collecting enhanced surveillance data on all notified cases of listeriosis in Australia. The information collected on cases includes laboratory data collected from the characterisation of *Listeria monocytogenes* isolates by molecular subtyping methods, and epidemiological data, which includes food consumption histories and clinical data. The overall aim of this enhanced surveillance is to enable timely detection of outbreaks and subsequent public health response.[41] Further information on OzFoodNet's National

Enhanced Listeriosis Surveillance System can be found in OzFoodNet annual reports (http://www.ozfoodnet.gov.au/internet/ozfoodnet/publishing.nsf/Content/reports-1).

Salmonellosis (non-typhoidal)

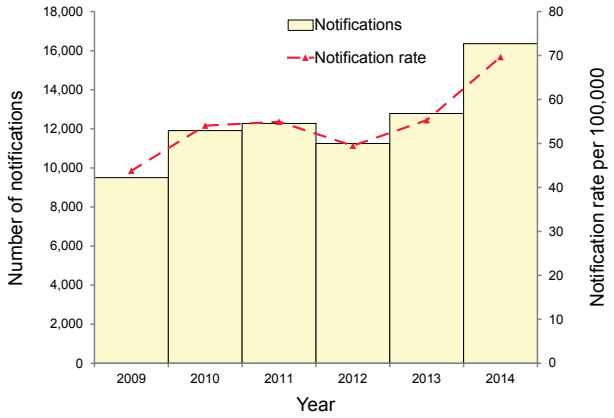
- In 2014, 16,358 cases of salmonellosis notified to the NNDSS.
- This was the highest number of notifications recorded in NNDSS since 1991.

Salmonellosis is a bacterial disease characterised by the rapid development of symptoms including abdominal pain, fever, diarrhoea, muscle pain, nausea and/or vomiting. People can become infected via faecal-oral transmission, ingesting contaminated food, through animal contact and from environmental exposures. The predominant mode of transmission is contaminated food, mainly of animal origin.[22]

Epidemiological situation in 2014

There were 16,358 notified cases of salmonellosis in 2014 (69.7 per 100,000). This was a 28% increase on the number of cases reported in 2013 (n=12,785) (Figure 24), and a 42% increase on the 5-year mean (n=11,545). The number of cases for 2014 was the highest recorded in NNDSS since 1991 when this disease became nationally notifiable, beating the previous record in 2013. Additionally, notified cases in 2014 exceeded 2 standard deviations of the previous 5-year mean (2009 to 2013) by more than 2,200 notifications.

Figure 24: Notifications and notification rate for salmonellosis, Australia, 2009 to 2014, by year



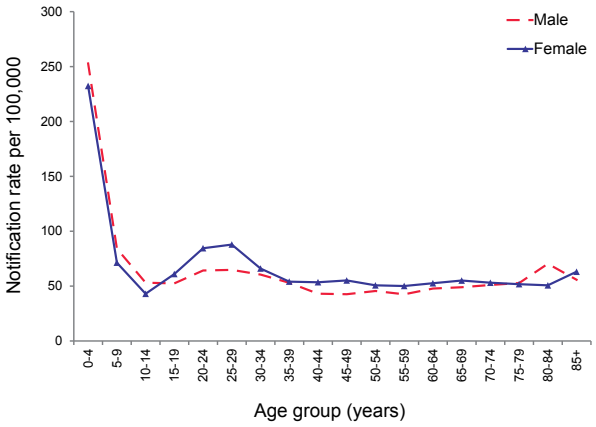
Geographical distribution

Notification rates ranged from 48.4 per 100,000 in Tasmania to 186.8 per 100,000 in the Northern Territory (Table 5).

Age and sex distribution

Salmonellosis was most frequently notified among the 0–4 years age group (23%, 3,709/16,355) (Figure 25) where age was recorded, with an age-specific rate of 210.4 per 100,000 population. The median age of notified cases was 27 years (range 0 to 102 years) and just over half (51%, 8,393/16,333) of cases where sex was recorded were female.

Figure 25: Notification rate for salmonellosis, Australia, 2014, by age group and sex (n=16,332)*



* Excludes notifications where age (n=1), sex (n=23) or both (n=2) were not reported.

Shigellosis

- In 2014, 1,051 notified cases of shigellosis to the NNDSS.
- Increase in notifications possibly associated with increased in culture-independent diagnostic testing.

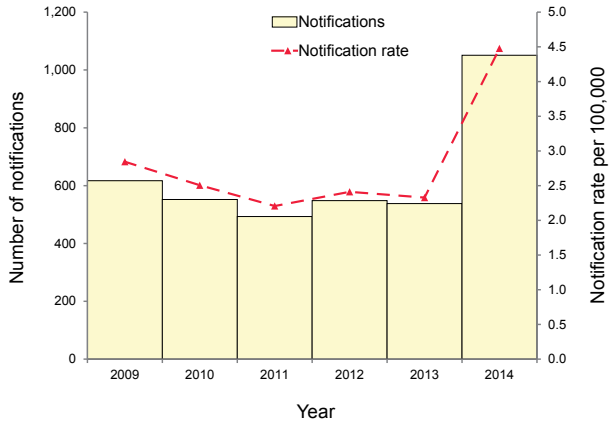
Shigellosis is a bacterial disease characterised by acute abdominal pain and fever, small-volume loose stools, vomiting and tenesmus. *Shigella* is transmitted via the faecal-oral route, either directly (such as male-to-male sexual contact) or indirectly through contaminated food or water.[22]

Epidemiological situation in 2014

There were 1,051 notified cases of shigellosis in 2014 (4.5 per 100,000). This was a 95% increase on

the number of cases in 2013 (n=538) (Figure 26), and a 91% increase on the 5-year mean (n=550). This increase may be associated with the increased use of culture-independent diagnostic testing (CIDT). The current CIDT methods are unable to differentiate between infection with *Shigella,* which is notifiable, and entero-invasive *Escherichia coli,* which is not.[42]

Figure 26: Notifications and notification rate for shigellosis, Australia, 2009 to 2014, by year



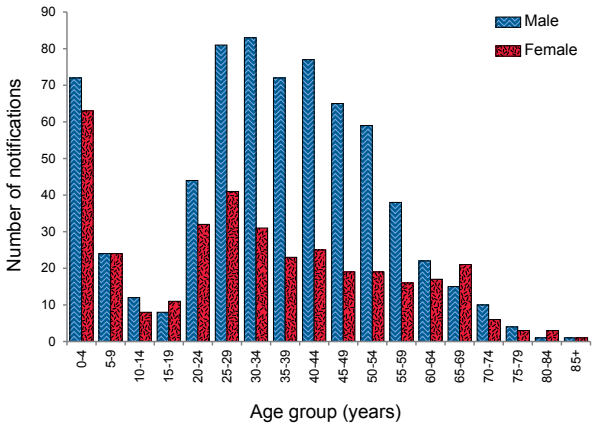
Geographical distribution

Notification rates ranged from 0.4 per 100,000 in Tasmania to 40.5 per 100,000 in the Northern Territory. State and territory rates for 2014 should be interpreted with caution as some jurisdictions require CIDT-positive samples to be confirmed by culture whilst others do not.

Age and sex distribution

Notifications for shigellosis were highest in the 0–4 year age group (13%, 135) (Figure 27). In 2014, the median age of notified cases was 34 years (range 0 to 94 years) and almost two-thirds (65%, 688) were male.

Figure 27: Notifications of shigellosis, Australia, 2014, by age group and sex



Indigenous status

Information on Indigenous status was available for 81% (853) of shigellosis cases. This proportion varied by state or territory, with Queensland being the only jurisdiction with less than 80% data completeness. Among states and territories with greater than or equal to 80% completeness, the proportion of notified cases who identified as being of Aboriginal and/or Torres Strait Islander origin was 11% (98/875).

Place of acquisition

Thirty-two per cent (333) of notified cases of shigellosis were reported as being acquired overseas. The top 5 countries of acquisition were Indonesia (n=86), India (n=54), Thailand (n=21), Vietnam (n=20) and Cambodia (n=17). The place of acquisition was inadequately described or unknown for half of notifications (51% 530) (Table 13).

Table 13: Notifications of shigellosis, Australia, 2009 to 2014, by place of acquisition

	Locally acquired		Overseas acquired		Unknown		
Year	n	%	n	%	n	%	Total
2009	227	37	83	13	307	50	617
2010	164	30	191	35	197	36	552
2011	152	31	133	27	208	42	493
2012	141	26	174	32	233	43	548
2013	137	25	209	39	192	36	538
2014	188	18	333	32	530	50	1,051

Shiga toxin-producing *Escherichia coli*

- In 2014, 116 notified cases of Shiga toxin-producing *Escherichia coli* infection to the NNDSS.

Shiga toxin-producing *Escherichia coli* is a common cause of diarrhoeal illness in humans. People can become infected via faecal-oral transmission, ingesting contaminated food, through animal contact and from environmental exposures. Severe illness can progress to HUS. Children under 5 years of age are most frequently diagnosed with infection and are at greatest risk of developing HUS.[22]

Epidemiological situation in 2014

There were 115 notified cases of STEC in 2014 (0.5 per 100,000). This was a 36% decrease on the number of cases in 2013 (n=180) and similar to the 5-year mean (n=119). A large outbreak (n=57) of STEC infection associated with a Queensland agricultural show contributed to the high number of notifications seen in 2013.[43]

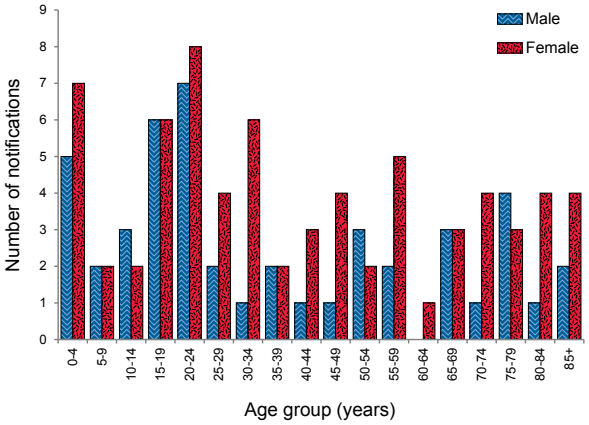
Geographical distribution

Detection of STEC infection is strongly influenced by jurisdictional practices regarding the screening of stool specimens.[41] South Australia continues to test all bloody stools for STEC using PCR and subsequently has the highest notification rate in the country; 2.7 cases per 100,000 compared with between 0.1 and 0.6 cases per 100,000 in other states and territories reporting cases. Additionally, South Australia ceased routinely culturing PCR positive STEC samples in 2014. The differences in testing practices among states and territories render comparison of notification data by jurisdiction and over time invalid.

Age and sex distribution

Notifications of STEC were highest in the 20–24 years age group (13%, 15/115) (Figure 28). In 2014, the median age of notified cases was 33 years (range 0 to 88 years) and 60% (69) of notified cases were female.

Figure 28: Number of notifications of Shiga toxin-producing *Escherichia coli* infection, Australia, 2014, by age group and sex



Typhoid fever

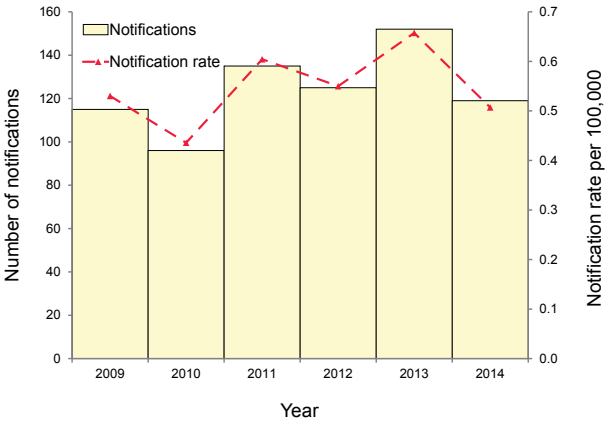
- In 2014, 119 notified cases of typhoid to the NNDSS.
- 92% of cases were acquired overseas.

Typhoid is a bacterial disease caused by *Salmonella enterica* serotype Typhi. Symptoms include sustained fever, marked headache, malaise and constipation more often than diarrhoea in adults. The transmission mode is the same as for salmonellosis, however, typhoid differs in that humans are the reservoir for the bacterium.[22]

Epidemiological situation in 2014

There were 119 notified cases of typhoid in 2014 (0.5 per 100,000). This was a 22% decrease on the number of cases in 2013 (n=152) (Figure 29) and similar to the 5-year mean (n=125).

Figure 29: Notifications and notification rate for typhoid, Australia, 2009 to 2014, by year



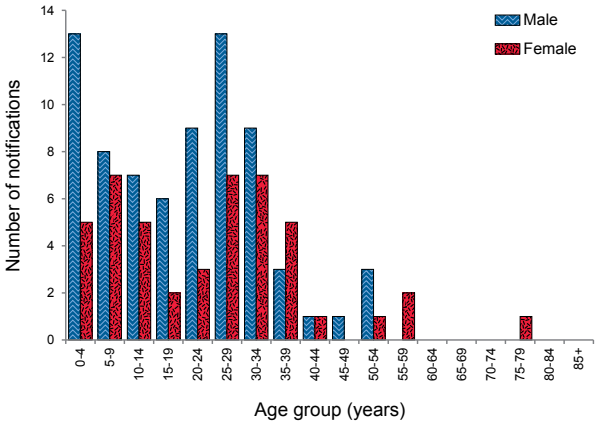
Geographical distribution

Almost two-thirds (62%, 74) of notifications were in residents from New South Wales (n=45) and Victoria (n=29).

Age and sex distribution

Typhoid was most frequently notified among the 20–24 years age group (17%, 20) (Figure 30). The median age of notified cases was 22 years (range 1 to 77 years), and 61% (73) were male.

Figure 30: Notifications of typhoid, Australia, 2014, by age group and sex



Place of acquisition

As in previous years, overseas travel was the primary risk factor for notified cases. In 2014, 92% (109) reported overseas travel during their exposure period and were considered overseas acquired. India continues to be the most frequently reported country of acquisition, accounting for 56% (61/109) of overseas-acquired cases in 2014. Six cases were listed as locally acquired and the place of acquisition was unknown for 4 cases (Table 14).

Quarantinable diseases

Human diseases covered by the *Quarantine Act 1908,* and notifiable in Australia and to the WHO in 2014 were cholera, plague, rabies, yellow fever, smallpox, highly pathogenic avian influenza in humans (HPAIH), severe acute respiratory syndrome (SARS) and 4 viral haemorrhagic fevers (Ebola, Marburg, Lassa and Crimean–Congo). These diseases are of international public health significance.

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

There were no cases of plague, rabies, smallpox, SARS, HPAIH or viral haemorrhagic fevers reported in Australia in 2014. While there were 2 cases of overseas-acquired cholera in 2014, Australia remains free of all the listed quarantinable diseases (Table 15).

Cholera

- In 2014, 2 cases of cholera notified to the NNDSS.

Cholera is an infection of the digestive tract (or gut) caused by certain strains of the bacterium *Vibrio cholerae* that produce toxins (poisons) and is most commonly acquired in parts of Africa, Asia, South America, the Middle East and the Pacific islands. *V. cholerae* is found in the faeces of infected people, and is spread by drinking contaminated water, eating food washed with contaminated water or prepared with soiled hands or eating fish or shellfish caught in contaminated water. Person-to-person spread of cholera is less common. Most people do not develop symptoms or have only mild illness but a small proportion of people will develop severe symptoms. Symptoms typically start between 2 hours and 5 days (usually 2 to 3 days) after ingesting the bacteria. Symptoms can include characteristic 'rice water' faeces (profuse, watery diarrhoea), nausea and vomiting, signs of dehydration, such as weakness, lethargy and muscle cramps. Only toxigenic *V. cholerae* o1 or o139 are notifiable in Australia.

Table 14: Notifications of typhoid, Australia, 2009 to 2014, by place of acquisition

	Locally acquired		Overseas acquired		Unknown		
Year	n	%	n	%	n	%	Total
2009	15	13	82	71	18	16	115
2010	2	2	92	96	2	2	96
2011	6	4	125	93	4	3	135
2012	9	7	109	87	7	6	125
2013	8	5	141	93	3	2	152
2014	6	5	109	92	4	3	119

Table 15: Australia's status for human quarantinable diseases, 2014

Disease	Status	Date of last record and notes
Cholera	Free	Small number of cases reported annually related to overseas travel. Very rare instances of local acquisition as described under the section 'Cholera'.
Plague	Free	Last case recorded in Australia in 1923[44]
Rabies	Free	Last case (overseas acquired) recorded in Australia in 1990[45]
Smallpox	Free	Last case recorded in Australia in 1938, last case world-wide in 1977, declared eradicated by the World Health Organization 1980[46,47]
Yellow fever	Free	Two cases in 2011 were the first recorded, related to overseas travel[37]
SARS	Free	Last case recorded in Australia in 2003[48]
HPAIH	Free	No cases recorded[49]
Viral haemorrhagic fevers		
Ebola	Free	No cases recorded
Marburg	Free	No cases recorded
Lassa	Free	No cases recorded
Crimean–Congo	Free	No cases recorded

Epidemiological situation in 2014

In 2014, there were 2 notifications of cholera in Australia. The following details are available about the relevant exposures or place of acquisition for the 2 cases in 2014:

- Case 1 was a 1-year-old female who acquired the infection whilst travelling in India;
- Case 2 was a 63-year-old female who was an international visitor and had acquired the infection in India;
- These cases both notified by Victoria, but were not known to have been linked.

There were 21 cases of cholera in total in Australia between 2009 and 2013. All cases of cholera reported since the commencement of the NNDSS in 1991 to 2013 have been acquired outside Australia except for 1 case of laboratory-acquired cholera in 1996,[50] 3 cases in 2006 linked to imported whitebait[51] and 1 laboratory-acquired case in 2013.[37]

Sexually transmissible infections

In 2014, the STIs reported to the NNDSS were chlamydial infection, donovanosis, gonococcal infection, and congenital and non-congenital syphilis. Other national surveillance systems that monitor STIs in Australia include the Australian Gonococcal Surveillance Programme (AGSP), which is a network of specialist laboratories monitoring antimicrobial susceptibility patterns of gonococcal infection; and the Kirby Institute.

Chlamydial infection

- In 2014, 86,108 cases of chlamydial infection were notified to the NNDSS.
- Notification rates have remained relatively stable from 2011.
- Almost 40% of notifications were among females aged 15–24 years.

Genital chlamydial infection is caused by the bacterium *Chlamydia trachomatis* serogroups D–K. Screening is important in detecting chlamydial infections, as a large proportion of infections are asymptomatic. Chlamydial infection is highly treatable, although reinfection is common.[52] If left untreated, complications such as epididymitis in males and infertility and pelvic inflammatory disease in females can arise.[22]

Epidemiological situation in 2014

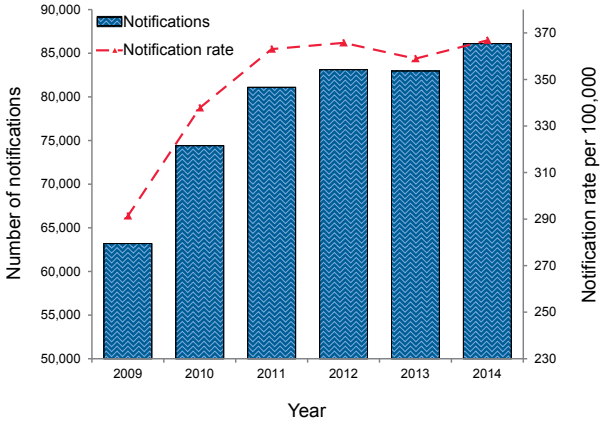
In 2014, chlamydial infection was the most frequently notified disease to the NNDSS, with 86,108 cases, representing 31% of all notifications reported to the NNDSS in 2014. Since 2011, notification rates have remained relatively stable, increasing marginally from 363.0 per 100,000 in 2011 to 366.8 per 100,000 in 2014. This follows a 25% increase in notification rates from 2009 to 2011 (291.4 to 363.0 per 100,000 respectively) (Figure 31).

Geographical distribution

In 2014, the notification rate for chlamydial infection was more than 3 times higher in the Northern Territory (1,225.2 per 100,000) than the overall

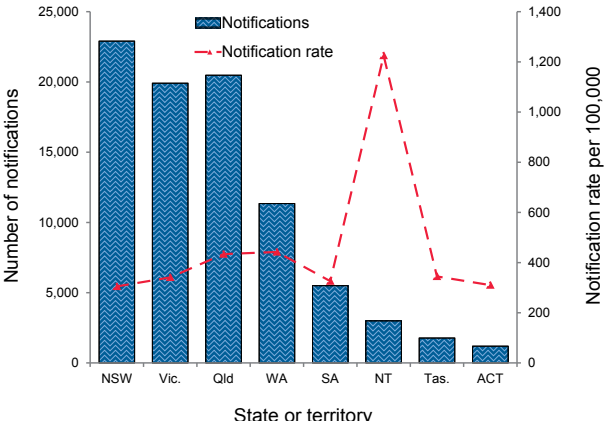
national rate (366.8 per 100,000) (Figure 32). This is mostly explained by the ongoing disproportion of young Aboriginal and Torres Strait Islander women affected by chlamydial infection, particularly those living in regional and remote areas (Table 5).[9]

Figure 31: Notifications and notification rate for chlamydial infection,* Australia, 2009 to 2014, by year



* Excludes notifications where the case was aged less than 13 years.

Figure 32: Notifications and notification rate for chlamydial infection, Australia, 2014, by state or territory

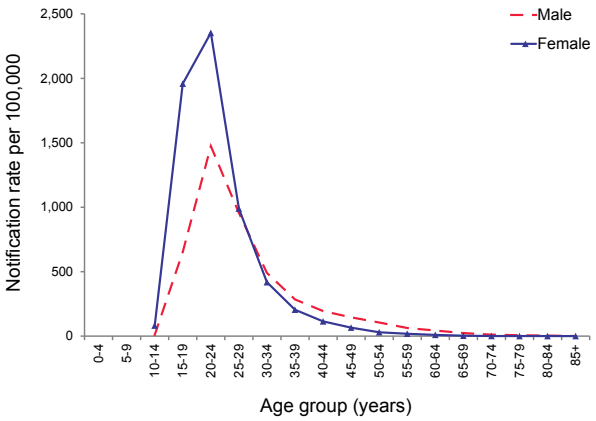


* Excludes notifications where the case was aged less than 13 years.

Age and sex distribution

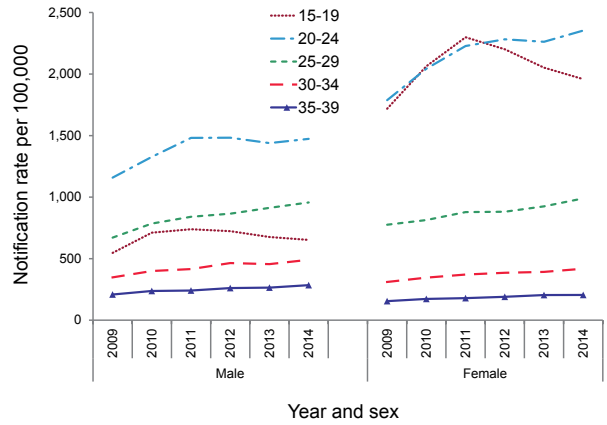
In 2014, chlamydial infection occurred predominately in females aged 15–24 years, accounting for 38% of all chlamydial infections (Figure 33). Similar to 2013, the national notification rate for chlamydial infection in 2014 was 314.8 per 100,000 in males and 417.5 per 100,000 in females. The overall higher rate among females may be partly attributable to preferential testing of women attending health services compared with men.[9,33] Notification rates for males and females increased overall from 2009 to 2014, by 31% (239.7 to 314.8) in males and 22% (340.9 to 417.5) in females, and across most age groups; however, rates decreased from 2011 and 2014 for females aged 15–19 years (Figure 34).

Figure 33: Notification rate for chlamydial infection, Australia, 2014, by age group and sex*



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

Figure 34: Notification rate for chlamydial infection, Australia, 2009 to 2014, by year, sex* and selected age groups



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

Indigenous status

The completeness of Indigenous status identification for chlamydial infection notification data var-

ies by year and by jurisdiction. Nationally in 2014, data on Indigenous status were complete for 37% (31,990) of chlamydial infection notifications, which was lower than the preceding 5-year average of 48% (range: 39%–51%). Four jurisdictions had greater than 50% completeness of the Indigenous status field in each year during the 2009–2014 period: the Northern Territory, Queensland, South Australia, and Western Australia. Among these jurisdictions, the combined age-standardised notification rate ratio between Indigenous and non-Indigenous populations in 2014 was 3.5:1, which was similar to the previous 5 years (range: 3.4–3.7).

Among the Indigenous population, the age-standardised notification rate declined from 1,378.8 per 100,000 in 2011 to 1,266.4 per 100,000 in 2014. This followed an increase from 1,112.4 per 100,000 in 2009 to 1,332.2 per 100,000 in 2011.

Age-standardised notification rates among the non-Indigenous population increased overall from 313.1 per 100,000 in 2009 to 369.9 per 100,000 in 2014. The rate increased each year except for a small decline from 358.5 per 100,000 in 2011 to 355.2 per 100,000 in 2012.

Between 2013 and 2014, age-standardised notification rates for chlamydial infection in the Indigenous population decreased by 7% in both Queensland (1,271.0 to 1,179.8) and Western Australia (1,433.8 to 1,327.8), and by 2% (1,906.6 to 1,863.6) in the Northern Territory. Conversely, rates increased in South Australia by 17% (871.4 to 1,020.6).

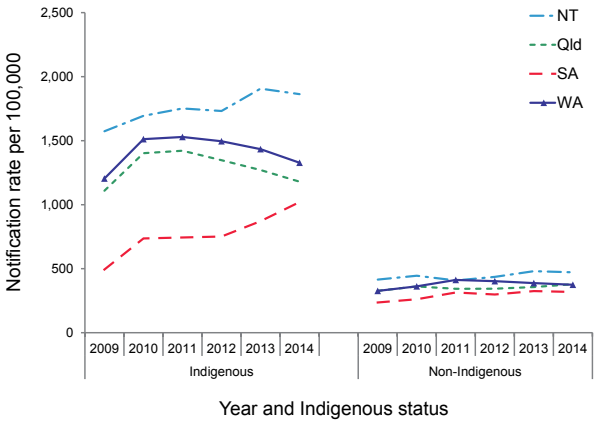
Between 2013 and 2014, age-standardised notification rates for chlamydial infection in the non-Indigenous population decreased by 3% (387.8 to 374.3) in Western Australia, and by 2% in both South Australia (325.1 to 318.5) and the Northern Territory (481.0 to 472.0). Conversely, rates increased in Queensland by 6% (356.8 to 378.7) (Figure 35).

Donovanosis

- In 2014, 1 case of donovanosis was notified to the NNDSS.
- This disease remains rare in Australia.

Donovanosis, caused by the bacterium *Klebsiella granulomatis*, is a chronic, progressively destructive infection that is primarily transmitted through sexual exposure. It affects the skin and mucous membranes of the external genitalia, inguinal and anal regions.[53] Once diagnosed, donovanosis is treated with a series of antibiotics.[54]

Figure 35: Age standardised notification rates for chlamydial infection, selected states and territories,* 2009 to 2014, by year and Indigenous status

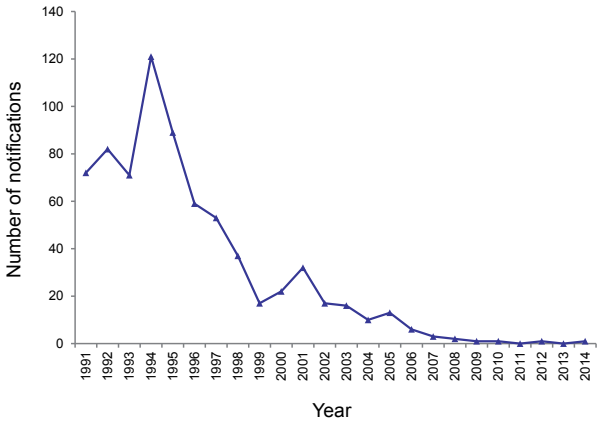


* Includes the states and territories where Indigenous status was reported for more than 50% of cases between 2009 and 2014: the Northern Territory, Queensland, South Australia and Western Australia.

All donovanosis notifications in Australia since 1991 were reported either in the Northern Territory, Western Australia or Queensland and have predominately occurred in Aboriginal and Torres Strait Islander people living in remote areas in northern and central Australia.

Donovanosis was targeted for elimination in Australia through the National Donovanosis Elimination Project 2001–2004.[55] It is now rare, with fewer than 17 cases notified each year since 2002, and fewer than 5 cases notified each year since 2007 (Figure 36).

Figure 36: Notified cases of donovanosis,* Australia, 1991 to 2014, by year



Epidemiological situation in 2014

In 2014, 1 case of donovanosis was notified in Australia, in an Indigenous female from Western Australia (Figure 36).

Gonococcal infection

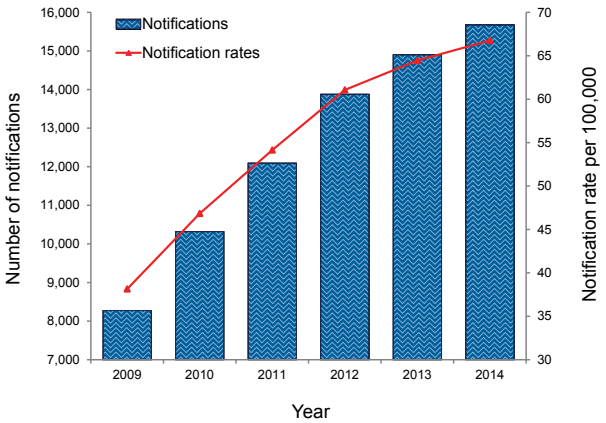
- In 2014, 15,675 cases of gonococcal infection were notified to the NNDSS.
- Notification rates for gonococcal infection continued to increase.
- Notifications occurred predominately in males aged 15–39 years and females aged 15–24 years.

Gonococcal infection is caused by the bacterium *Neisseria gonorrhoeae,* which affects the mucous membranes causing symptomatic and asymptomatic genital and extra-genital tract infections. The most common source of transmission is via unprotected sexual intercourse with an infected person.[22] If left untreated, it can lead to pelvic inflammatory disease in women and infertility in both men and women. Gonococcal infection also increases the risk of both acquisition and transmission of HIV. [53]

Epidemiological situation in 2014

In 2014, there were 15,675 cases of gonococcal infection reported to the NNDSS, a notification rate of 66.8 per 100,000. This was a 4% increase compared with the rate reported in 2013 (64.5 per 100,000). In the past 6 years, gonococcal infection notification rates increased, on average, 12% each year since 2009 (range: 4%–23%). Overall, gonococcal infection notification rates increased by 75% from 2009 (38.1 per 100,000) to 2014 (66.8 per 100,000) (Figure 37).

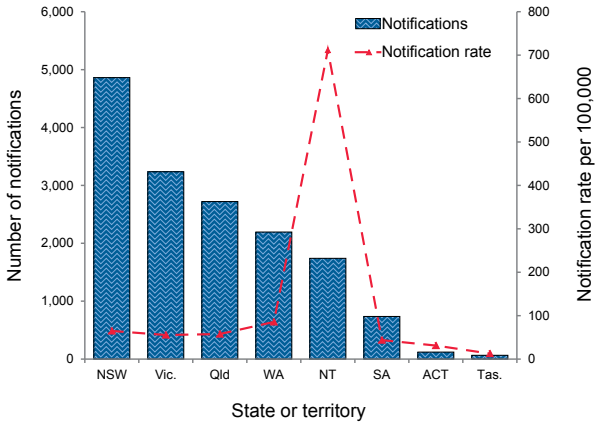
Figure 37: Notifications and notification rate for gonococcal infection, Australia, 2009 to 2014, by year



Geographical distribution

In 2014, the notification rate for gonococcal infection was almost 11 times higher in the Northern Territory (711.7 per 100,000) than the overall national rate (66.8 per 100,000) (Figure 38).

Figure 38: Notifications and notification rate for gonococcal infection, Australia, 2014, by state or territory



Age and sex distribution

Nationally in 2014, the notification rate for gonococcal infection was 97.9 per 100,000 in males and 35.3 per 100,000 in females. Notification rates in males increased by 8% and decreased in females by 7% when compared with 2013 (91.0 and 37.8 per 100,000 respectively). In 2014, 50% of notifications occurred in males in the 20–39 years age group. Notification rates in males exceeded those in females across all age groups above 20 years (Figure 39). This was consistent with previous years where, with the exception of Indigenous persons, notifications were largely reported in men who have sex with men (MSM).[56]

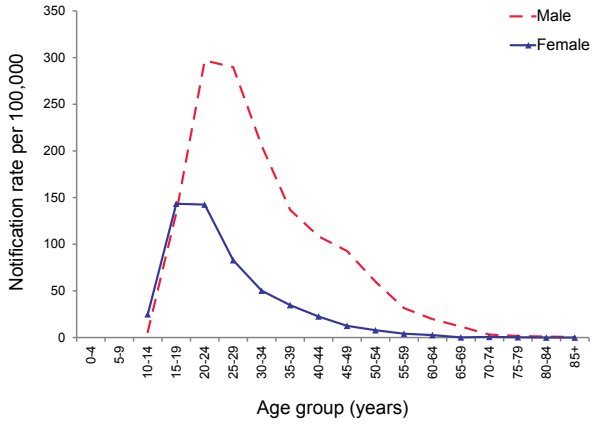
From 2009 to 2014, notification rates of gonococcal infection increased annually for males aged 20–39 and 45–49 years. The biggest overall increase was seen in the 45–49 years age group, with notification rates increasing by 171% (from 34.3 to 92.8 per 100,000), followed by the 30–34 years age group, with notification rates increasing by 131% (from 89.0 to 205.6 per 100,000). Compared with males, female rates were lower overall, peaking in 2011–12 in the 15–19 years age group, followed by a decline from 2012 to 2014 (Figure 40).

Indigenous status

The completeness of Indigenous status identification in the notification data varies by year and by

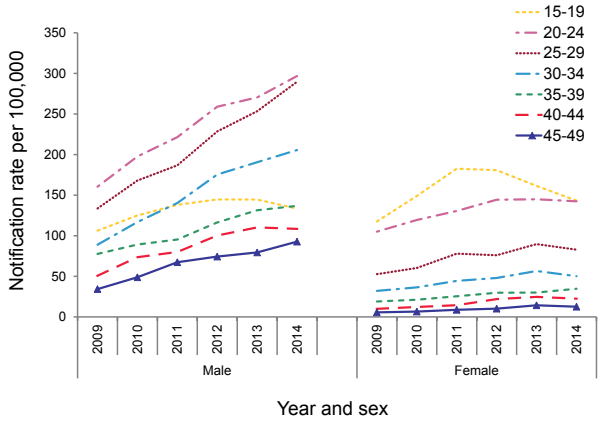
jurisdiction. Nationally in 2014, data on Indigenous status were complete for 66% of notifications, which was similar to the preceding 5-year mean of 68% (range: 66% to 72%). All states and territories except New South Wales had greater than 50% completeness of the Indigenous status field across the 2009 to 2014 period. Among the states and territories with greater than 50% completeness, the combined age-standardised notification rate ratio between Indigenous and non-Indigenous populations in 2014 was 26.5:1, increasing from 18.4:1 in 2013. Overall, the rate ratio has declined by 6% from 2009 to 2014 (from 28.1:1 to 26.5:1).

Figure 39: Notification rate for gonococcal infection, Australia, 2014, by age group and sex*



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

Figure 40: Notification rate for gonococcal infection, Australia, 2009 to 2014, by year, sex and selected age groups*



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

Among the Indigenous population, the age-standardised notification rate decreased by 25% from 2013 to 2014 (from 770.3 to 577.3 per 100,000) and the age-standardised Indigenous rate in 2014 (577.3 per 100,000) was 12% lower than in 2009 (659.7 per 100,000).

Among the non-Indigenous population, the age-standardised notification rate has decreased by 6% from 2009 to 2014 (28.1 and 26.5 per 100,000 respectively).

From 2009 to 2014, notification rates decreased in all states and territories in which Indigenous status was more than 50% complete except Tasmania, (which stabilised following a brief increase, from 4.6 to 4.9 per 100,000) (Figure 41).

Microbiological trends

The AGSP is the national surveillance system for monitoring the antimicrobial resistance of *N. gonorrhoeae* isolates. These results are published in more detail in the AGSP annual report in CDI.[57]

In 2014, the AGSP reported that a total of 4,804 gonococcal isolates were referred for antibiotic susceptibility testing, representing 31% of gonococcal infections notified to the NNDSS. This was slightly lower than the proportion of NNDSS cases tested in 2013 (33%, 4,896/14,902).

Eighty-three per cent of the isolates (n=4,009) were from males and 17% (n=791) were from females (M:F, 5.1:1). There were 4 isolates for which gender was unknown. The proportion of gonococcal isolates from males and females tested by the AGSP has remained stable over recent years.

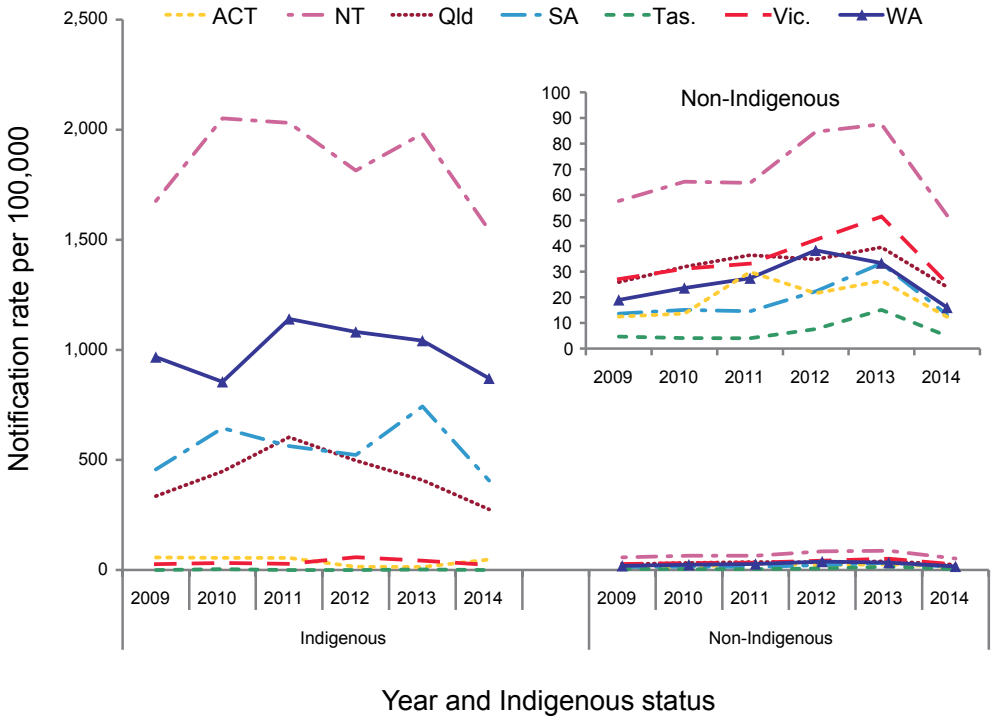
Syphilis (non-congenital categories)

- In 2014, 3,930 cases of syphilis (non-congenital categories) were notified to the NNDSS, a rate of 16.8 per 100,000.
- Cases of non-congenital syphilis were more frequently reported in MSM.

Syphilis is a sexually transmitted infection caused by the bacterium *Treponema palladium*. Infection is characterised by a primary lesion, a secondary eruption involving skin and mucous membranes, long periods of latency and late lesions of skin, bone, viscera, cardiovascular and nervous systems.[22]

In 2004, all jurisdictions except South Australia began reporting non-congenital syphilis infections to the NNDSS separately categorised as: infectious syphilis (primary, secondary or early latent) of less

Figure 41: Age-standardised notification rates for gonococcal infection, selected states and territories,* 2009 to 2014, by Indigenous status and year. Inset: Non-Indigenous notification rates



* Includes the states and territories where Indigenous status was reported for more than 50% of cases between 2009 and 2014: The Australian Capital Territory, the Northern Territory, Queensland, South Australia, Tasmania, Victoria, and Western Australia.

than 2 years duration; and syphilis of more than 2 years or unknown duration. From 2004 to 2011, South Australia reported only cases of infectious syphilis, and then commenced reporting syphilis of more than 2 years or unknown duration in 2012.

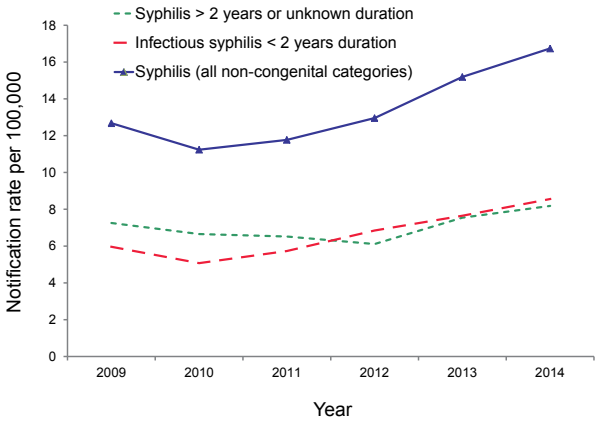
Epidemiological situation in 2014

In 2014, a total of 3,930 cases of syphilis (non-congenital) were reported to the NNDSS. This represented a rate of 16.8 per 100,000, an 11% increase compared with 2013 (15.2 per 100,000) (Figure 42). In 2014, 49% of syphilis notifications were categorised as greater than 2 years or unknown duration, and 51% of cases were categorised as infectious syphilis.

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

- In 2014, 2,009 cases of infectious syphilis were notified to the NNDSS.
- Of all notifications, 81% occurred in males aged 20–54 years.
- Cases of infectious syphilis were almost completely in MSM.

Figure 42: Notification rate for non-congenital syphilis infection (all categories),*† Australia, 2009 to 2014, by category and year



* Notifications were excluded where the case was aged less than 13 years and the infection was able to be determined as non-sexually acquired (8 notifications).

† For syphilis of more than 2 years or unknown duration, excludes South Australia from 2009–2011.

Epidemiological situation in 2014

In 2014, 2,009 notified cases of infectious syphilis <2 years duration were reported to the NNDSS, representing a rate of 8.6 per 100,000. This was a

13% increase compared with the rate reported in 2013 (7.6 per 100,000) and a 43% increase from 2009 (6.0 per 100,000) to 2014 (Table 6).

Geographical description

In 2014, notification rates for infectious syphilis were highest in the Northern Territory (29.8 per 100,000), Victoria (11.1 per 100,000) and New South Wales (9.8 per 100,000) (Table 16). This likely reflects the large proportions of at-risk individuals living in these jurisdictions; Indigenous persons in the Northern Territory and MSM in Victoria and New South Wales.[31,58] Increased screening in at-risk individuals may partly explain increased infection rates; however, the majority of the increase is likely to have been due to increased transmission.[59]

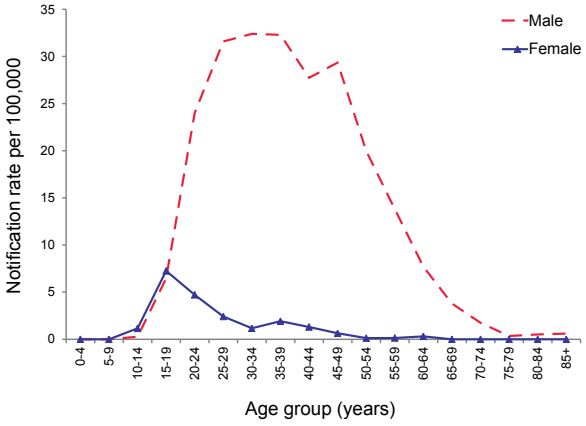
Age and sex distribution

Nationally in 2014, the notification rate for infectious syphilis was 15.8 per 100,000 in males and 1.4 per 100,000 in females, a male to female rate ratio of 11.3:1, which was consistent with previous years. In males, this was an increase of 13% when compared with the 2013 rate (14.0 per 100,000). The notification rate for females in 2014 did not markedly change from the rate seen in 2013 (1.3 per 100,000). In 2014, 81% (1,617/2008) of all notifications occurred in males aged 20–54 years (Figure 43). Similar to that seen in 2013, it is expected that diagnoses of infectious syphilis in 2014 were almost completely confined to MSM.[33]

Notification rates for males aged 15 years or over varied widely across age groups and increased overall from 2009 to 2014 for all age groups. For the majority of age groups, rates were at their lowest in 2010 after which rates steadily increased and reached maximum values for the period in 2014 (Figure 44).

In females aged 15 years or over, rates did not vary as noticeably across age groups as males (Figure 44). In females, notification rates over the 2009 to 2014 period averaged 2.1 per 100,000

Figure 43: Notification rate for infectious syphilis (primary, secondary and early latent), less than 2 years duration, Australia, 2014, by age group and sex*



* Excludes notifications where age and/or sex were not reported and notifications where the case was aged less than 13 years and the infection was able to be determined as non-sexually acquired (1 notification).

Table 16: Notifications and notification rate for infectious syphilis (less than 2 years duration),* Australia, 2014, by state or territory and sex

State or territory	Total*		Male		Female*	
	Notified cases	Notification rate‡	Notified cases	Notification rate‡	Notified cases	Notification rate‡
ACT	18	4.7	18	9.4	0	0.0
NSW	739	9.8	714	19.1	25	0.7
NT	72	29.8	40	30.9	32	28.6
Qld	394	8.3	317	13.5	77	3.2
SA	29	1.7	28	3.4	1	0.1
Tas.	14	2.7	12	4.7	2	0.8
Vic.	649	11.1	633	21.9	16	0.5
WA	93	3.6	82	6.3	11	0.9
Total	2,008	8.6	1,844	15.8	165	1.4

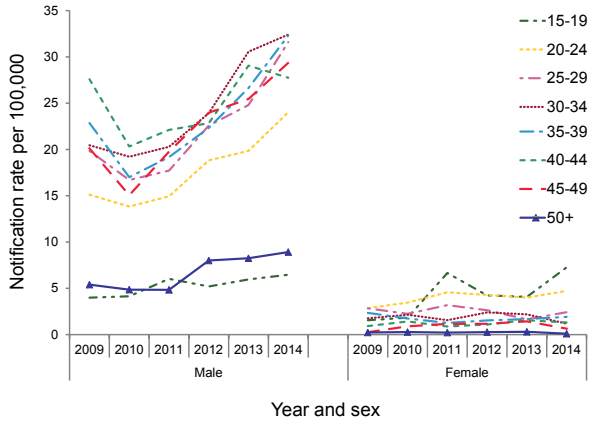
* Notifications were excluded where the case was aged less than 13 years and the infection was able to be determined as non-sexually acquired (1 notification).

† Includes notified cases where sex was not reported.

‡ Per 100,000 population.

(range: 0.1 to 7.3 per 100,000). Over the 6-year period, the notification rates remained low for females across all age groups.

Figure 44: Notification rate for infectious syphilis (primary, secondary and early latent), less than 2 years duration, Australia, 2009 to 2014, by year, sex and selected age groups*



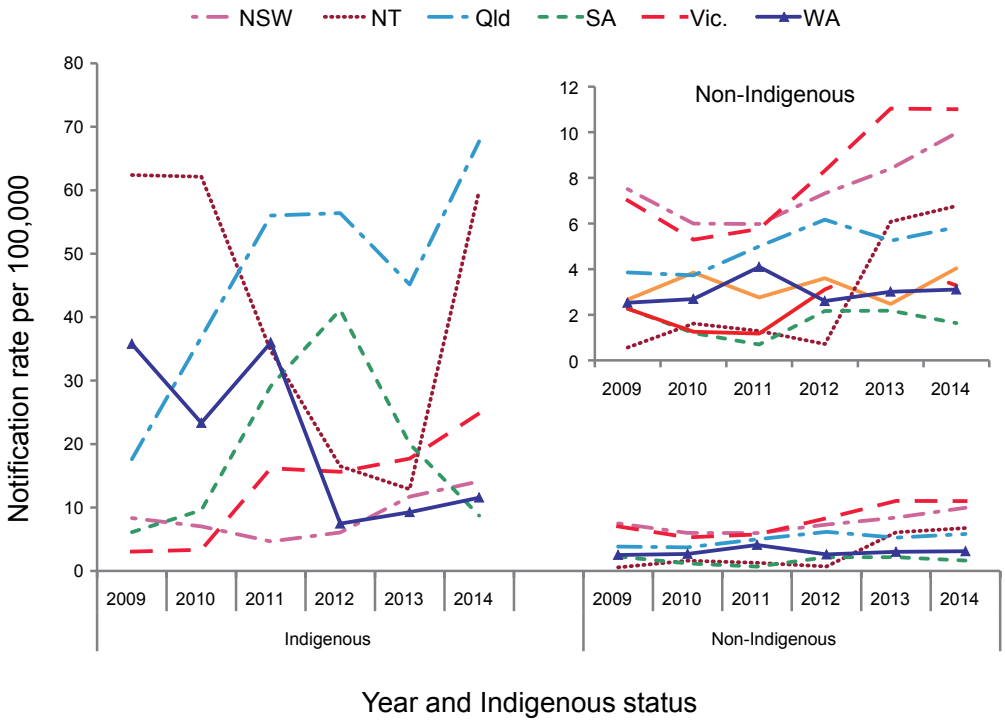
* Excludes notifications where age and/or sex were not reported and those less than 15 years of age (54 notifications).

Indigenous status

The completeness of Indigenous status identification in the notification data varies by year and by jurisdiction. Nationally in 2014, data on Indigenous status were complete for 92% of notifications of infectious syphilis, not changing from 2013, but lower than the preceding 5-year mean of 94% (range: 91% to 96%). All states and territories had greater than 50% completeness of the Indigenous status field across the 2009 to 2014 period.

In 2014, where rates were calculated for Indigenous and non-Indigenous persons, the age-standardised rates were higher for Indigenous persons than non-Indigenous persons in all jurisdictions (Figure 45). For all states and territories, the combined age standardised notification rate ratio between the Indigenous and non-Indigenous populations in 2014 was 4.4:1, which was the same as the preceding 5–year mean (range: 3.0 to 5.9). In 2014, for jurisdictions where cases were notified in both Indigenous and non-Indigenous persons, the age standardised notification rate ratio between Indigenous and non-Indigenous populations ranged from 1.4:1 in New South Wales to 11.6:1 in Queensland. Between 2013 and 2014, the largest increase in the difference between Indigenous and non-Indigenous age standardised notification rates

Figure 45: Age-standardised notification rate for infectious syphilis (primary, secondary and early latent), less than 2 years duration, selected states and territories,* 2009 to 2014, by Indigenous status and year. Inset: Non-Indigenous notification rates



* All states and territories reported Indigenous status for more than 50% of notifications between 2009 and 2014. The Australian Capital Territory and Tasmania were excluded due to low numbers of notifications.

was 318% in the Northern Territory (2.1 to 8.9 per 100,000). The only jurisdiction where the difference between Indigenous and non-Indigenous age standardised notification rates decreased from 2013 to 2014 was South Australia (a decrease of 42%).

An outbreak of infectious syphilis spanning northern areas of Queensland, the Northern Territory, and Western Australia and affecting largely young heterosexual Indigenous persons[5,12] was first detected in north-western Queensland in 2012 and in Central Australia in mid-2013,[60,61] continuing to 2014. Increased transmission along with targeted and opportunistic syphilis screening in each of these jurisdictions is likely to have contributed to an increase in Indigenous age-standardised rates for Queensland, the Northern Territory, and Western Australia between 2012–2014.[61]

Syphilis of more than 2 years or unknown duration

- In 2014, 1,921 cases of syphilis of more than 2 years or unknown duration were notified to the NNDSS.
- Notification rates decreased from 7.3 per 100,000 in 2009 to 6.1 per 100,000 in 2012 then increased to 8.2 per 100,000 in 2014.
- The notification rate among males (12.2 per 100,000) was nearly 3 times that in females (4.2 per 100,000) in 2014.

Epidemiological situation in 2014

In 2014, 1,921 cases of syphilis of more than 2 years or unknown duration were reported to the NNDSS. Notification rates increased by 12% between 2009 (7.3 per 100,000) and 2014 (8.2 per 100,000), and increased by 8% between 2013 (7.6 per 100,000) and 2014 (Table 6). This may have been due to increased testing in persons or populations with little previous testing history or it may have been due to an actual increase in the number of persons with non-infectious syphilis.

Geographical distribution

In 2014, notification rates for syphilis of more than 2 years or unknown duration were highest in the Northern Territory (29.8 per 100,000), followed by Victoria (13.7 per 100,000) (Table 17). Similar to infectious syphilis, this geographical distribution likely reflects the large proportions of at-risk individuals living in these jurisdictions (Indigenous persons in the Northern Territory and MSM in Victoria).[31,58]

Age and sex distribution

Nationally in 2014, the notification rate for syphilis of more than 2 years or unknown duration was 12.2 per 100,000 in males and 4.2 per 100,000 in females, a male to female rate ratio of 2.9:1. Between 2013 and 2014, the notification rate in males increased by 13% (10.8 to 12.2 per 100,000) and by 8% (3.9 to 4.2 per 100,000) in females. In 2014, approximately 73% (1404/1919) of all notifications for which sex was reported, occurred in males aged 20 years or over (Figure 46).

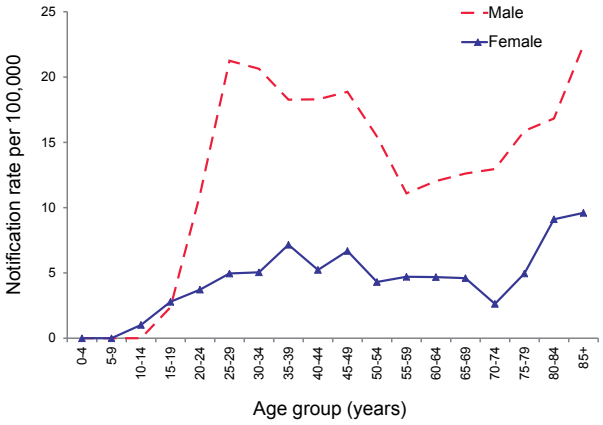
Table 17: Notifications and notification rate for syphilis (more than 2 years or unknown duration), Australia, 2014, by state or territory and sex

State or territory	Total*		Male		Female	
	Notified cases	Notification rate†	Notified cases	Notification rate†	Notified cases	Notification rate†
ACT	26	6.7	20	10.4	6	3.1
NSW	536	7.1	411	11.0	124	3.3
NT	73	29.8	40	30.9	33	28.6
Qld	279	5.9	182	7.7	97	4.1
SA	123	7.3	74	8.9	49	5.8
Tas.	19	3.7	14	5.5	5	1.9
Vic.	801	13.7	636	22.0	164	5.6
WA	64	2.5	45	3.5	19	1.5
Total	1,921	8.2	1,422	12.2	497	4.2

* Includes notified cases where sex was not reported.

† Per 100,000 population.

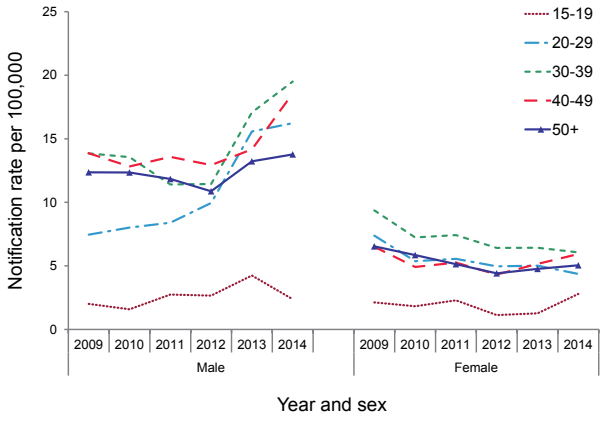
Figure 46: Notification rate for syphilis of more than 2 years or unknown duration,* Australia, 2014, by age group and sex



* Excludes notifications where age and/or sex were not reported and notifications where the case was aged less than 13 years (2 notifications).

Notification rates in males for all age groups except the 15–19 years age group increased overall from 2009 to 2014 (Figure 47). This increase is particularly prominent from 2012 to 2014. Notification rates in females for all age groups except the 15–19 years age group declined overall from 2009 to 2014 (Figure 47). Notification rates in males in the 15–19 years age group were lower than those of the other age groups and fluctuated across the time period. Notification rates in females in the 15–19 years age group were also lower than those of the other age groups with an increasing trend from 2012 to 2014 (Figure 47).

Figure 47: Notification rate for syphilis of more than 2 years or unknown duration, Australia,* 2009 to 2014, by year, sex and selected age groups†



* Data from all states and territories except South Australia in 2009–2011.

† Excludes notifications where age and/or sex were not reported and those aged less than 15 years (61 notifications).

Congenital syphilis

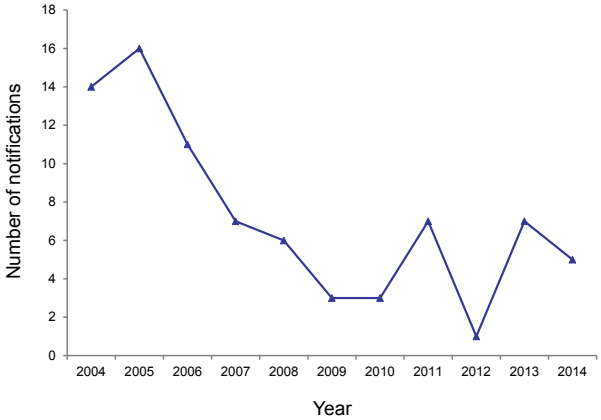
- In 2014, 5 cases of congenital syphilis were notified to the NNDSS.
- Congenital syphilis remains rare in Australia.

Congenital syphilis is caused by fetal infection with the bacterium *T. pallidum*. Syphilis is acquired by infants either in-utero or at birth from women with untreated early infection. Infections commonly result in abortion or stillbirth and may cause the death of a new-born infant. Congenital syphilis can be asymptomatic, especially in the first weeks of life.[22]

Epidemiological situation in 2014

There were 5 notifications of congenital syphilis in 2014, all occurring in Indigenous persons. This compared with 7 notifications of congenital syphilis in 2013. The preceding 5-year mean was 4.2 notifications (Table 6). Considering the previously mentioned syphilis outbreak in remote Indigenous communities, the increase in the number of cases seen in 2013 and 2014 (Figure 48) reflects the increased risk to neonates and mothers that outbreak situations pose.[62,63] Despite these peaks, case numbers remain low after a downward trend observed over the past decade (Figure 48). Routine antenatal screening for syphilis with follow-up and adequate treatment is considered to be a contributor to this overall decline.[64] Congenital syphilis, particularly in Indigenous persons, is targeted for elimination. This target is stated in the 4th National Aboriginal and Torres Strait Islander Blood-borne Viruses and Sexually Transmissible Infections Strategy and the third National Sexually Transmissible Infections Strategy, both for 2014–2017.[65,66]

Figure 48: Notifications of congenital syphilis, Australia, 2004 to 2014



Vaccine preventable diseases

This section summarises the national surveillance data for notifiable diseases targeted by the National Immunisation Program (NIP) in 2014. These include diphtheria, invasive *Haemophilus influenzae* type b (Hib) infection, laboratory confirmed influenza, measles, mumps, pertussis, invasive pneumococcal disease (IPD), poliomyelitis, rubella, tetanus and varicella zoster virus (VZV) infections (unspecified, chickenpox and shingles). Data on hepatitis B and invasive meningococcal disease, which are also targeted by the NIP, can be found in this report under 'Bloodborne diseases' and 'Other bacterial infections' respectively. Other vaccine preventable diseases (VPDs) presented in this report include hepatitis A and Q fever which can be found under the 'Gastrointestinal' and 'Zoonoses' sections respectively. For more detailed reports on historical data, including notifications, hospitalisations and deaths, readers are referred to the journal supplements 'Vaccine Preventable Diseases in Australia' and the 'Australian Vaccine Preventable Diseases Epidemiological Review Series' for additional analysis on individual diseases, which are published in CDI.

In 2014, there were 101,400 VPD notifications reported to the NNDSS, representing 37% of all reported cases and a 70% increase compared with 2013 (n=59,630). Influenza was the most commonly notified VPD with 67,742 cases (67%) reported, followed by pertussis (11,863, 12%). The number of notifications and notification rates for VPDs in Australia are shown in Table 4, Table 5 and Table 6.

Vaccination coverage is an important factor influencing the incidence of VPDs. Since the commencement of the Australian Childhood Immunisation Register in 1996, vaccination coverage in children has been high by international standards, although geographical pockets of lower coverage, in which there is an increased potential for VPD cases still remain. As no vaccine is 100% effective, infections with these diseases sometimes do occur in fully vaccinated people. However, evidence shows vaccines do provide a substantially lower chance of developing infection or can reduce the severity of disease. [67–71]

Information on a case's vaccination history was previously recorded in the NNDSS using the 'vaccination status' field (fully or partially vaccinated for age or not vaccinated), plus fields capturing the number of doses, the last vaccination date and how the vaccination information was validated. In January 2008 new, more detailed fields were incorporated for recording 'vaccine type', and 'vaccination date' for each dose of vaccine given. The new fields were intended to replace the old fields, with a transition period allowing either field to be utilised. In 2014, all jurisdictions, except the Australian Capital Territory, were using the new fields. In this report the vaccination status of a case is interpreted according to the data provided by the states and territories from the 2 different formats. A case is described as fully vaccinated if they have received all doses of the relevant vaccine according to the most recent edition of *The Australian Immunisation Handbook,*[32] and at least 14 days prior to disease onset.

Diphtheria

- In 2014, there were 2 imported cases of diphtheria notified to the NNDSS.
- Diphtheria is rare in Australia.

Diphtheria is an acute pharyngeal or cutaneous infection caused mainly by toxigenic strains of *Corynebacterium diphtheriae.* The exotoxin acts locally on the mucous membranes of the respiratory tract, and on damaged skin, although this is not as common. Disease is mainly due to local membranous inflammation, which for pharyngeal diphtheria can cause airway obstruction. Occasionally, systemic infections occur and cause damage to the myocardium, nervous system and kidneys. Diphtheria is spread by respiratory droplets or direct contact with nasopharyngeal secretions or skin lesions. While there are non-toxigenic strains of *C. diphtheriae,* they usually only cause mild throat or skin infection and are not nationally notifiable.[22]

Epidemiological situation in 2014

In 2014, there were 2 notifications† of diphtheria reported. Both cases were cutaneous, reported in Queensland and were imported from Tokelau and Cambodia. One case was reported as vaccinated and the other was of unknown vaccination status.

† This number may underrepresent the number of diphtheria cases in Australia due to a change in the national case definition for this disease. In mid-2013, the national case definition for diphtheria was revised, requiring clinical and laboratory evidence for confirmed cases. This change may have inadvertently excluded some notifications of cutaneous toxigenic diphtheria, as cutaneous presentations were not listed as clinical evidence in the revised definition.

Diphtheria is rare in Australia, with most cases associated with sporadic importations from countries in which the disease remains endemic. From 2001 to 2013, there were 7 cases of diphtheria reported to the NNDSS, including 1 case in 2001, a cluster of 3 cases and a sporadic case in 2011 and 2 cases in 2013. Of these, 5 were imported and 2 were linked to an imported case.

Influenza

- The seasonal increase in laboratory confirmed influenza notifications for 2014 was slightly earlier and reached a higher peak than recent years, excluding the 2009 influenza pandemic.
- Nationally, influenza A was the predominant influenza virus type. However, the distribution of influenza types and subtypes was variable between jurisdictions and changed as the season progressed. Unlike the rest of the country where influenza A(H1N1)pdm09 predominated throughout the season, New South Wales and the Australian Capital Territory saw influenza A(H3N2) circulating at higher levels.

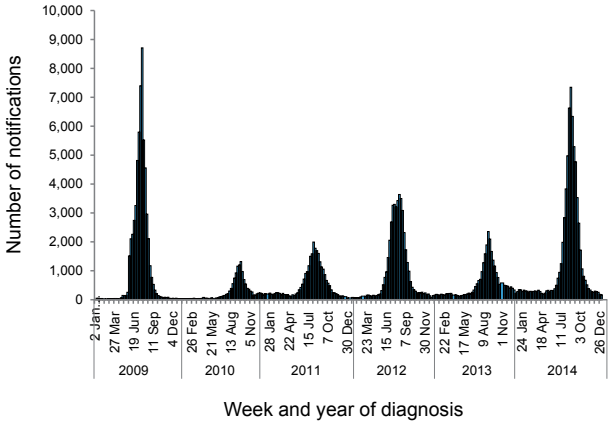
Influenza is a common, highly infectious acute respiratory disease caused by infection with influenza viruses. The virus is transmitted from person to person by airborne droplets of exhaled respiratory secretions, especially by coughing or sneezing.[72] The disease ranges from asymptomatic[73] through to mild upper respiratory tract illness, to severe complications including pneumonia. The severity of disease is determined by features intrinsic to the virus including its similarity to previous circulating and vaccine strains and by host factors including the age, level of immunity and presence of chronic medical conditions.[74,75]

Annual influenza vaccination is the primary means of preventing or attenuating influenza and its complications and is included in the NIP for individuals who are at increased risk of complications from influenza infection. In 2014, the NIP funded influenza vaccination for people aged 6 months and over with medical conditions placing them at risk of serious complications due to influenza, Aboriginal and Torres Strait Islander people aged 15 years or over, pregnant women and people aged 65 years or over.[32]

Epidemiological situation in 2014

In 2014, there were 67,742 notifications of laboratory confirmed influenza, which was almost 2.4 times the number of notified cases reported in 2013 (n=28,311) (Figure 49). The number of notifications recorded in 2014 is the highest on record and was 21% higher than 2009 (n=56,026), the year of the last influenza pandemic.

Figure 49: Notifications of laboratory confirmed influenza, Australia, 1 January 2009 to 31 December 2014, by week and year of diagnosis



Geographical distribution

Notification rates were highest in South Australia (655 per 100,000) and Queensland (380 per 100,000). Notifications rates in the Australian Capital Territory, New South Wales, and the Northern Territory were somewhat similar to the national notification rate of 289 per 100,000, while rates reported in Tasmania, Victoria and Western Australia were substantially lower than the national rate at 131, 170 and 205 per 100,000 respectively. New South Wales reported the highest number of influenza cases of any jurisdiction (n=20,877), comprising 31% of notifications nationally (Figure 50).

Age and sex distribution

The highest number of influenza notifications occurred in the 0–4 years and 5–9 years age groups (n=8,415 and 5,510, respectively), which together accounted for 21% of all notifications (Figure 51). Notification rates were highest in the 0–4 years and over 85 years age groups (551 and 547 notifications per 100,000 respectively) with an additional peak in the 35–39 years age group (312 notifications per 100,000) (Figure 51).

In seasons dominated by the influenza A(H1N1) pdm09 virus, such as 2009, 2010 and 2011, the age distribution of influenza notification rates showed a downward trend with increasing age (Figure 52). For comparison, in 2012, which was dominated by influenza A(H3N2), the age distribution of influ-

Figure 50: Notifications of laboratory confirmed influenza, Australia, 2014, by week and state or territory

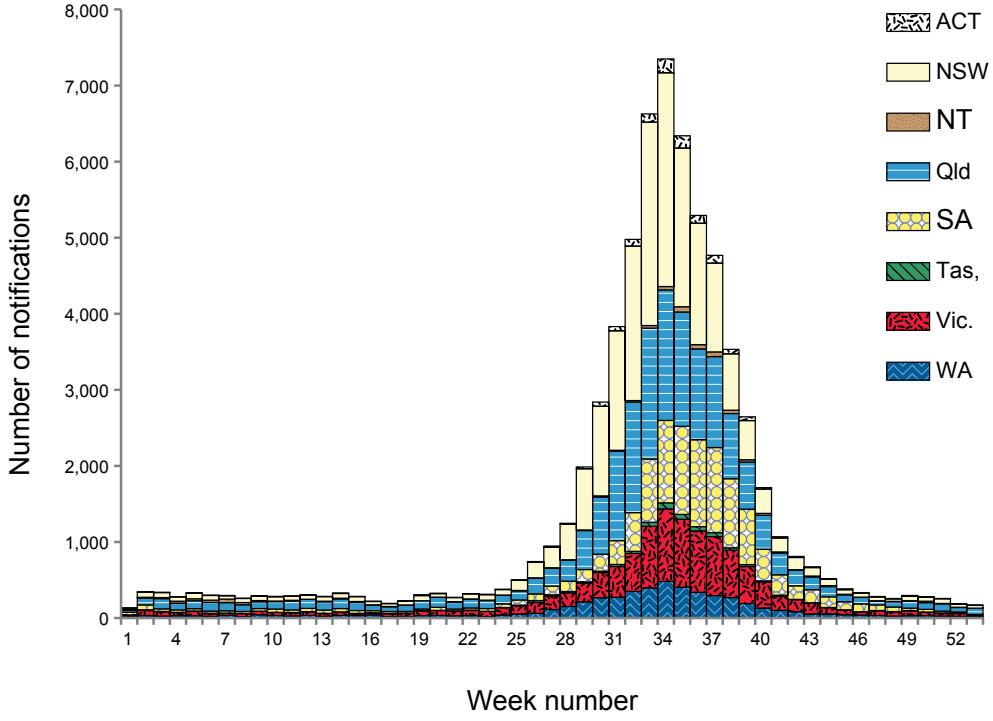


Figure 51: Notifications and notification rate for laboratory confirmed influenza, Australia, 2014, by age group and sex

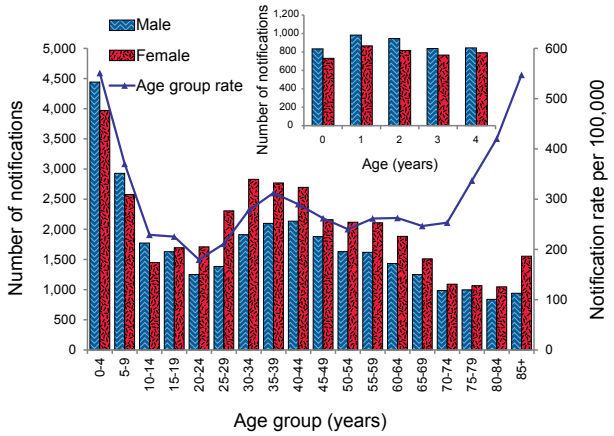
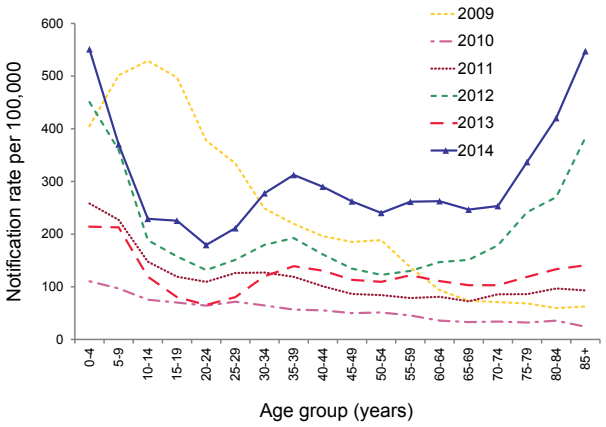


Figure 52: Notification rate for laboratory confirmed influenza, Australia, 2009 to 2014, by age group and year



enza notifications was bimodal with peaks in those aged under 10 years and in those aged 70 years or over. The 2014 influenza season was characterised by co-circulation of A(H1N1)pdm09 and influenza A(H3N2) with the proportion of influenza B viruses rising towards the end of the year. This broad strain distribution has seen the burden of disease carried across a breadth of age groups.

In 2014, females accounted for 54% (n=35,538) of the influenza notifications for which sex was reported. The age group-specific rate of influenza in males exceeded that in females in age groups less than 15 years and greater than 75 years.

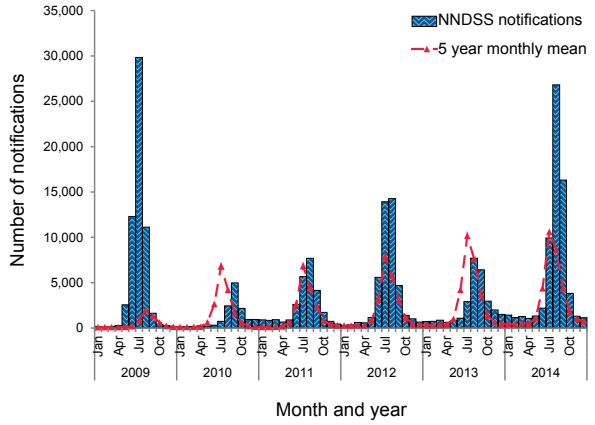
Seasonality

Influenza notifications during the 2013–14 inter-seasonal period were the highest on record with an average of 1,358 notifications per month. It is unclear whether this was a reflection of a higher prevalence of influenza circulating in the com-

munity at this time, an increased rate of testing or another factor. Queensland reported the largest number of inter-seasonal influenza notifications.

The seasonal increase of influenza notifications in 2014 started in June, rose sharply and peaked in August. This was slightly earlier than the seasonal patterns in the past 3 influenza seasons (Figure 53). The peak was higher than previous years, excluding the 2009 influenza pandemic. The majority of jurisdictions peaked in activity around late August, followed by a steady decline in influenza activity back to inter-seasonal levels by November.

Figure 53: Notifications of laboratory confirmed influenza, Australia, 2009 to 2014, by month and year



Indigenous status

Nationally in 2014, Indigenous status was reported in 40% (n=27,052) of laboratory confirmed notifications of influenza. Indigenous status completeness was greater than 50% in 3 jurisdictions: the Northern Territory (100%), South Australia (87%) and Western Australia (93%). Among these, the combined notification rate for influenza in Indigenous peoples was 584 per 100,000 and 371 per 100,000 among non-Indigenous population, representing a notification rate ratio of 1.6.

Mortality

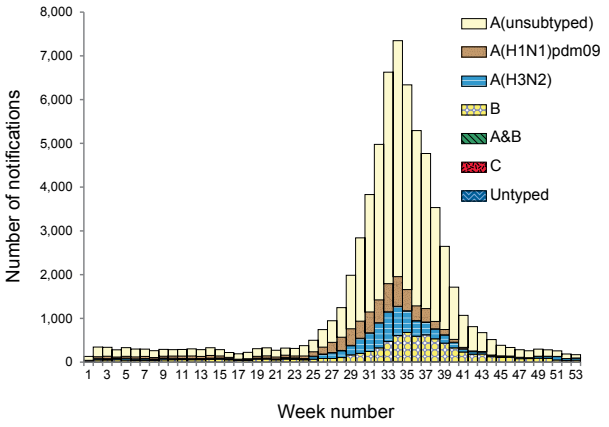
Nationally, there were 132 influenza-associated deaths notified to the NNDSS, with a median age of 75 years (range 1–103 years). The majority of deaths were associated with influenza A infections (n=126; 95%), and of these, 93 were associated with influenza A(unsubtyped) infections, 24 were A(H1N1)pdm09 and 9 were A(H3N2). Indigenous status was reported for 81% (n=107) of the influenza-associated deaths; and Indigenous peoples accounted for 6% (n=6) of these deaths. The number of influenza-associated deaths reported to the NNDSS is reliant on the follow-up of cases to determine the outcome of their infection and most likely underestimates the true mortality associated with this disease.

Microbiological trends

National Notifiable Diseases Surveillance System

In 2014, typing data were reported for all but 18 laboratory confirmed influenza notifications. Of notifications with typing information, 88% were due to influenza type A (n=59,563) and 12% were due to influenza type B (n=8,052). Whilst the majority of notifications of influenza A were reported as unsubtyped (69%, n=46,771), influenza A(H1N1)pdm09 and influenza A(H3N2) circulated in similar proportions (10%, n=6,922 and 9%, n=5,870 respectively). Mixed influenza type A and B infections accounted for less than 1% of notifications (n=96). There were 13 notifications of influenza type C (Figure 54).

Figure 54: Notifications of laboratory confirmed influenza, Australia, 2014, by week and subtype

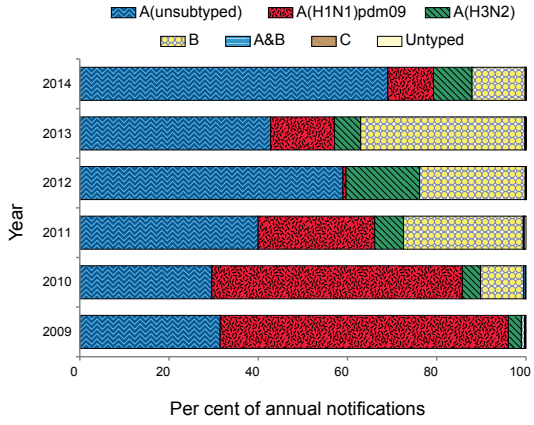


As in previous years, influenza A remained the predominant virus type in 2014 (Figure 55). Influenza A(H3N2) accounted for a larger burden of disease due to influenza A than has been seen in previous years, with the exception of 2012 where almost all disease due to influenza A was caused by influenza A(H3N2). Influenza B circulated at lower levels in 2014 when compared with the previous 3 years.

While influenza A(H1N1)pdm09 and influenza A(H3N2) circulated in similar proportions nationally, distribution varied between jurisdictions. Influenza A(H1N1)pdm09 predominated across most jurisdictions throughout the season. However,

influenza A(H3N2) was predominant in New South Wales and the Australian Capital Territory, with late season increases noted in Queensland, Western Australia, the Northern Territory and Tasmania.

Figure 55: Per cent of annual notifications of laboratory confirmed influenza, Australia, 2009 to 2014, by subtype

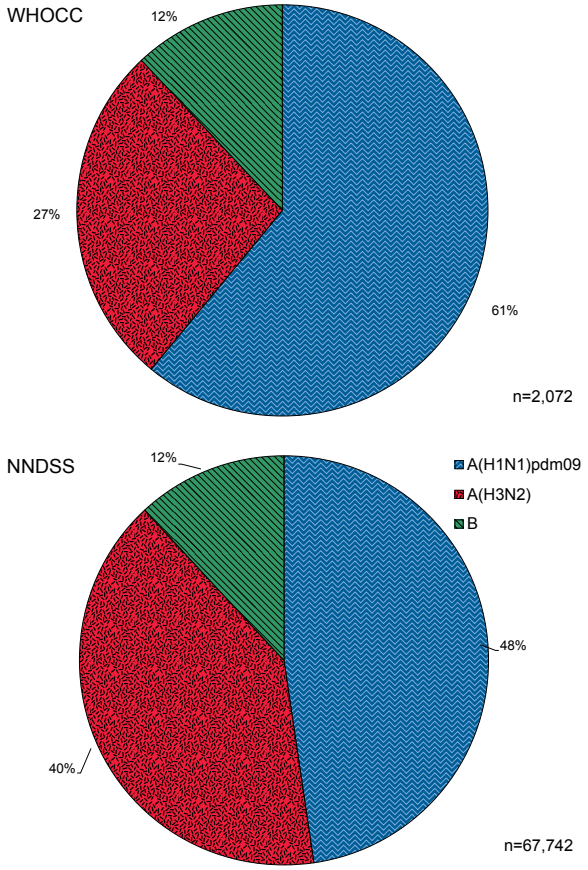


WHO Collaborating Centre for Reference and Research on Influenza

For 2014, the WHO Collaborating Centre for Reference and Research on Influenza (WHOCC) analysed 2,072 specimens from influenza cases identified in Australia. Specimens are submitted to the WHOCC from laboratories according to guidelines that aim for successful isolation of viruses and likelihood of obtaining a vaccine candidate. WHOCC specimens therefore do not constitute a representative sample of influenza infections, which most likely accounts for differences in virus subtype distribution between NNDSS and WHOCC.

The number of samples analysed by the WHOCC represented approximately 3% of the 67,742 laboratory confirmed cases reported to the NNDSS compared with 1,532 isolates in 2013 representing 5.4% of notifications. The majority of specimens were influenza A(H1N1)pdm09 (61%, n=1,263) with 27% influenza A(H3N2) (n=562) and 12% influenza B (n=247) (Figure 56). While the proportion of WHOCC isolates typed as influenza B was similar to that reported in laboratory confirmed notifications, the distribution of influenza A(H1N1)pdm09 and influenza A(H3N2) differed, assuming that the notifications to NNDSS of influenza A reported as unsubtyped were similarly distributed as the subtyped notifications.

Figure 56: Subtyped influenza virus samples WHO Collaborating Centre for Reference and Research on Influenza versus National Notifiable Diseases Surveillance System, Australia, 2014



The WHOCC assessed the antigenic similarity of circulating influenza virus isolates to reference strains included in the trivalent vaccine from recent years using the haemagglutination inhibition assay. The 2014 seasonal influenza vaccine contained 2 changes from 2013 and included an A/Texas/50/2012 (H3N2)-like virus and a B/Massachusetts/2/2012-like virus.

The majority of the A(H1N1)pdm09 isolates (1,257 of 1,263) were antigenically similar to the A(H1N1) component of the influenza vaccine, which has been used each year since 2010. The remaining 6 (0.5%) isolates were characterised as 'low reactors'. This suggests that the A(H1N1) viruses, which have been circulating since the 2009 pandemic continue to be genetically and antigenically stable. By comparison, approximately 6% (33/562) of A(H3N2) isolates were antigenically drifted from the A/Texas/50/2012 vaccine strain and, characterised as 'low reactors', the remainder of the A(H3N2) viruses were genetically distinct from A/Texas/50/2012.

Of the influenza B viruses tested (n=247), 83% (206) were from the B/Yamagata lineage, with the remainder from the B/Victoria lineage. A high proportion of B/Yamagata viruses (70%, 145/206) were low reactors to the B component of the 2014 vaccine (B/Massachusetts/2/2012-like) thus the vaccine was a poor match to the circulating lineages. Further studies determined that the majority (86%, 178/206) of B/Yamagata viruses circulating in 2014 were more antigenically similar to the 2013 trivalent influenza vaccine B component (B/Wisconsin/1/2010–like). B/Massachusetts/2/2012 and B/Wisconsin/1/2010 were both B/Yamagata viruses but sit within genetically-distinct clades in the influenza phylogenetic tree.

Viruses submitted to the WHOCC in 2014 were also tested for sensitivity to the neuraminidase inhibitor class of antiviral drugs. Neuraminidase inhibition assays were performed on 2,025 virus isolates consisting of 1,242 A(H1N1)pdm09, 551 A(H3N2) and 232 influenza B viruses. Reduced inhibition by oseltamivir was detected in 3 A(H1N1)pdm09 isolates and reduced inhibition by zanamivir was detected in a single A(H3N2) isolate. In recent years, resistance to oseltamivir in Australian-sourced isolates, has been mediated primarily through the well characterised H275Y mutation,[76] however, this was not the case in 2014 where none of the resistant isolates carried this mutation.

Due to the circulation of drifted A(H3N2) viruses and the predominance of a different B/Yamagata-lineage in Australia and elsewhere in the Southern Hemisphere, there were 2 updates recommended for the 2015 trivalent influenza vaccine for Australia, with the incorporation of an A/Switzerland9715293/2013-like A(H3N2) virus and a B/Phuket/3073/2013-like B/Yamagata virus (the latter virus being isolated at the WHOCC) being added to the existing A/California/7/2009-like A(H1N1)pdm09 virus.

Enhanced surveillance datasets

In addition to NNDSS data, a series of targeted influenza surveillance systems operated during 2014. Together these systems collected data, which were used to describe the season with respect to epidemiology, morbidity, mortality and virology and supported the conclusions drawn from analyses of NNDSS notification data. Enhanced influenza surveillance was based on the following additional sources of data:

- the number and proportion of calls to a national health call centre network for influenza or influenza-like illness (ILI);
- rates of ILI from a community survey;
- consultation rates for ILI identified by sentinel general practitioners;
- consultation rates for ILI identified by hospital emergency departments in Western Australia, New South Wales and the Northern Territory;
- hospitalised cases of influenza from 17 sentinel hospitals (adult and paediatric) across Australia;
- mortality data from the New South Wales Registry of Births, Deaths and Marriages; and
- typing and subtyping for influenza from sentinel laboratories in New South Wales, Victoria, Western Australia and Tasmania.

These data sources were used to inform the overall picture of influenza activity in Australia and comprehensive analysis of these data are provided in the fortnightly Australian Influenza Surveillance Report, which was published during the season, and in the annual National Influenza Surveillance Scheme report.

Discussion

In Australia, the 2014 influenza season was slightly earlier than in previous seasons with active transmission of influenza virus commencing in mid-June, sharply increasing in mid-July and peaking in mid-August. Influenza A predominated, accounting for 88% of cases, while influenza B made up 12% of notifications. While the majority of the influenza A cases were unsubtyped (69%), of those subtyped, A(H1N1)pdm09 (33%, 6,922/20,953) was predominant nationally throughout the season, with increasing proportions of A(H3N2) virus towards the end of the season.

Rates of influenza were highest among those in the less than 5 years, 30–44 years and ≥80 years age groups. The age distribution, especially in the younger and middle aged populations, is consistent with the observations of previous years associated with influenza A(H1N1)pdm09 virus, whereas infections in older age groups are typical of influenza A(H3N2).

Taking into account additional data from other targeted influenza surveillance systems monitored throughout the season, the severity of the 2014 influenza season was moderate across most jurisdictions. However, more severe activity was noted in New South Wales, where influenza A(H3N2) circulated at higher levels and affected people in older age groups, which led to a substantial number of outbreaks in residential care facilities.

Invasive *Haemophilus influenzae* type b

- In 2014, 21 cases of invasive Hib reported to the NNDSS.
- Of the cases reported 57% were male and 52% were under the age of 14 years.
- The 2014 notification rate of Hib remains low at 0.1 per 100,000 population.

Hib is a gram negative bacterium that causes disease with symptoms dependant on which part of the body is infected. Clinical categories of invasive disease caused by Hib include septicaemia (infection of the blood stream); meningitis (infection of the membranes around the brain and spinal cord); epiglottitis (severe swelling of the epiglottis at the back of the throat); and a range of other infections. Hib is mostly carried as a commensal (present without causing symptoms) in the nasopharynx of healthy individuals and is spread by respiratory secretions, including aerosol transmission or contact with articles soiled with discharges from the nose or throat.[77] The case fatality rate of Hib meningitis is at least 3% in developed countries, even with treatment. Approximately 15% to 30% of survivors have permanent neurological sequelae.[78]

Epidemiological situation in 2014

In 2014, there were 21 notifications of invasive Hib infection in Australia. This was similar to the number of cases notified in 2013 (n=20) and represented a ratio of 1.1 compared with the mean of the previous 5 years. The 2014 notification rate was 0.1 per 100,000 population, consistent with the very low rates seen since the introduction of the vaccine on the NIP in July 1993 (Figure 57). Cases occurred in 6 states or territories, with 9 cases reported in Queensland, 6 cases in New South Wales, 3 cases in Victoria and 1 case each reported in the Northern Territory, South Australia and Western Australia. Notification rates were consistent between states and territories, ranging from 0.1 per 100,000 in New South Wales, South Australia and Victoria to 0.4 per 100,000 in the Northern Territory. There was 1 Hib associated death in 2014 reported in a 2-month-old Indigenous female who was unvaccinated.

Age and sex distribution

Just over half of notified invasive Hib cases in 2014 were male (57%, n=12). Approximately half of the cases (52%, n=11) were in children aged less than 14 years, and 46% (n=5) of these were among infants less than 1 year of age (Figure 58). Consistent with previous years, the 0–4 years age group had the highest notification rate (0.5 per 100,000).

Figure 57: Notifications and notification rate for invasive *Haemophilus influenzae* type b infection, Australia, 1994 to 2014, by year

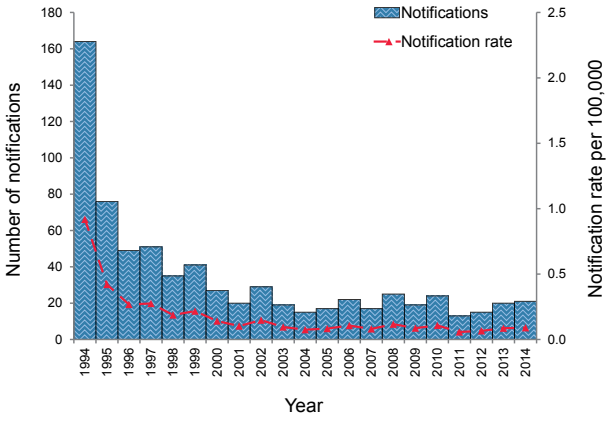
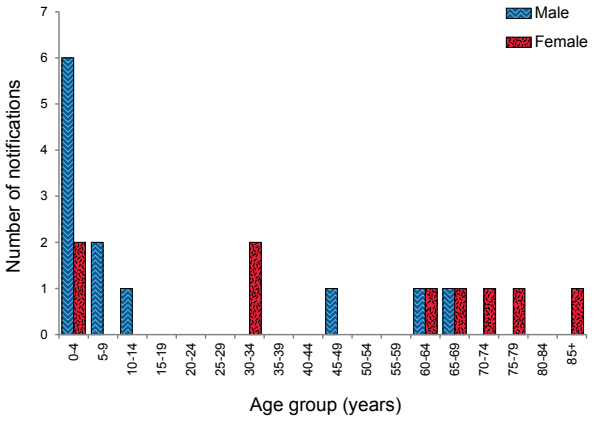


Figure 58: Notifications of invasive *Haemophilus influenzae* type b infection, Australia, 2014, by age group and sex



Indigenous status

Indigenous status was reported for all Hib cases in 2014. Five cases were reported as Indigenous Australians, representing a notification rate of 0.8 per 100,000. This was higher than the average of the previous 3 years (0.5 per 100,000) but lower than 2010 (1.4 per 100,000).

Vaccination status

In 2014, persons less than 22 years of age were eligible for Hib vaccination under the NIP during their infancy. Of the 21 Hib cases reported in 2014, 11 were eligible for vaccination. Six cases were 12 months of age or older, and therefore eligible for the full primary vaccine course and the booster. Of these, 4 were partially vaccinated, 1 was not vaccinated and 1 was of unknown vaccination status. Five cases were less than 12 months of age, of which 3 were reported as partially vaccinated (2 doses) and 2 were not vaccinated.

Invasive pneumococcal disease

- In 2014, 1,564 cases of invasive pneumococcal disease were notified to the NNDSS.
- Compared with 2013, the notification rate of invasive pneumococcal disease remains unchanged.

IPD is a disease in which *Streptococcus pneumoniae* is isolated from a normally sterile site such as blood, cerebrospinal fluid or pleural fluid. Transmission of the bacterium from person to person is usually via the inhalation of infected respiratory droplets. Many of the signs and symptoms of IPD are non-specific including fever, chills, headache, stiff neck and a general feeling of being 'out-of-sorts', severe symptoms can include seizures and occasionally coma.[22]

Epidemiological situation in 2014

There were 1,564 cases reported in 2014, representing a notification rate of 6.7 per 100,000. This notification rate was unchanged from the rate reported in 2013 and maintains the 20% rate reduction observed following the introduction of the 13-valent pneumococcal conjugate vaccine (13vPCV) to the NIP for infants in July 2011.

Geographic distribution

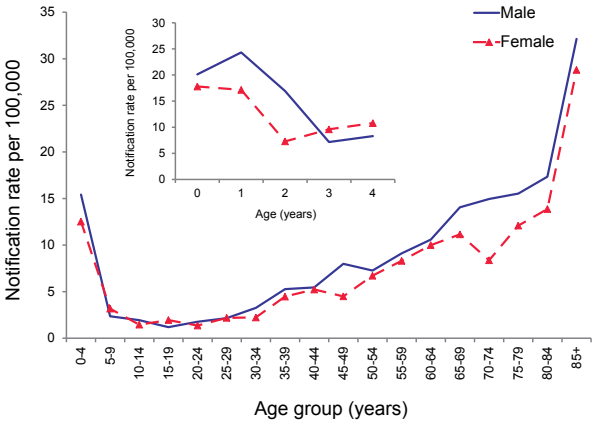
In 2014, the Australian Capital Territory, New South Wales, Tasmania, South Australia and Western Australia all reported an increase in the number of cases, with South Australia reporting the greatest increase on the previous year (20%, 111 to 133). The Northern Territory, Queensland and Victoria all reported a reduction in the number of cases, with the Northern Territory reporting the greatest reduction when compared with the previous year (26%, 58 to 43). Changes in notification rates in the jurisdictions reflected the changes in the number of cases, with rates ranging from 3.9 per 100,000 in the Australian Capital Territory to 17.6 per 100,000 in the Northern Territory.

Age and sex distribution

In 2014, males accounted for 53% (n=827) of cases of IPD. The rate of disease in males exceeded that in females in all age groups except for the 5–9 years, 15–19 years and 25–29 years age groups (Figure 59).

In 2014, the notification rate for IPD was highest in older Australians and in young children, with an age distribution similar to that seen in 2013.[37] In older Australians, the highest notification rate was

Figure 59: Notification rate for invasive pneumococcal disease, Australia, 2014, by age group and sex



in those aged 85 years or older (30.0 per 100,000), while the highest rate in children aged less than 5 years was in those aged 1 year (20.8 per 100,000) (Figure 59).

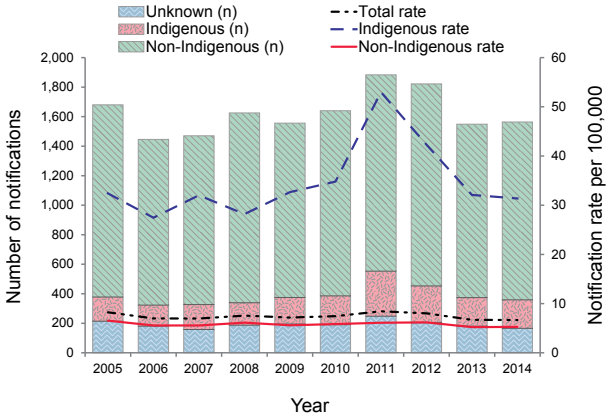
Seasonality

Many respiratory diseases, including IPD, are known to show a distinct seasonal trend that generally peaks during the winter months. In 2014, the seasonal trend of IPD peaked in July (n=213), 1 month earlier than the preceding 3 years.

Indigenous status

In 2014, 89% (n=1,398) of IPD cases were reported with a known Indigenous status. Of those with a known Indigenous status, 14% (n=193) were reported as Indigenous. The notification rate in the Indigenous population (31.4 per 100,000) was approximately 6 times the rate in non-Indigenous people (5.3 per 100,000) (Figure 60).

Figure 60: Notifications and notification rates of invasive pneumococcal disease, Australia, 2005 to 2014, by year and Indigenous status



Vaccination status

In Australia, pneumococcal vaccination is recommended as part of routine immunisation for the medically at risk, children under 5 years of age, Aboriginal and Torres Strait Islander peoples aged 50 years or over and other Australians aged 65 years or over. More information on the scheduling of the pneumococcal vaccination can be found in *The Australian Immunisation Handbook,* 10th edition.[32] The history of pneumococcal vaccination recommendations and practice is available through the National Centre for Immunisation Research and Surveillance of Vaccine Preventable Diseases.[79]

Microbiological trends

Although there are over 90 *S. pneumoniae* serotypes, a relatively limited number cause the majority of IPD. Monitoring the profile of *S. pneumoniae* serotypes causing invasive pneumococcal disease in the community is critical for evaluating the impact of the NIP funded vaccines as well as for the early detection of emerging serotypes and serotype specific outbreaks. IPD serotypes were reported in 95% (n=1,487) of notified cases in 2014.

In 2014, 68% (1,005) of all notifications with a known serotype were a result of a serotype included in the 23vPPV, and 39% (586) were included in the 13vPCV. Across all ages, the most frequently reported serotypes were 3 (n=150), 19A (n=139), 7F (n=132), 22F (n=120), 19F (n=80) and 6C (n=66) with these 6 serotypes accounting for 46% (687) of all notifications with serotype information. Serotypes 3, 19A, 7F and 19F are included in both the 13vPCV and the 23vPPV. Serotype 22F is only included in the 23vPPV and 6C is not covered by a vaccine. The remaining 56% (n=800) of cases were distributed across 58 other different serotypes.

In Indigenous children aged under 5 years, there were 34 notifications, with serotype 23B (n=6) being the most frequently reported serotype. Serotype 23B is not included in the 13vPCV. In non-Indigenous children aged under 5 years, there were 180 notifications, with serotype 19A (n=30) being the most frequently reported serotype. Serotype 19A is included in the 13vPCV.

In 2014, 37% (n=79) of notifications in children aged under 5 years were a result of a serotype included in the 13vPCV. This was similar to the 38% (n=72) of notifications reported in 2013 and maintains the large reduction in notifications in this age group observed following the introduction of the 13vPCV to the NIP.

In Indigenous adults aged 50 years or over, there were 54 notifications, with serotype 3 (n=6) being the most frequently reported serotype. In non-Indigenous adults aged 65 years or over, there were 502 notifications, with serotype 3 (n=51) and serotype 22F (n=51) being the most frequently reported. Both serotypes 3 and 22F are included in the 23vPPV.

In 2014, 57% (n=31) of notifications in Indigenous peoples aged 50 years or over, and 55% (n=278) of notifications in non-Indigenous Australians aged 65 years or over, were a result of a serotype included in 23vPPV. This continues a general downward trend observed in both these adult population groups in recent years. The 13 serotypes included in the 13vPCV are also included in the 23vPPV and the downward trend in notifications caused by serotypes included in the 23vPPV is likely to be a result of the herd immunity effect afforded to them by the vaccination of infants with 13vPCV.

Enhanced surveillance data sets

Enhanced data are available for IPD notifications. Further analyses, including risk factors and antibiotic susceptibilities can be found in annual and quarterly IPD surveillance report series published regularly in CDI. In addition, a subset of IPD notification data, including serotype, age, sex, Indigenous status, clinical categories and vaccination history are publicly available in the NNDSS IPD Public Dataset (http://www.health.gov.au/internet/main/publishing.nsf/Content/cda-surveil-nndss-ipd-reports.htm).

Measles

- Measles is no longer endemic in Australia.
- In 2014, there were 340 notified cases of measles, representing a national notification rate of 1.4 per 100,000 population.
- Seventy-five per cent of cases were either imported or import-related.
- The largest outbreak of measles in 2014 consisted of 29 cases and lasted approximately 7 weeks.

Measles is a highly infectious acute viral illness, caused by the measles virus, which is spread by respiratory secretions, including aerosol transmission.[80] Initial symptoms last 2 to 4 days and are characterised by fever and malaise, followed by a cough, coryza and conjunctivitis. It is usually followed by a red blotchy rash, which typically begins on the face, and then becomes generalised. Measles is often a severe disease with complications more common in the chronically ill, including otitis media, pneumonia, diarrhoea and acute encepha-

litis.[81] Subacute sclerosing panencephalitis is a late, rare (approximately 1 in 100,000 cases) complication of measles caused by persistent infection and is always fatal.[32] Complications are more common in children under 5 years of age and in adults over 20 years of age.[82]

Epidemiological situation in 2014

In 2014, there were 340 notifications of measles. This represents a notification rate of 1.4 per 100,000 population, which was 2.3 times the mean of the previous 5 years (n=146) and an increase of 110% compared with 2013 (n=162) (Figure 61). In 2014, cases of measles occurred in all states and territories, with the 23% of cases occurring in Victoria (n=77) (Figure 62).

In temperate climates and where measles transmission remains endemic, the majority of cases usually occur in late winter to early spring.[83] In Australia, this seasonal pattern is no longer evident (Figure 62).

Age and sex distribution

The majority of notified measles cases were male (59%, n=199) in 2014. There was a wide variation in the male to female rate ratio across the age groups with the most notable difference in the 20–24 years and 30–34 years age groups, where there were 2 times as many males as females (Figure 63).

In 2014, age at diagnosis ranged from 0–64 years, with a median age of 19 years. Compared with 2013, notification rates increased in all age groups in 2014. Consistent with recent years, infants less than 1 year of age had the highest age specific rate (10.7 per 100,000). Rates have remained below 2.5 per 100,000 in all age groups between 2009 and 2014, with the exception of the less than 1 year age group in 2011, 2012 and 2014 and the 10–19 years age group in 2014 (Figure 64).

Forty-nine cases occurred in those born between 1978 and 1982 (32–36 years old in 2014), a cohort previously identified as susceptible to measles

Figure 61: Notifications and notification rate for measles, Australia, 1998 to 2014, by year

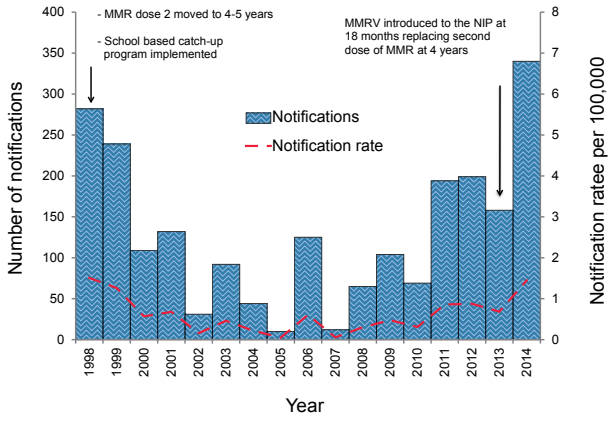
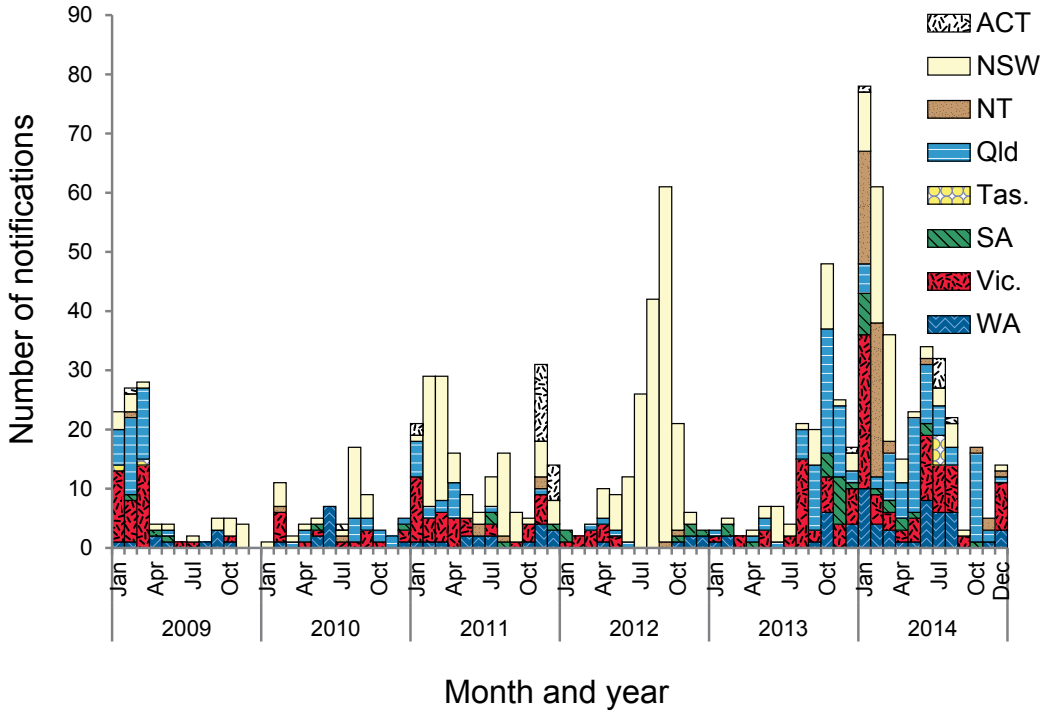


Figure 62: Notifications of measles, Australia, 2009 to 2014, by month, year and state or territory



infection.[84] Four cases were born before 1966, a cohort that is considered to have high levels of natural immunity.[85]

Figure 63: Notification rate for measles, Australia, 2014, by age group and sex

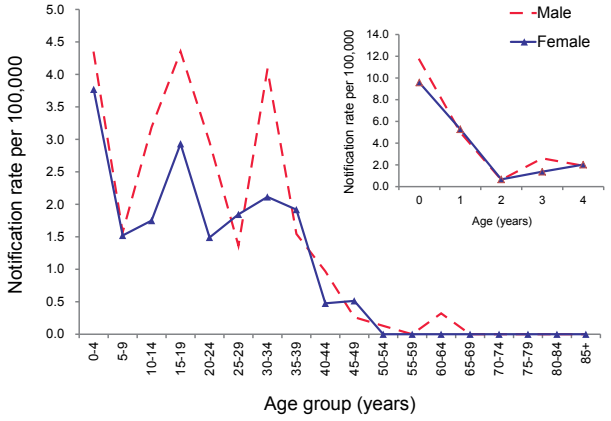
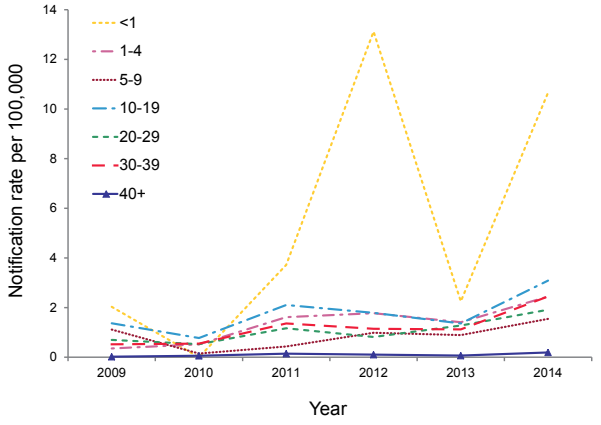


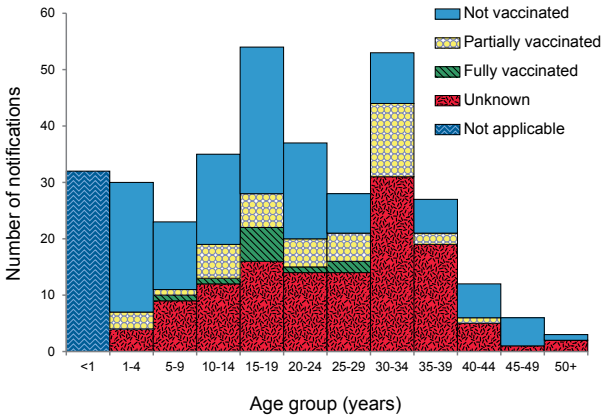
Figure 64: Notification rate for measles, Australia, 2009 to 2014, by year and selected age groups



Vaccination status

Two doses of the measles containing vaccine are recommended for all persons born during or after 1966. Of the 340 cases notified in 2014, 90% (n=305) were born after 1965 or were 12 months of age or older and therefore eligible for at least 1 dose of a measles-containing vaccine. Eighty-three per cent (n=252) of cases eligible for vaccination were either not vaccinated (42%, 127/305) or of unknown vaccination status (41%, 125/305). Of the remaining 17% (n=53) who were vaccinated, 11 had received the full course of 2 doses of a measles-containing vaccine and 42 were partially vaccinated with 1 dose (Figure 65). Young children and adolescents between 5 and 19 years of age accounted for 61% (77/127) of all unvaccinated cases. In 2014, 28% (25/88) of cases less than 15 years of age were reported as of unknown vaccination, in contrast to 46% (100/217) of cases 15 years or over (Figure 65).

Figure 65: Notifications of measles, Australia, 2014, by age group and vaccination status



The measles-mumps-rubella (MMR) vaccine induces long term measles immunity in 95% of recipients after a single dose and 99% of recipients after the second dose.[32]

Indigenous status

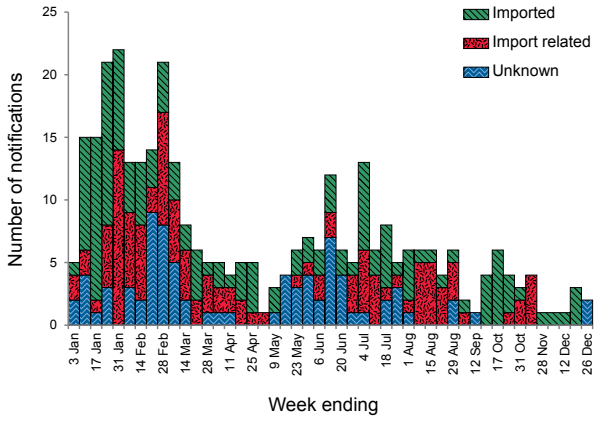
Indigenous status was completed for 96% of cases in 2014 (n=327), an increase in completeness compared with the 93% of cases in 2013. Of the cases reported in 2014, 3% (n=11) were reported as Indigenous.

Source of infection and outbreaks

Seventy-five per cent of cases in 2014 were either imported (n=140) or import-related (n=115) with the remaining 25% (n=85) of unknown source (Figure 66). Of the imported cases, 64% (90/140) were from the WHO Western Pacific Region, with the majority of cases imported from the Philippines (n=50) followed by Papua New Guinea (n=20) and Vietnam (n=13). Of the remaining imported cases, 43 were imported from the WHO South East Asia Region, 2 from the WHO European Region and 1 from the African Region.

There were 42 clusters of 2 or more epidemiologically linked cases (outbreaks) in 2014 accounting for 60% (n=204) of all cases. The remaining cases comprised sporadic imported cases (n=98) and sporadic cases acquired in Australia of unknown source (n=38). Seventy-eight per cent of clusters were import related (33/42). There were 9 clusters of locally acquired cases of unknown source, which occurred in 6 separate states or territories

Figure 66: Notifications of measles, Australia, 2014, by week and source of infection



including Western Australia, 1 cluster of 5 cases; New South Wales, 3 clusters – 1 of 2 cases, 1 of 3 cases and another of 7 cases; Queensland, 1 cluster of 8 cases; the Australian Capital Territory, 1 cluster of 4 cases; The Northern Territory, 1 cluster of 3 cases; Victoria, 1 cluster of 11 cases; and 1 cluster of 6 cases involving both Victoria and the Northern Territory.

Transmission was interrupted quickly in all outbreaks in 2014. The median outbreak duration was 14 days (range 0 to 72 days) between the onset of symptoms in the index and the last case. The median generations of transmission [86] was 1 (range 0 to 7). Thirty-nine of 42 clusters had fewer than 10 cases with a median of 3 cases (range 2 to 9). Of the 3 outbreaks with 10 or more cases, 2 were import related and all were genotyped as B3. The largest of these outbreaks comprised 29 cases and occurred principally in the Northern Territory (n=28), with 1 linked case in a resident of Western Australia. This outbreak commenced in mid-January, lasted approximately 7 weeks, included 5 generations of transmission and was associated with an imported case from Singapore.

Microbiological trends

Genotyping data were available for 41 clusters with 2 or more linked cases in 2014. Genotype B3 was associated with 22 separate clusters (n=126 cases), D8 with 11 clusters (n=47 cases), D9 with 4 clusters (n=14 cases) and H1 with 4 clusters (n=14 cases). Of the 136 sporadic cases 84% (n=114) were genotyped.

Imported genotypes varied by WHO region. In 2014, there was 1 B3 importation from the African Region and 2 separate D8 importations from the European Region. Multiple genotypes were imported from the South East Asia Region (B3, D8, D9, H1 and G3) and the Western Pacific Region (B3, D8, D9 and H1).

Discussion

The increasing prevalence of measles in some parts of the world, and the continued circulation of the virus in countries of close geographical proximity to Australia, will result in a continual source of imported virus in Australia. This was particularly the case in 2014 with 157 separate importations occurring. Despite this large number of importations in 2014, the majority were sporadic (n=98) and did not lead to local transmission.

Evidence suggests that endemic measles has been eliminated from Australia, since at least 2005,[83] and this was verified by the WHO in 2014.[87] In 2014, none of the outbreaks persisted for more than 12 months and there was no evidence of a single genotype continuously circulating. Ongoing evidence of high population immunity was demonstrated by the short duration and small number of cases in the majority of outbreaks.

Due to the highly infectious nature of measles, local transmission and outbreaks will continue to occur in Australia, mostly among susceptible contacts of non-immune travellers from countries where measles remains prevalent.

Mumps

- There were 190 cases of mumps reported in 2014.
- Since 2009, the notification rate of mumps has remained below 1.0 per 100,000 population.

Mumps is an acute viral illness caused by the mumps virus. Transmission is usually by respiratory secretions, including aerosol transmission, or by direct contact with saliva. Asymptomatic infections occur in one-third of cases. Symptomatic disease ranges from mild upper respiratory tract infections to systemic involvement. The characteristic bilateral, or occasionally unilateral, parotid swelling occurs in 60% to 70% of clinical cases, however, a high proportion have non-specific symptoms including fever, headache, malaise, myalgia and anorexia.[88] Mumps encephalitis has been estimated to occur in 1 to 2 per 10,000 cases, with a case fatality rate of around 1%.[22]

Epidemiological situation in 2014

In 2014, there were 190 notifications of mumps, which was a 13% decrease compared with the

218 cases reported in 2013 and a ratio of 1.1 compared with the 5-year mean (n=167). Since 2009, the national notification rate of mumps has remained below 1.0 per 100,000, ranging from 0.4 per 100,000 in 2010 to 0.9 per 100,000 in 2012 and 2013, and 0.8 per 100,000 in 2014. Cases of mumps were reported from all states and territories in 2014, with the highest rates occurring in South Australia and New South Wales (1.1 per 100,000 each) (Figure 67).

Place of acquisition was complete for 54% (n=102) of cases in 2014, of which 25% (25/102) were imported from overseas: 13 from Asia, 4 from Africa, 4 from the Americas, 2 from Europe and 1 each from the Middle East and New Zealand. The remaining 77 cases were reported as locally acquired in Australia.

Age and sex distribution

In 2014, just over half of all of notified mumps cases were reported in males (52%, n=98) and persons under the age of 40 years (67%, n=128) (Figure 68). The highest number of notifications for males occurred in the 0–4 years age group with 13 cases, while for females notifications were highest in the 35–39 years age group with 11 cases. Consistent with recent years, adults in the 30–39 years age group had the highest rates of infection (1.4 per 100,000) (Figure 69). Since 2010, there has been a steady increase in age-specific rates across all age groups. This is particularly evident in the 1–4 years age group rates, which have increased from 0.3 per 100,000 in 2010 to 1.1 per 100,000 in 2014.

Figure 68: Notifications of mumps, Australia, 2014, by age group and sex

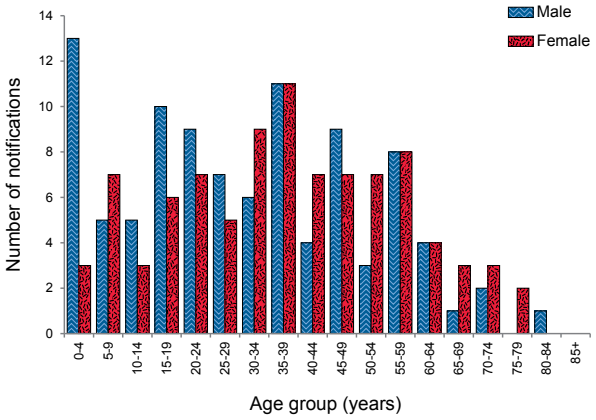


Figure 69: Notification rate for mumps, Australia, 2009 to 2014, by year and selected age groups

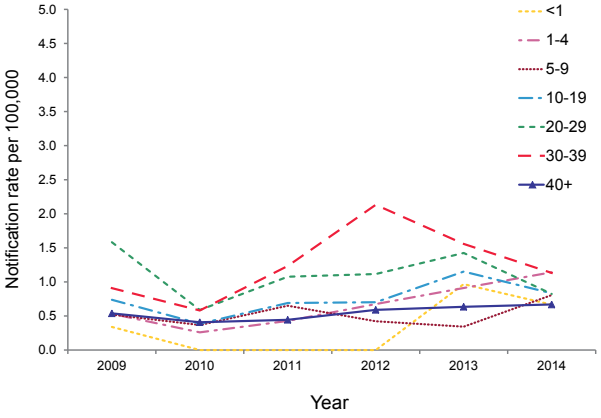
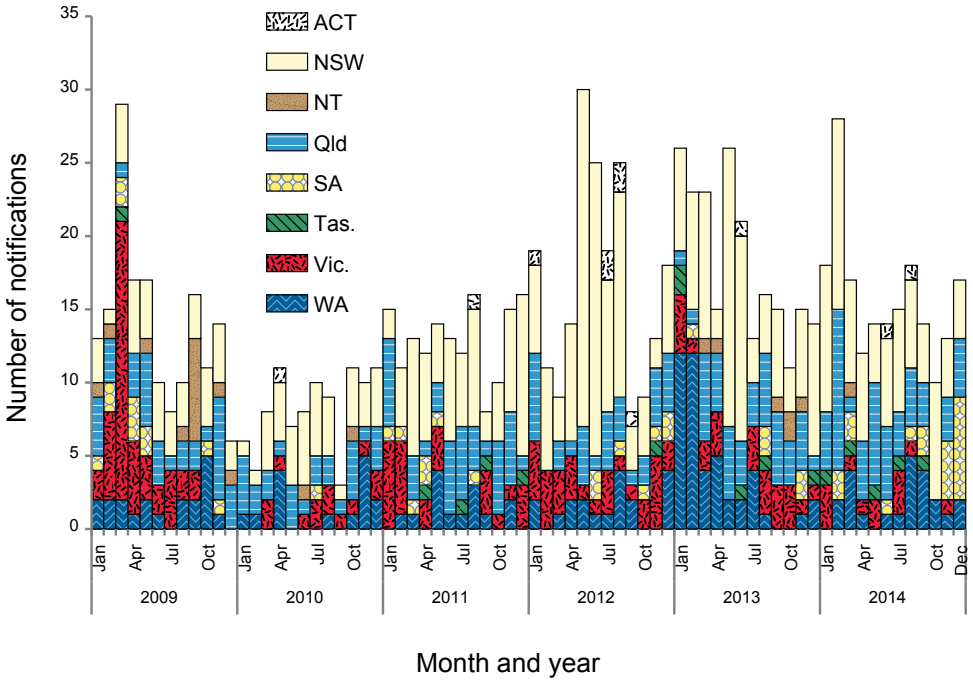


Figure 67: Notifications of mumps, Australia, 2009 to 2014, by month, year and state or territory



Indigenous status

Indigenous status was reported for 77% (n=147) of notified mumps cases in 2014. This is higher than the mean completeness of the previous 5 year period (64%, range 51% to 80%). Of the cases with a known Indigenous status, 3 (2%) were reported as Indigenous. The proportion of mumps notifications reported as Indigenous has remained below 5% since 2010.

Vaccination status

The mumps vaccine was first funded on the NIP schedule in 1982 for infants at 12 months of age, with people born after 1980 eligible for at least 1 dose of a mumps containing vaccine. Of the 190 cases notified in 2014, 49% (n=94) were eligible for at least 1 dose of a publicly funded mumps-containing vaccine. Of these, 17% (16/94) were unvaccinated and 38% (36/94) were of unknown vaccination status, 23% (22/94) were fully vaccinated, having received 2 doses of a mumps containing vaccine and 15% (14/94) were partially vaccinated with 1 dose of a mumps containing vaccine. Six cases were reported as vaccinated but had no dose information provided.

The mumps component of the MMR vaccine is considered to be the least effective of the 3 components with the reported 1 dose vaccine effectiveness varying between 60% and 90%.[89–91] While protection is greater in 2-dose vaccine recipients, recent outbreaks have been reported in 2-dose recipients, particularly young adults who received their vaccines more than 10 years previously.[92,93] Reduced effectiveness of the mumps vaccine over time may partially account for the proportion of vaccinated cases notified and contribute to mumps outbreaks in older vaccinated populations.[94]

Outbreaks

The outbreak reference field was completed for 6% (n=11) of cases in 2014. There were 3 outbreaks of 2 or more epidemiologically linked cases reported, of which, 2 were import related, 1 in Western Australia consisting of 3 cases, and 1 in South Australia consisting of 5 cases. The third outbreak comprised of 2 locally acquired cases in Western Australia.

Pertussis

- Pertussis remains highly prevalent in Australia.
- In 2014, there were 11,863 cases of pertussis reported to the NNDSS.
- National notifications continued to decline in 2014 and were the lowest level since 2007.
- In 2014, children under 15 years of age had a notification rate 2.4 times higher than those 15 years of age or over.

Pertussis, commonly known as whooping cough, is a highly infectious acute respiratory disease caused by the bacteria *Bordetella pertussis*. Spread by respiratory droplets, infection is often characterised by paroxysmal cough with inspiratory whoop, which is frequently seen among unvaccinated children but uncommon in individuals who have acquired some immunity through vaccination or infection.[95] The highest risk of infection and severe morbidity from pertussis occurs in infants who are too young to have received at least 2 doses of a pertussis-containing vaccine.[32] Complications include pneumonia, atelectasis, seizures, encephalopathy, and hernias, with pneumonia as the most common cause of death.[22]

Epidemiological situation in 2014

In 2014, pertussis notifications were at their lowest levels since 2007 and continued to display a downwards trend since reaching a peak in 2011, at the height of the most recent epidemic period, 2008 to 2012. There were 11,863 notifications of pertussis, which was a 4% decrease in notified cases compared with 2013 (n=12,362) and 51% less than in 2012 (n=24,101) (Figure 70). There were 2 pertussis related deaths reported. Both of these cases were reported as unvaccinated, with 1 case aged 7 months and the other 85 years.

In 2014, all jurisdictional specific rates had returned to pre-epidemic levels with rates remaining below 90 per 100,000 population. Compared with 2013, rates declined in all jurisdictions except New South Wales, Victoria and Western Australia (Figure 71). Rates in Victoria increased from 51 per 100,000 in 2013 to 81 per 100,000 in 2014, in New South Wales from 32 per 100,000 in 2013 to 42 per 100,000 in 2014 and in Western Australia from 65 per 100,000 in 2013 to 68 per 100,000 in 2014.

Age and sex distribution

Males accounted for 56% (n=6,657) of cases in 2014 and had higher rates across all age groups from 10 years of age (Figure 72). The highest noti-

Figure 70: Notifications of pertussis, Australia, 2009 to 2014, by month, year and state or territory

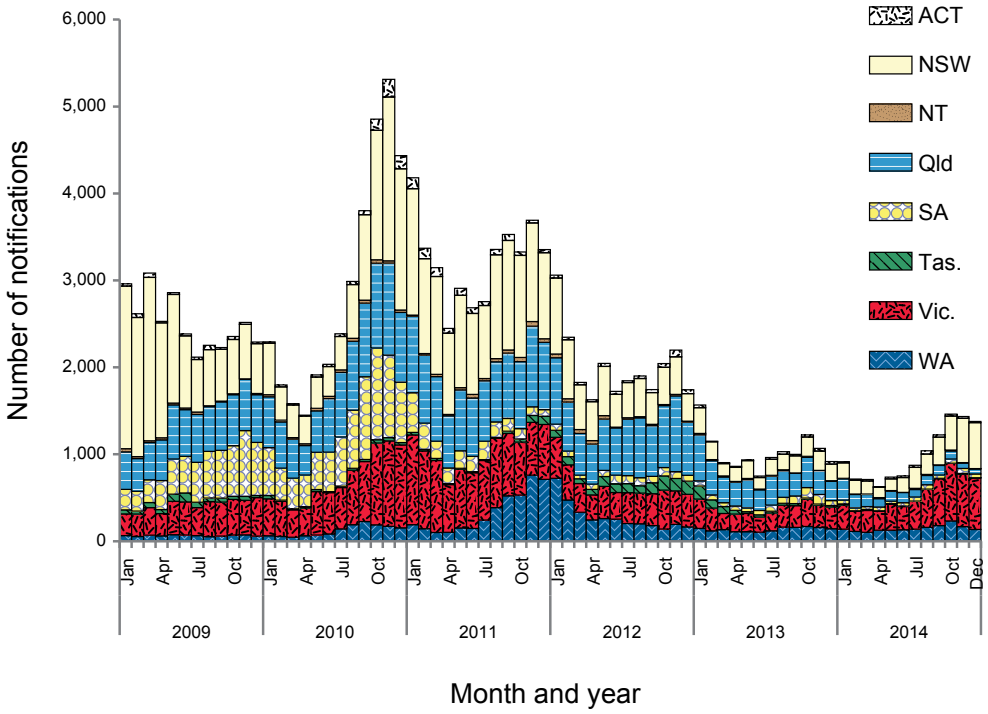


Figure 71: Notification rate for pertussis, Australia, 2009 to 2014, by year and state or territory

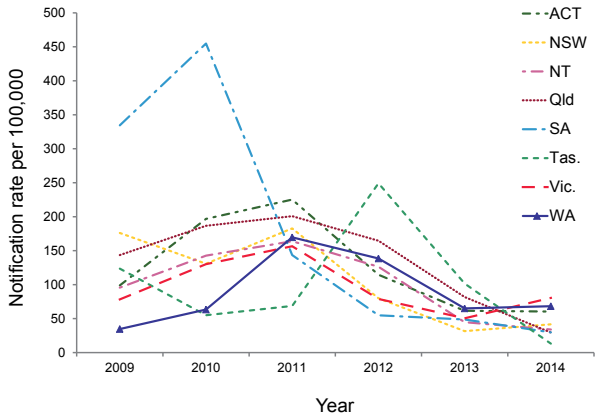
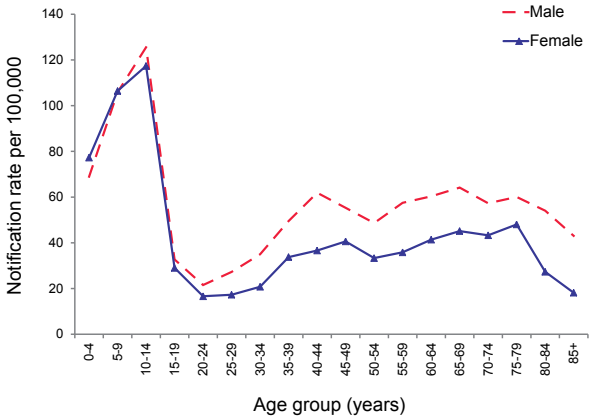


Figure 72: Notification rate for pertussis Australia, 2014, by age group* and sex†



* Excludes 16 cases reported without age.

† Excludes 5 cases reported without sex.

fication rate in both males and females occurred in the 10–14 years age group at 126 and 117 per 100,000 respectively.

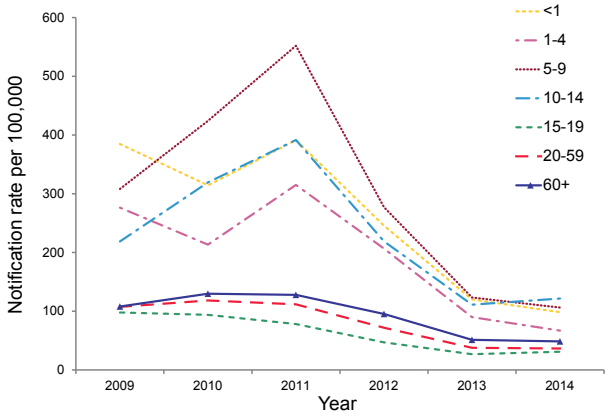
In 2014, children less than 15 years of age represented 37% (n=4,407) of notifications and had a notification rate (100 per 100,000) 2.6 times higher compared with those 15 years of age or over (36 per 100,000). After reaching a peak in 2011 rates in children less than 15 years of age have declined steeply, with the ratio of cases under 15 years compared with those over 15 years falling from 3.7 in 2011 to 2.7 in 2014. The highest age specific rates in 2014 occurred in the 10–14 years age group (122 per 100,000), which was higher than the rates reported in 2013 (111 per 100,000) (Figure 73).

Vaccination status

The NIP schedule in 2014 included a primary course of 3 doses of vaccine at 2, 4, and 6 months of age, with additional booster doses provided at 4 years of age and between 10 and 15 years of age.[32]

In order to determine the vaccination status of cases, public health follow-up is required. As per the pertussis national guidelines for public health units,[96] jurisdictions prioritise case follow-up to those less than 5 years of age. During 2014, those aged less than 5 years accounted for 9% (n=1,117) of all notified cases, of which information about vaccination status was available for 89% (n=992).

Figure 73: Notification rates for pertussis, Australia, 2009 to 2014, by year and selected age groups*



* Excludes 16 cases reported without age.

Of the children eligible to receive a pertussis-containing vaccine in 2014, 33% (n=255) of cases had received the full primary course of 3 doses and 22% (n=34) had received the full scheduled course of 4 doses (Table 18). Fifty-nine per cent (n=636) of eligible cases less than 5 years of age had received at least 2 doses of a pertussis-containing vaccine in 2014.

Pertussis vaccine effectiveness among Australian children has been estimated to range from 82% to 89% with the lower figure representing the cohort of children who would not have been eligible for the 18-month booster dose, which was removed from the NIP in 2003.[97] Immunity to disease decreases over time post-vaccination, with estimates of protection remaining for 4 to 12 years.[98–100] While pertussis can affect people of any age, infants are at highest risk of more severe disease as adequate immunity is not achieved through infant vaccination until receiving at least the second vaccine dose at 4 months of age.[101]

Discussion

Epidemics of pertussis have historically occurred at regular intervals of approximately 4 years on a background of endemic circulation in Australia, with the most recent epidemic peaking in 2011. In 2014, all jurisdictions reported pertussis activity consistent with pre-epidemic levels and national rates were at their lowest since 2007. However nationally, an increasing trend was evident from mid-2014, which was driven by a significant increase in pertussis activity in New South Wales and Victoria and a return to epidemic level activity in 2015 would not be unexpected.

All jurisdictions, except for the Northern Territory, ceased their respective cocooning programs in 2012, which included various combinations of providing free booster vaccinations to mothers and carers of infants.

Poliomyelitis

- There were no notifications of poliomyelitis in 2014.
- Australia, along with the Western Pacific Region, remains poliomyelitis free.

Table 18: Notifications of pertussis in children aged 0 to 5 years, Australia, 2014, by age group and number of vaccine doses

Age group	Number of vaccine doses					Unknown	Total
	0	1	2	3	4		
Less than 6 weeks of age (not eligible for vaccination)	25	0	0	0	0	14	39
6 weeks to <4 months (eligible for 1 dose of vaccine)	14	67	3	0	0	14	98
4 to < 6 months (eligible for 2 doses of vaccine)	3	23	27	0	0	3	56
6 months to < 4 years (eligible for 3 doses of vaccine)	61	125	250	255	5	73	769
4 to 5 years (eligible for 4 doses of vaccine)	18	20	51	11	34	21	155
Total	121	235	331	266	39	125	1,117

Poliomyelitis is an acute illness following gastrointestinal infection by 1 of the 3 types of poliovirus. Transmission occurs primarily from person-to-person via the faecal-oral route. In most cases poliovirus infection is not symptomatic but, in less than 1% of cases the virus may invade the nervous system and cause acute flaccid paralysis (AFP).[22]

Epidemiological situation in 2014

In 2014, there were no notifications of poliomyelitis. Australia, along with the Western Pacific Region, remains poliomyelitis free.

Poliovirus infection, both paralytic (poliomyelitis) and non-paralytic, is a notifiable disease in Australia. Clinical and laboratory investigation is conducted for cases involving patients of any age with a clinical suspicion of poliomyelitis, following the WHO protocol, which focuses on investigating cases of AFP in children under 15 years of age. The WHO target for AFP surveillance in a polio free country is 1 case of AFP per 100,000 children less than 15 years of age. Australia has achieved this surveillance target since 2008. However, the virological surveillance indicator of adequate stool specimen collection in 80% of AFP cases has never been met. More details can be found in the annual report series published in the CDI by the Australian Enterovirus Reference Laboratory who coordinate poliovirus surveillance activities in Australia.

Rubella and congenital rubella

- Rubella is a rare disease in Australia.
- Since 2003, rubella notifications have been less than 0.3 per 100,000.
- In 2014, there were 17 cases of rubella and no cases of congenital rubella syndrome reported.

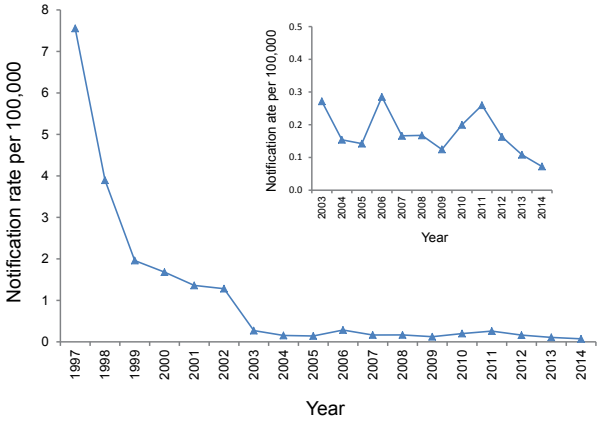
Rubella is generally a mild and self-limiting infectious disease caused by a rubella virus. It is spread from person-to-person through contact with respiratory secretions, including aerosol transmission. A rash, usually starting on the face before spreading across the body, may appear around 2 weeks after exposure to the virus and usually lasts for 3 days. Children usually show few or no constitutional symptoms of infection, but adults may experience 1 to 5 days of early low grade symptoms, such as fever, malaise, headaches and mild head colds.[22] Clinically, rubella can be difficult to distinguish from other diseases that also cause febrile rash, such as measles, and is asymptomatic in up to 50% of cases.

Rubella infection in the first trimester of pregnancy can result in miscarriages, foetal deaths or stillbirths, and a collection of birth defects known as congenital rubella syndrome (CRS) in over 90% of cases.[22,102] CRS can result in single or combined defects such as hearing impairment, eye abnormalities (including retinopathy, cataract and microphthalmia) congenital glaucoma, microcephaly, meningoencephalitis, development delay, purpura, jaundice radiolucent bone disease and congenital heart disease.

Epidemiological situation in 2014

In 2014, there were 17 cases of rubella reported, representing a rate of 0.1 per 100,000. While this was consistent with the low rates of this disease experienced since 2003, it was a marked decline from the peak rate of more than 7.5 per 100,000 in 1997 (Figure 74). Cases were reported from all states except Tasmania. No cases were reported from the Australian Capital Territory or the Northern Territory. There were no cases of CRS reported in 2014.

Figure 74: Notification rate for rubella, Australia, 1997 to 2014, by year



Age and sex distribution

In 2014, the majority of notified rubella cases were female (65%, n=11), of which 64% (7) were of child bearing age (15–44 years of age) (Figure 75). The median age of cases was 35 years, with a range of 7–56 years. Consistent with previous years, the majority of cases (82%, 14/17) occurred among adults over the age of 20 years (Figure 75), and age-specific rates remained below 0.7 per 100,000 across all age groups (Figure 76).

Vaccination status

Rubella vaccine is provided in the combined MMR or measles-mumps-rubella-varicella vaccine, and in 2014 was provided under the NIP schedule at

Figure 75: Notifications of rubella, Australia, 2014, by age group and sex

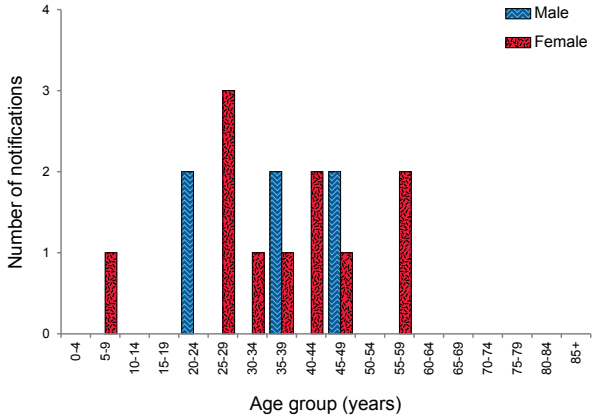
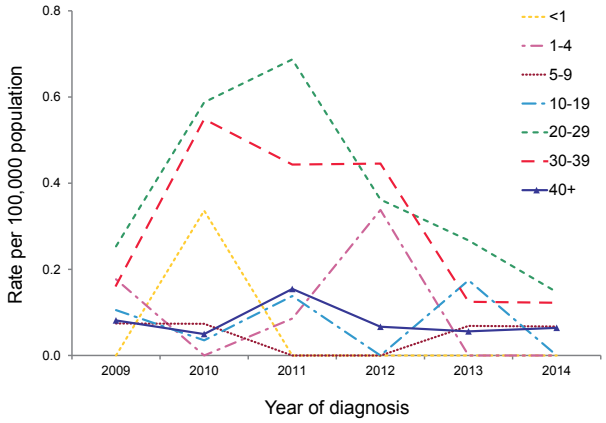


Figure 76: Notification rate for rubella, Australia, 2009 to 2014, by year and selected age groups



12 months and 18 months of age. A dose at 4 years of age was also recommended for those who did not receive the second dose at 18 months of age.[32]

Of the 17 cases notified in 2014, 4 were reported as vaccinated; 1 fully vaccinated, receiving 2 doses of a rubella-containing vaccine; 1 as partially vaccinated, receiving 1 dose; and 2 reported as vaccinated with no dose information provided. Two were reported as not vaccinated and the remaining 11 were of unknown vaccinations status.

The primary aim of immunisation against rubella is to prevent cases of CRS.[103] Two doses of a rubella-containing vaccine are recommended for all non-immune persons born during or since 1966 and who are greater than 18 months of age.

Discussion

Evidence suggests that endemic rubella is well controlled in Australia. A marked decline in rubella notifications since 2003 has seen rates in Australia remain well below the 1.0 per 100,000 population WHO goal indicative of rubella control.[104] The increasing trend in age of notifications likely reflects the declining rates of rubella among children since routine MMR immunisation was implemented and the subsequent achievement of high 2 dose coverage. Males, historically more susceptible as universal vaccination was not introduced until 1989, no longer appear to be at greater risk of infection compared with females.

CRS is rare in Australia and in recent years has mainly occurred among infants of women who were born overseas.[105]

Tetanus

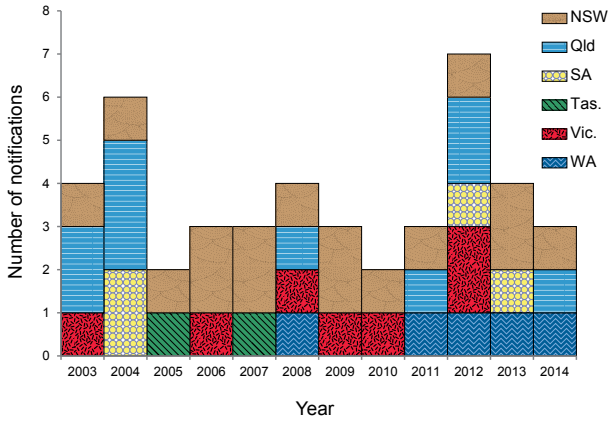
- Cases of tetanus are uncommon in Australia.
- Cases generally occur in older, unvaccinated people or in those who have not received a booster vaccination in the last 10 years.
- In 2014, there were 3 cases of tetanus and no deaths reported.

Tetanus is an acute, often fatal, disease caused by the toxin produced by the bacterium *Clostridium tetani*. *C. tetani* spores usually enter the body through contamination of a wound with manured soil. [22] The neurotoxin acts on the central nervous system to cause muscle rigidity with painful spasms. Generalised tetanus, the most common form of the disease, is characterised by increased muscle tone and generalised spasms. The disease usually occurs after an incubation period of 3 to 21 days (ranging from 1 day to several months), with a median time of onset at 10 days post injury. In Australia, tetanus is rare occurring primarily in older adults who have never been vaccinated or have not received a booster dose in the past 10 years. A high level of diagnostic awareness of tetanus is important in the elderly, as most deaths occur in people over 70 years of age, especially females, and may be associated with an apparent minor injury.[106]

Epidemiological situation in 2014

In 2014, there were 3 notifications of tetanus (Figure 77). This was consistent with the low number of this disease notified in recent years. All cases were adult males 30 years of age or over. Place of acquisition was reported for all cases, with 1 case reported to have acquired their infection in Italy and 2 in Australia. There were no reported deaths due to tetanus in 2014.

Figure 77: Notifications of tetanus, Australia, 2003 to 2014, by year and state or territory



Vaccination status

The NIP schedule in 2014 recommends a primary course of tetanus vaccination including 3 doses provided at 2, 4, and 6 months of age. Two booster doses are provided at 4 years and between 10 and 15 years delivered through school based programs. Booster doses are additionally recommended for all adults at the age of 50 years who have not received 1 in the previous 10 years.[32]

Of the 3 cases notified in 2014, 2 were reported as not vaccinated and 1 was of unknown vaccination status.

Complete immunisation induces protection that lasts throughout childhood but by middle age, 50% of vaccine recipients have low or undetectable levels of antibodies. Tetanus is however uncommon in people who have received 4 or more doses of a tetanus-containing vaccine, and in those who received their last dose within 10 years.[105]

Varicella zoster virus

- In 2014, there were 19,658 cases of varicella zoster virus infection reported, an increase of 16% from 2013.
- Of these, 62% were unspecified VZV infection, 28% were shingles and 11% were chickenpox.

The VZV is a highly contagious member of the herpesvirus family and causes 2 distinct illnesses; chickenpox as the primary infection, and shingles (herpes zoster), which occurs following reactivation, often many years later, of latent virus in approximately 20% to 30% of all chickenpox cases. Shingles occurs more frequently among older adults (most commonly after 50 years of age) and in immunocompromised people.[22]

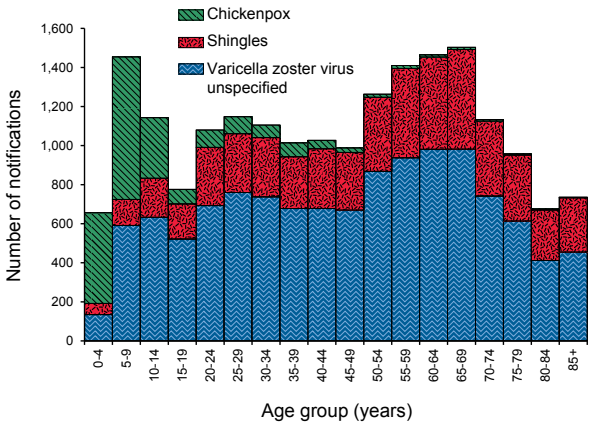
In 2006, the CDNA agreed 3 categories of VZV infection were nationally notifiable: chickenpox, shingles and varicella zoster virus unspecified. By 2009, all jurisdictions were notifying VZV infections to the NNDSS against these 3 categories, except New South Wales, where VZV is not a notifiable disease.

The ability to categorise a VZV infection as chickenpox or shingles depends on follow-up to determine the clinical presentation of the case. The majority of VZV infections are reported as unspecified as follow-up does not occur (Table 5). Notification rates for chickenpox, shingles and VZV unspecified, including any comparisons made between jurisdictions and age groups, should be interpreted with caution as they are affected by the varying levels of follow-up undertaken in each jurisdiction.

Epidemiological situation in 2014

In 2014, there were 19,658 VZV notifications from the 7 reporting jurisdictions. This was 16% more than the total cases notified in 2013 (n=16,986). Of the total VZV notifications in 2014, 62% (n=12,097) were reported as unspecified VZV infection, 28% (n=5,471) as shingles and 12% (n=2,088) as chickenpox (Figure 78).

Figure 78: Notifications of varicella zoster virus infection, 2014, Australia,* by age group†



* Excluding New South Wales.

† Age of onset missing for 123 notifications.

Varicella zoster virus (unspecified)

- In 2014, there were 12,097 cases of VZV unspecified reported, an increase of 10% from 2013.

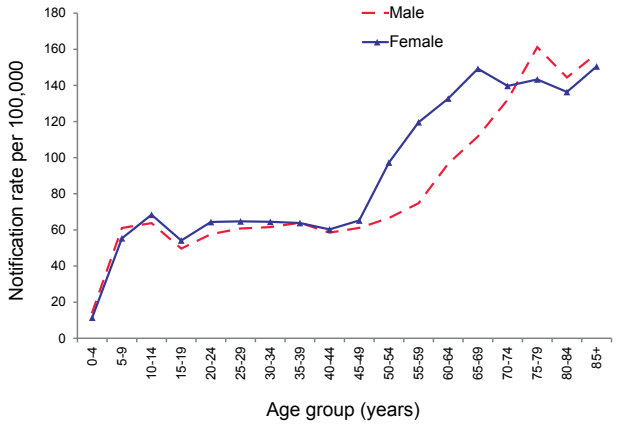
Epidemiological situation in 2014

In 2014, there were 12,097 cases of VZV unspecified infections reported, representing a notification rate of 76 per 100,000 population and a 10% increase in notifications compared with 2013 (n=10,983). The highest notification rate for VZV unspecified was reported from Queensland at 117 per 100,000 (n=5,544) and the lowest from the Northern Territory at 3 per 100,000 (n=8) (Table 5).

Age and sex distribution

In 2014, the majority of VZV unspecified cases were reported in females (54%, n=6,518). Overall, females have a higher notification rate (81 cases per 100,000) compared with males (70 per 100,000), which predominated across all ages except young children (under 10 years of age) and adults aged 75 years or over (Figure 79). The highest age-specific rates for females occurred in the 85 years or over age group (151 per 100,000) and for males in the 75–79 years age group (161 per 100,000). The lowest age-specific rates occurred in the 0–4 years age group for both males and females.

Figure 79: Notification rate for varicella zoster virus unspecified, Australia,* 2014, by age group and sex†



* Excluding New South Wales.

† Age of onset missing for 7 notifications and sex missing for 1 notification.

Chickenpox

- In 2014, there were 2,088 cases of chickenpox reported to the NNDSS, a 2% decrease from 2013 (n=2,127).
- Fifty-three per cent of notified chickenpox cases were male and 72% (n=1,501) occurred in children less than 14 years of age.

Chickenpox is a highly contagious infection spread by respiratory secretions, including aerosol transmission, or from the vesicle fluid of skin lesions from a patient with chickenpox or shingles infection. Chickenpox is usually a mild disease of childhood; however, complications occur in approximately 1% of cases. It is more severe in adults, and in persons of any age who are immunocompromised.[32]

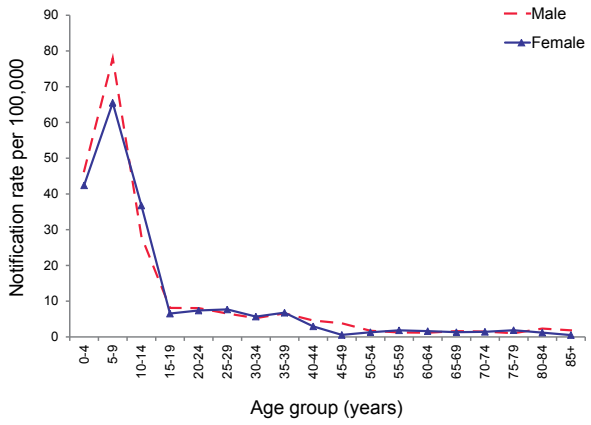
Epidemiological situation in 2014

In 2014, there were 2,088 cases of chickenpox reported, representing a notification rate of 13 per 100,000 population and a 2% decrease in the number of notifications compared with 2013 (n=2,127). The national notification rate of chickenpox has remained stable between 12 and 14 per 100,000 since 2009. The highest notification rate for chickenpox was reported in the Northern Territory (42 per 100,000) (Table 5).

Age and sex distribution

In 2014, 53% (n=1,103) of notified chickenpox cases were male and 72% (n=1,501) occurred in children less than 14 years of age (Figure 80). Consistent with recent years, children under the age of 10 years had the highest notification rates in 2014. Rates were highest in the 5–9 years age group at 72 per 100,000. Compared with 2013, age-group specific rates either declined or remained stable except for the 10–19 years age group which increased from 17 per 100,000 in 2013 to 20 per 100,000 in 2014 (Figure 81).

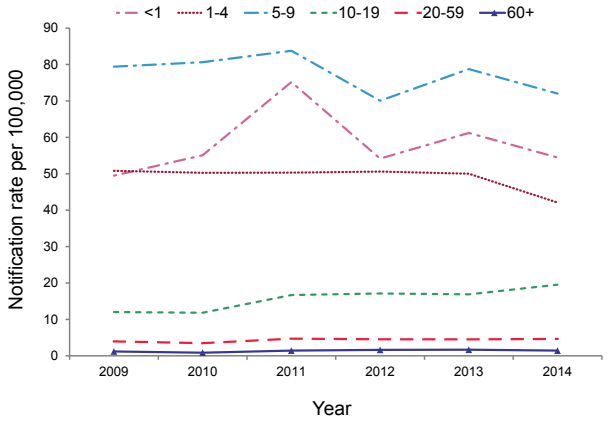
Figure 80: Notification rate for chickenpox, Australia,* 2014, by age group† and sex



* Excluding New South Wales.

† Age of onset missing for 57 notifications.

Figure 81: Notification rate for chickenpox, Australia,* 2009 to 2014, by year and selected age groups†



* Excluding New South Wales.

† Age of onset missing for 4 notifications in 2009, 10 notifications in 2010, 11 notifications in 2011, 22 notifications in 2012, 38 notifications in 2013 and 57 notifications in 2014.

Vaccination

Routine use of a varicella containing-vaccine in children was first recommended in Australia in 2003. In November 2005, the vaccine was funded under the NIP for all children at 18 months of age, with a school based catch-up program included for children 10 to 13 years of age with no history of disease or previous vaccination.

In 2014, the oldest cohort of children eligible for varicella vaccination at 18 months of age under the NIP would be 10 years of age. Of those eligible for vaccination (n=1,068), 41% (n=443) were vaccinated and 9% were unvaccinated (n=95) and the remaining 50% (n=530) were of unknown vaccinations status.

Shingles

- In 2014, there were 5,473 cases of shingles reported to the NNDSS, a 9% increase from 2013.
- Fifty-six per cent of notified shingles cases were female and rates were highest in the older age groups.

Shingles occurs most commonly with increasing age, impaired immunity, and a history of chickenpox in the first year of life.[32] Reactivation of VZV that causes shingles is thought to be due to a decline in cellular immunity to the virus. Shingles typically presents as a unilateral vesicular rash localised in a dermatomal distribution. Associated symptoms may include headache, photophobia, malaise, itching, tingling, or severe pain in the affected dermatome. In the majority of patients, shingles is an acute and self-limiting disease however, complications develop in approximately 30% of cases, the most common of which is chronic severe neuropathic pain or post herpetic neuralgia.[22]

A single dose of zoster vaccine is recommended for adults aged 60 years or over who have not previously received a dose of zoster vaccine. However, in 2014 this vaccination was not yet funded through the NIP. [32]

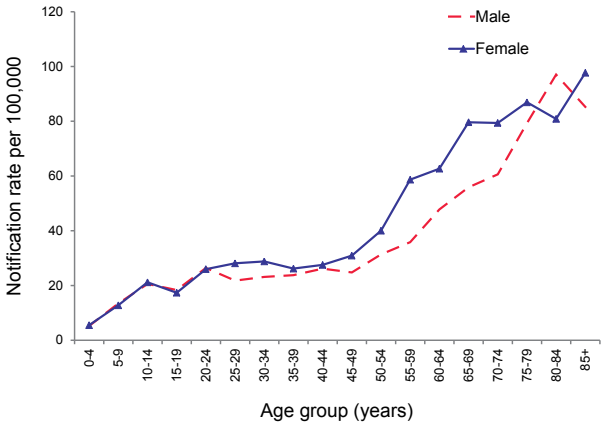
Epidemiological situation in 2014

In 2014, there were 5,473 cases of shingles reported, representing a notification rate of 34 per 100,000 and a 9% increase compared with 2013 (n=5,038). The highest rate of shingles occurred in South Australia, (121 per 100,000) followed by the Northern Territory, (100 per 100,000) (Table 5).

Age and sex distribution

In 2014, 56% (n=3,052) of notified shingles cases were female. As expected, notification rates increased with age, with the highest rates occurring in the 80 years or over age group across all reported years (Figure 82). Since 2009, rates in the adult age groups (greater than 20 years) have been rising, with the largest increase occurring in the 70–79 years age group with a 98% increase in rates between 2009 and 2014 (Figure 83). Rates among children and adolescents have been more stable, remaining below 20 per 100,000 since 2009.

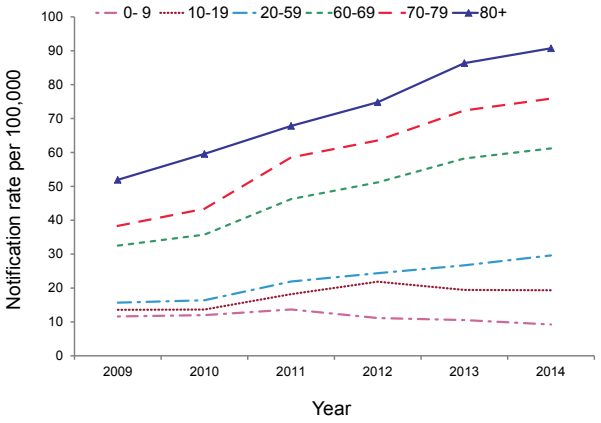
Figure 82: Notification rate for shingles, Australia,* 2014, by age group and sex†



* Excluding New South Wales.

† Age of onset missing for 59 notifications and sex missing for 1 notification.

Figure 83: Notification rate for shingles, Australia,* 2009 to 2014, by year and selected age groups†



* Excluding New South Wales

† Age of onset missing for 1 notification in 2009, 13 notifications in 2010, 18 notifications in 2011, 32 notifications in 2012, 59 notifications in 2013 and 59 notifications in 2014.

Vectorborne diseases

Vectorborne diseases are infections transmitted by arthropods such as mosquitoes and ticks. A vectorborne disease may involve a simple transfer via the arthropod, or may involve replication of the disease-causing organism in the vector.[22] Vectorborne diseases of public health importance in Australia listed in this chapter are: arbovirus not elsewhere classified (NEC); Barmah Forest virus (BFV) infection; dengue virus (DENV) infection; Japanese encephalitis virus (JEV) infection; infections with the Kunjin lineage of West Nile virus (KUNV, which is probably limited to the Australian mainland or possibly Papua New Guinea), and other lineages of West Nile virus (WNV), malaria, Murray Valley encephalitis virus (MVEV) infection and Ross River virus (RRV) infection. Some vectorborne diseases, including yellow fever virus infection, plague and certain viral haemorrhagic fevers, are listed under quarantinable diseases. The National Arbovirus and Malaria Advisory Committee (NAMAC) provides expert technical advice on vectorborne diseases to the Australian Health Principal Protection Committee through CDNA.

Alphaviruses

Viruses in the genus *Alphavirus* that are notifiable in Australia are BFV and RRV. These viruses are unique to the Australasian region.[107] Infection can cause a clinical illness, which is characterised by fever, rash and polyarthritis. The viruses are transmitted by numerous species of mosquito that breed in diverse environments.[108] The alphavirus chikungunya was not nationally notifiable in 2014, and thus not included in this annual report. However, it is notifiable in all states and territories except the Australian Capital Territory, and states and territories send information about cases to the Australian Government for national collation and analysis.[109,110] Chikungunya virus infection was made nationally notifiable in January 2015.

The national case definitions for RRV and BFV currently require only a single IgM positive test to 1 virus, in the absence of IgM to the other.[111] False positive IgM diagnoses for BFV in particular are a known issue, thus it is unclear what proportion of notifications represent true cases. Importantly, the case definitions were reviewed by the CDWG and endorsed by CDNA. The revised case definitions were implemented on 1 January 2016.

Barmah Forest virus infection

- The number of notifications and the notification rate decreased in 2014.
- Numbers and rates had previously increased in 2012 and 2013 due to false positive IgM diagnoses.

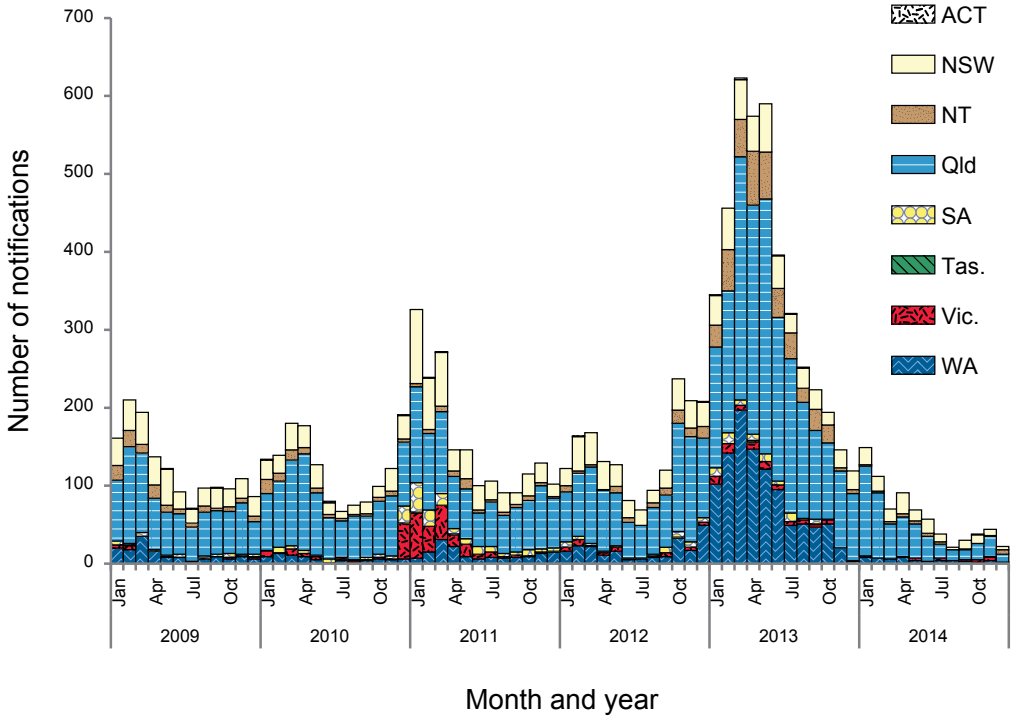
Epidemiological situation in 2014

In 2014, there were 741 notifications of BFV infection, representing a rate of 3.2 per 100,000. This compared with a 5-year mean of 2,155 notifications and a 5-year mean rate of 9.6 per 100,000. The number of notifications of BFV was dramatically decreased compared with 2013, when there were 4,239 notifications, representing a rate of 18.3 per 100,000 (Figure 84). This previous increase in 2013 was considered likely to have been due to a high rate of false positive IgM test results produced by a commercial test kit in private laboratories, and which resulted in a recall of the affected kits in September 2013.[112]

Geographic distribution

Comparisons between regions are likely to be influenced by the accuracy of case-ascertainment, which may vary between jurisdictions due to differences in reporting criteria and diagnostic tests used and seasonal trends vary between states and territories. More details are reported in the NAMAC annual reports.[110] More than half of all BFV notifications in 2014 were from Queensland (64%, 473) and population rates were highest in the Northern Territory (12.3 per 100,000) and Queensland (10.0 per 100,000). These rates were less than half the 5-year mean, with rate ratios of 0.2 and 0.5 respectively for the Northern Territory

Figure 84: Notifications of Barmah Forest virus infection, Australia, 2009 to 2014, by month and year and state or territory

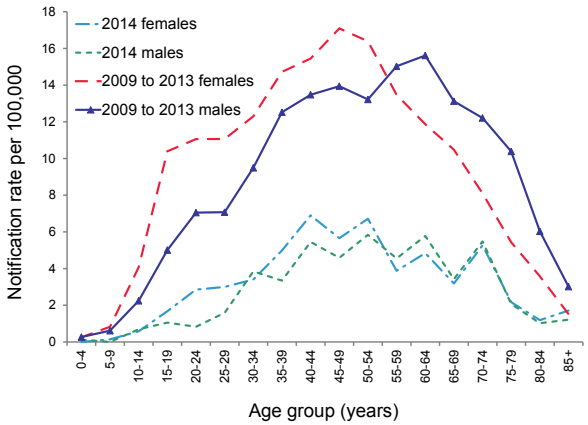


and Queensland for 2014 compared with the 5-year mean rate, noting that the 5-year mean rate is strongly affected by the increase between late 2012 and late 2013.

Age and sex distribution

In 2014, BFV infection was most frequently notified in people aged between 40 and 54 years (median 47 years, range 3 to 92 years). Age and sex specific rates were highest among females in the 40–44 and 50–54 years age groups (Figure 85). In 2014, 54% (403) of cases were female and rates were higher in females overall than in males (3.4 and 2.9 per 100,000 respectively).

Figure 85: Notification rates for Barmah Forest virus, 2014 and 2009 to 2013, by age group and sex



Seasonality

Peak incidence of BFV could be expected to occur during the warmer months (or during wetter months in northern areas of Australia) when mosquito numbers are high. However, seasonality of notifications is less marked than expected (Figure 84), and a high proportion of interseasonal notifications are thought to be due to false positive diagnoses. Peak notification of BFV in 2014 was between January and April, with 57% (422) of notifications being during this period, a stronger seasonality than observed between 2009 and 2012 (45%, 4,898/10,775) and 2013 (47% 1,998/4,239).

Discussion

The previously reported dramatic increase due to false positive IgM diagnoses declined in late 2013. In addition to the withdrawal of some affected batches of the test kit, the widely acknowledged unreliability of the IgM test kit may have led to a sustained decline in testing for BFV infection in private laboratories, which may not have an alternative diagnostic method to the IgM test kits. On recommendation from NAMAC, the CDWG undertook a review of the surveillance case definitions for BFV infection and for RRV infection. The CDNA surveillance case definition for BFV in 2014 allowed for confirmation based on a single positive IgM, in the absence of IgM to other alphaviruses. Under the revised case definition, a single IgM positive result will no longer constitute

laboratory evidence for infection, and where a single result is IgM and IgG positive, it may be notified as a probable case. A confirmed case will require IgG seroconversion or a significant increase in IgG antibody level (e.g. 4-fold or greater rise in titre). There is currently no plan to undertake a retrospective revision of notifications to apply the new case definitions because there is insufficient information on the diagnosis method information available in NNDSS. Therefore, the historical data prior to the upcoming change of case definition will continue to be considered unreliable. The new case definition was implemented from 1 January 2016.[111]

Ross River virus infection

- Notifications of RRV infections were similar to the 5-year mean.

Epidemiological situation in 2014

In 2014, there were 5,316 notifications of RRV, representing a rate of 22.6 per 100,000. This compares with a 5-year mean of 4801.0 cases and a 5-year mean rate of 21.5 per 100,000.

Geographic distribution

In 2014, similar to previous years, nearly half of all RRV infections were from Queensland (44%, 2,344), representing a rate of 49.6 per 100,000), but population rates were highest in the Northern Territory (168.4 per 100,000).

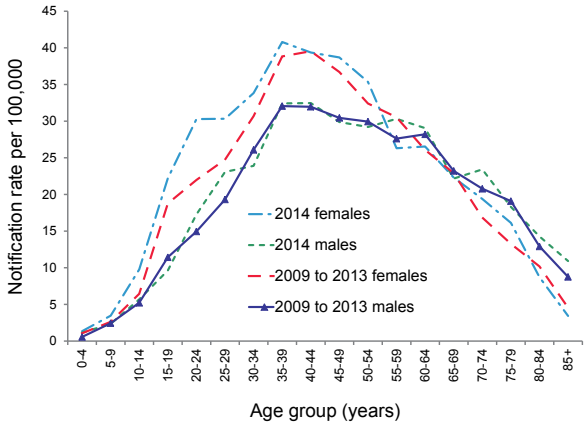
Age and sex distribution

RRV was most frequently reported in adults aged in their 30s or 40s (median 44 years, range 1 to 98 years), similar to previous years. Rates were similar in females and males (rates of 24.9 and 20.4 per 100,000 respectively for a rate ratio of 1.2), similar to previous years. In 2014, age specific rates were highest among females in the 35–49 years age group, and males in the 35–44 years age group (Figure 86).

Seasonality

The peak notification for RRV in 2014 was between January and April (Figure 87), and 39% (2,077) of cases were diagnosed during these months. Between 2009 and 2012, 58% (11,358/19,689) of notifications were between January and April, indicating that in 2014, similar to 2013,[37] the proportion of inter-seasonal notifications was higher than in previous years. It is important to note that seasonal trends vary between and within states and territories according to differences in mosquito vectors, hosts and climate. In addition, comparisons between regions are likely to be influenced by the accuracy of case-ascertainment, which may vary between jurisdictions because of some differences in reporting criteria and also quality of diagnostic tests used, with false positive IgMs a long term issue.[113,114]

Figure 86: Notification rates for Ross River virus, Australia, 2014, by age group and sex



Discussion

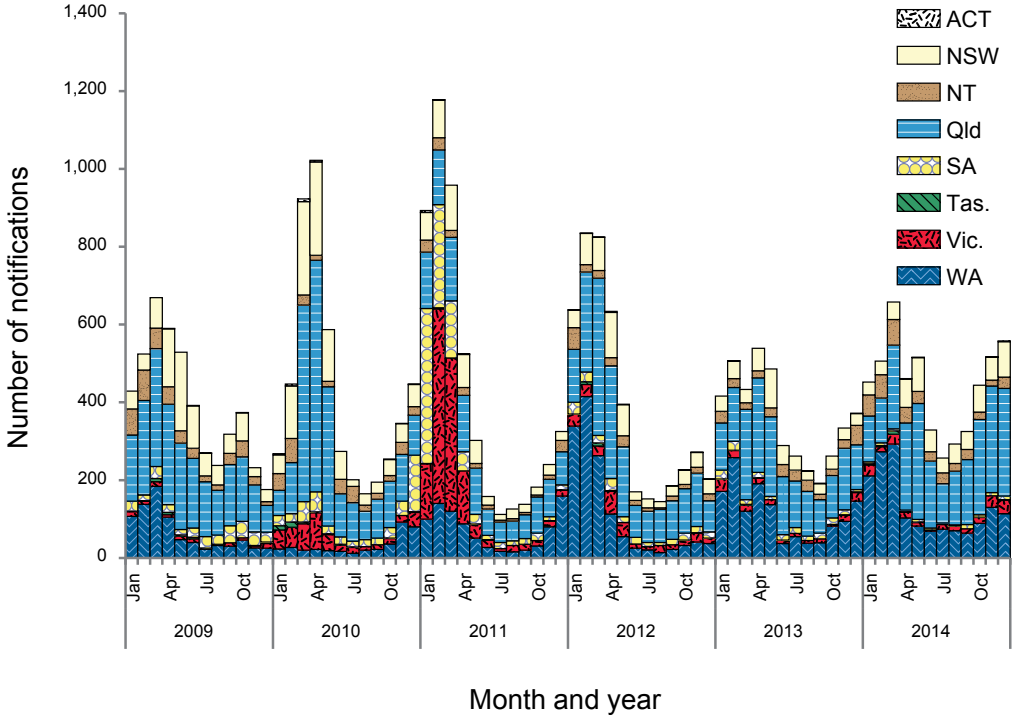
The CDNA surveillance case definition for RRV in 2014 allowed for confirmation based on a single positive IgM, in the absence of IgM to other alphaviruses, and there have been differences in laboratory and notification practices. Similar to BFV, there has been a widely acknowledged unreliability of diagnosis based on IgM-only, particularly during the off-season.[114] As mentioned under BFV, the case definition was reviewed by the CDWG. Under a revised definition a confirmed case requires IgG seroconversion or a significant increase in IgG antibody level (e.g. 4-fold or greater rise in titre). The revised definition was implemented on 1 January 2016.

Flaviviruses

No specific treatment is available for infections with the flaviviruses DENV, WNV/KUNV, MVEV or JEV and care is largely supportive. A vaccine is available to prevent JEV infection[32] and YFV infection, but there are no vaccines currently for DENV, MVEV or KUNV infection.

Infection with MVEV, KUNV or JEV is usually asymptomatic or produces a non-specific illness, but a small percentage of cases progress to encephalomyelitis of variable severity. *Culex annulirostris* is the major vector of MVEV, JEV and KUNV while *Aedes aegypti* is the major vector of DENV in Australia.

Figure 87: Notifications of Ross River virus, Australia, 2009 to 2014, by month and year and state or territory



The clinical illness for DENV infection illness is characterised by mild to severe febrile illness with fever, headache, muscle/joint pain and sometimes a rash. A minority of cases progress to severe dengue with haemorrhage and shock. DENV has 4 serotypes, each containing numerous genotypes. The serotypes isolated from returning travellers (and thus involved in local outbreaks) vary by year and geographical region. Infection with 1 serotype probably confers lifelong immunity to that serotype,[22] but subsequent infection with a different serotype is 1 factor thought to increase the risk of severe outcomes, along with the infecting serotype and genotype, and host factors.[22,115–117]

Arbovirus NEC /Flavivirus unspecified

- There were 28 of notifications arbovirus NEC in 2014.

Unspecified flavivirus infections are reported under arbovirus NEC. From 2015, arbovirus NEC has been renamed flavivirus unspecified.

Epidemiological situation in 2014

In 2014, there were 28 notifications of arbovirus NEC, compared with an average of 12.0 during the previous 5 years. Most notifications were from Queensland (22), with 5 from New South Wales and 1 from Victoria. These notifications comprised Kokobera (1 cases) and Zika virus (ZIKV) infection and probable ZIKV infection (16 cases) and 11 other notifications for which the infecting flavivirus could not be determined or was not supplied (Table 19). Of particular note were the 11 ZIKV and 1 probable ZIKV infections in March and April acquired in the Cook Islands and 1 in February acquired in Samoa. The first report of an outbreak of ZIKV in the South Pacific was through PacNet[118] in February 2013, and reported in the Cook Islands. Outbreaks were later reported in New Caledonia and French Polynesia and these continued to mid 2014.

Information about the country of acquisition was available for 93% of cases (26) and all of these were acquired overseas (Table 19).

The median age of cases was 39 years (range 18 to 79 years) and 46% of cases (13).

Dengue virus infection

- There was a continuing increase in the number of overseas acquired cases.
- There were 186 cases acquired in Australia in 2014, all of them acquired in North Queensland.

Local transmission of DENV in Australia is normally restricted to areas of northern Queensland where the key mosquito vector, *Ae. aegypti* is

Table 19: Notifications of arbovirus NEC, Australia, 2014, by country of acquisition and infecting species

Country of acquisition	Virus species				Total
	Kokobera	Zika	Zika (probable)*	Unspecified	
Australia	1	0	0	0	1
Central and West Africa, nfd	0	0	0	1	1
Cook Islands	0	10	1	1	12
Fiji	0	0	0	1	1
French Polynesia	0	1	0	0	1
Indonesia	0	0	0	2	2
Maldives	0	1	0	0	1
Papua New Guinea	0	0	0	2	2
Philippines	0	0	0	1	1
Samoa	0	1	0	0	1
Sub-Saharan Africa, nfd	0	0	0	1	1
Thailand	0	1	0	0	1
Vanuatu	0	0	0	1	1
Unknown	0	1	0	1	2
Total	1	15	1	11	28

nfd Not further defined.

* This case was a confirmed flavivirus infection, and there was some laboratory evidence of the infecting species being Zika virus, but this was not conclusive.

present.[119] DENV is not endemic in North Queensland, but local transmission can occur upon introduction of the virus to the mosquito vector by a viraemic tourist or a resident returning from a dengue-affected area overseas.[120]

Epidemiological situation in 2014

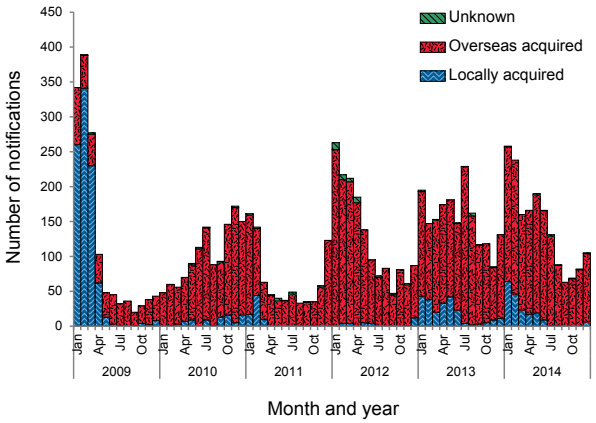
There were 1,716 notifications of DENV infection in 2014, which was 1.3 times the 5-year mean of 1,366.4 notifications. Most infections were known to have been acquired overseas (n=1,520) (Figure 88). There were 186 infections acquired in Australia. For 10 cases, no information was supplied on the place of acquisition.

Geographic distribution

More than 99% (1,706) of notifications in 2014 contained complete information on the place of acquisition. Overseas acquired infections comprised 89% of notifications (1,520) (Table 20). The number of overseas-acquired infections was slightly lower than in 2013 (n=1,596), which was the largest number ever reported (Table 21).

Cases acquired in Indonesia continue to account for the largest number and proportion of all notifications, accounting for 53% (811/1,520) of all overseas-acquired cases in 2014 (Table 21). This was similar to the percentage in 2012 and 2013. The serotype of DENV infections acquired in Indonesia, where known, was frequently serotype 2 or 3 although data completeness for serotype was very low. Other frequently reported source countries in 2014 included Thailand, Fiji and Malaysia.

Figure 88: Notifications of dengue virus infection, Australia, 2009 to 2014, by month and year and place of acquisition



In Queensland, a single case of locally acquired DENV infection is considered an outbreak. All of the of the 186 locally acquired cases in 2014 were reported in NNDSS to have been associated with 1 of the outbreaks of locally acquired infection in Queensland in 2014 and/or acquired in North Queensland and reported by other states or territories.

Table 20: Notified cases of dengue virus, Australia, 2014, by serotype and place of acquisition

Place of acquisition	Serotype						Unknown/ untyped	Total
	DENV 1	DENV 1 and 3	DENV1 And 4	DENV 2	DENV 3	DENV 4		
Locally-acquired								
Australia	130	0	0	0	1	0	55	186
Overseas-acquired								
Indonesia	267	2	0	70	52	21	399	811
Thailand	16	0	0	19	1	7	96	139
Fiji	2	0	0	9	25	0	70	106
Malaysia	8	0	0	16	2	2	53	81
Philippines	5	0	0	11	2	6	42	66
Sri Lanka	7	0	0	1	0	2	30	40
India	2	0	0	5	1	1	29	38
Timor-Leste	12	0	0	0	4	0	20	36
Vanuatu	1	0	1	0	12	1	18	33
Singapore	8	0	0	0	0	0	7	15
Tonga	0	0	0	0	3	0	12	15
Vietnam	1	0	0	4	0	1	9	15
Nauru	0	0	2	0	5	0	4	11
Papua New Guinea	0	0	0	3	4	0	4	11
Cambodia	0	0	0	1	0	1	8	10
French Polynesia	2	0	0	0	0	0	8	10
Other countries	6	0	2	9	2	0	61	80
Overseas-country unknown	0	0	0	0	0	0	3	3
Total overseas-acquired	337	2	5	148	113	42	873	1,520
Unknown								
Place of acquisition unknown	4	0	0	1	0	0	5	10
Total	471	2	5	149	114	42	933	1,716

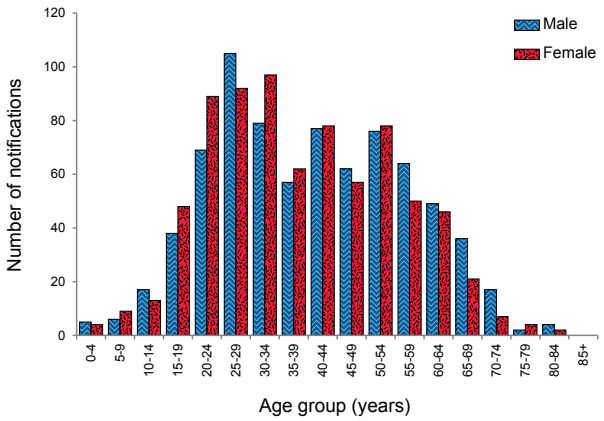
Table 21: Notifications of dengue virus infection acquired overseas, Australia, 2009 to 2014, by selected countries of acquisition

Country of acquisition	Year of diagnosis						Total
	2009	2010	2011	2012	2013	2014	
Indonesia	172	717	461	804	801	811	3,766
Thailand	25	124	84	279	269	139	920
Fiji	8	1	6	32	14	106	167
Malaysia	16	17	21	20	53	81	208
Philippines	9	42	24	55	63	66	259
Sri Lanka	0	4	12	26	28	40	110
India	15	43	31	60	58	38	245
Timor-Leste	25	37	12	52	49	36	211
Vanuatu	10	4	0	0	5	33	52
Singapore	1	4	5	3	18	15	46
Tonga	15	1	0	0	0	15	31
Vietnam	20	34	14	21	25	15	129
Papua New Guinea	13	21	15	16	35	11	111
Nauru	0	0	0	0	0	11	11
French Polynesia	3	0	0	0	5	10	18
Cambodia	5	11	6	31	31	10	94
Other/unknown countries	137	80	42	76	142	83	560
Total	474	1,140	733	1,475	1,596	1,520	6,939

Age and sex distribution

DENV infections acquired overseas in 2014 were most commonly reported among younger and middle aged adults (median 38 years, range 0 to 83 years), with a slight peak of notifications among females aged 25–34 years and males aged 25–29 years (Figure 89). Females comprised 50% (757) of overseas acquired cases.

Figure 89: Notifications of overseas-acquired dengue virus infection, 2014, by age group and sex (n=1,520)

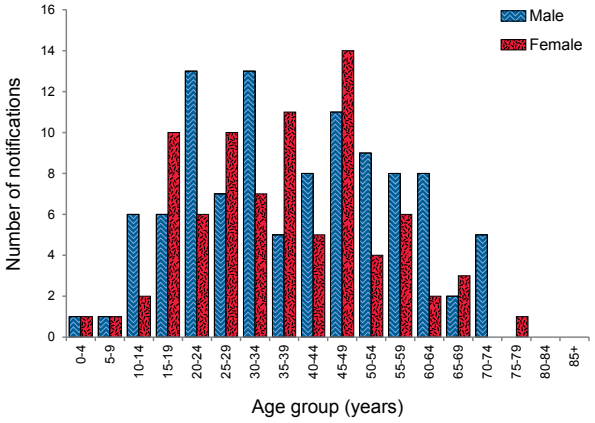


Locally acquired cases peaked in several adult age groups, but was less common among people aged less than 15 years or more than 64 years (Figure 90). The median age of locally-acquired cases was 38 years (range 1 to 75 years). Females comprised 45% (83/186) of locally-acquired cases.

Seasonality

In 2014, the largest number of overseas-acquired cases were diagnosed in January (193 cases) and the lowest number in September and October (63 and 68 cases respectively), but there is no consistent pattern of seasonality from year to year (Figure 88). For locally acquired cases, only 11 cases were reported between June and November demonstrating that there is no on-going local transmission of dengue during the cooler months and that DENV is not endemic in North Queensland.

Figure 90: Notifications of dengue virus infection acquired in Australia, 2014, by age group and sex (n=186)



Microbiological trends

In 2014, serotype information was available for 46% of notifications (783/1,716), which was a decrease compared with the 5-year mean of 53% (Table 22). In 2014, 60% (471/783) of cases with a known serotype were due to DENV 1, similar to 2013, but in contrast to 2012 when DENV2 was more frequently reported, noting the low completeness (Table 22).

Discussion

The number of overseas-acquired cases reported in Australia has tended to increase each year, although numbers in 2014 were lower than the previous year. In recent years, improved diagnostic techniques,

Table 22: Serotype of dengue virus infections, Australia, 2009 to 2014

Serotype	2009	2010	2011	2012	2013	2014
DENV1	82	190	140	138	507	471
DENV 1 and DENV 3	0	0	0	0	0	2
DENV 1 and DENV 4	0	0	0	1	0	5
DENV 2	54	255	159	477	179	149
DENV 3	771	106	78	113	142	114
DENV 4	43	47	43	16	55	42
Untyped/unknown	452	630	401	796	957	933
Total	1,402	1,228	821	1,541	1,840	1,716
% with a serotype supplied	68	49	51	48	48	46

in particular the availability of the rapid non-structural protein 1 (NS1) antigen detection kit, have improved detection and would have contributed to the observed increase in reported numbers of overseas-acquired dengue in Australia.[121] The dramatic re-emergence and geographical expansion of DENV overseas over the past 50 years with explosive outbreaks,[117] as well as increases in the number of Australians travelling overseas each year to DENV endemic countries, particularly tourist destinations such as Bali, Indonesia would also have contributed to this increase.

While local outbreaks of DENV infection occur during the warmer months in North Queensland, each outbreak since 2010 has been relatively small, and prompt and effective responses by public health authorities in Queensland have ensured that the disease has not become endemic.

The number of DENV infections that are serotyped continues to decline. The decreased reporting of a serotype may reflect the increasing use of NS1 antigen detection and/or other diagnostic methods, which do not provide a serotype.

Japanese encephalitis virus infection

- In 2014, there was 1 notification of JEV.

Epidemiological situation in 2014

There was 1 notification of JEV infection in 2014, which was reported to have been acquired in Indonesia. There were 4 notifications in 2013, all of which were acquired overseas. The last locally acquired case was in 1998.[122]

West Nile virus/Kunjin virus infection

- In 2014, there was 1 notification of WNV/KUNV.

Epidemiological situation in 2014

There was 1 notification of WNV/KUNV infection in 2014, which was reported to have been acquired in Indonesia. This overseas-acquired case was likely to be from a WNV lineage other than the KUNV lineage due to the limited range of KUNV. There were 2 notifications of WNV/KUNV infection in 2013 (previously published as 3 notifications[37] but this number has since been updated).

Murray Valley encephalitis virus infection

- In 2014, there was 1 notification of MVEV.
- MVEV is rare disease in Australia, but may also be acquired overseas in the region.

Epidemiological situation in 2014

There were no notifications of MVEV infection in 2014. MVEV is a rare disease in Australia with a 5-year mean of 4.4 cases.

The largest number of cases in recent years was in 2011, when 16 cases were reported, including an outbreak in south-east Australia, and these have been described elsewhere.[123–126]

Malaria

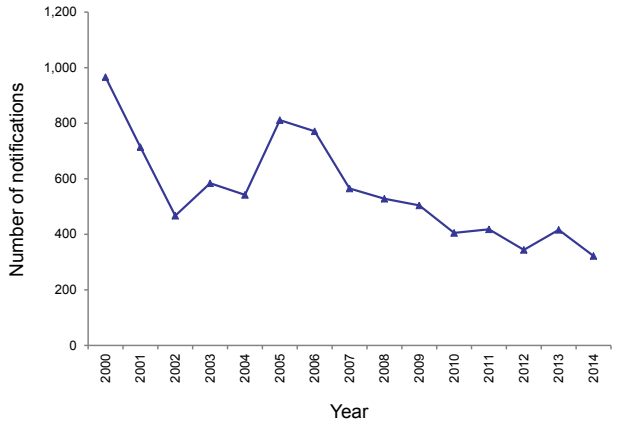
- There were 322 notifications of malaria in 2014, all were acquired overseas.
- The gradual decline observed since 2005 is continuing.

Malaria is caused by a protozoan parasite in the genus *Plasmodium*, and 5 species are known to infect humans; *Plasmodium vivax, P. falciparum, P. malariae, P. ovale* and *P. knowlesi*.[22,127] Malaria is a serious acute febrile illness that is transmitted from person-to-person via the bite of an infected mosquito of the genus *Anopheles*. Australia is free of endemic malaria as declared in 1981,[128] but suitable vectors are present in northern Australia, and the area remains malaria-receptive. Malaria is the most frequently reported cause of fever in returned travellers worldwide.[129] A case series in the Northern Territory showed that malaria cases were reported in travellers returning from endemic areas, but also reflected current events such as military operations and increased refugee arrivals from malaria endemic areas.[130] Malaria cases in Australia can be found either through testing of symptomatic persons with a compatible travel history, or through screening of refugees who may be asymptomatic, and people may be tested for other reasons.

Epidemiological situation in 2014

There were 322 cases of malaria notified in Australia in 2014, a 23% decrease compared with a 5-year mean of 417.4 cases, and continuing the trend of gradually decreasing notifications since 2005 (Figure 91). The largest number of cases was reported by Queensland (86 cases).

Figure 91: Notifications of malaria, Australia 2000 to 2014, by year

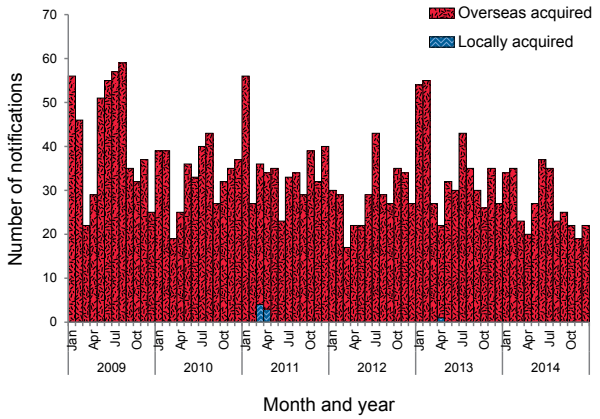


Geographic distribution

Malaria in Australia is a disease associated with residing or travelling overseas in areas with endemic transmission. The last cases acquired on mainland Australia were during an outbreak in North Queensland in 2002.[131] Limited transmission occurs occasionally in the Torres Strait following importation, with the most recent being a single case in 2013 acquired on Saibai Island in the Torres Strait and 7 locally acquired cases in the Torres Strait in 2011.

All cases of malaria notified in 2014 were known to have been acquired overseas; however, for 12 cases (4%) the country of acquisition information was incomplete or missing. The most frequent countries of acquisition for overseas acquired cases in 2014 were India (13%, 39/310 of cases with complete information) and Papua New Guinea (10%, 31/310) (Table 23) (Figure 92). Most cases acquired in Papua New Guinea were reported by Queensland (19/31 cases).

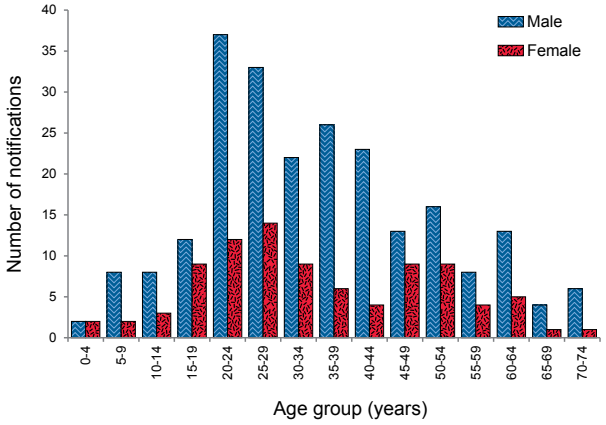
Figure 92: Notifications of malaria, Australia, 2009 to 2014, by month and year and place of acquisition



Age and sex distribution

In 2014, malaria was most commonly reported in males (72%, 232 cases) with a peak of notifications in males in the 25–29 years age group (Figure 93). The median age of cases was 32 years (range 2 to 72 years).

Figure 93: Notified cases of malaria, Australia, 2014, by age group and sex (n=321)*



* Age was not reported for 1 case and this case is excluded.

Seasonality

Increases in notifications or an observable pattern of seasonality in a predominantly overseas-acquired infection can relate to the seasonality of travel patterns, or to local disease epidemiology in the source countries. In 2014, while there were some fluctuations in monthly notifications, there was no clear pattern of seasonality, with notifications ranging between 19 and 37 per month.

Microbiological trends

The infecting species was supplied for 95% (307/322) of cases in 2014 (Table 23). The most frequent infecting species was *P. falciparum* (reported in 51% of cases with complete information). *P. vivax* was associated with Asia and the Pacific, whilst most cases acquired in African countries were *P. falciparum*.

Zoonoses

Overview

Zoonoses are those infectious diseases that are naturally transmitted between vertebrate animals and humans.[132] Approximately 60% to 70% of emerging

Table 23: Notified cases of malaria, Australia 2014, by infecting species and region and country of acquisition

Country	*P. falciparum*	*P. malariae*	*P. ovale*	*P. vivax*	*P.* species	Total
Oceania						
Papua New Guinea	7	1	0	22	1	31
Solomon Islands	0	0	0	4	1	5
North Africa and the Middle East						
Egypt	0	1	0	0	0	1
Sudan	23		3	2	1	29
Western Sahara	1	0	0	0	0	1
South Sudan	3	0	0	0	0	3
United Arab Emirates	1	0	0	0	0	1
South-east Asia						
Myanmar, The Republic of the Union of	0	0	0	1	0	1
Cambodia	0	0	0	4	0	4
Laos	0	0	0	1	0	1
Thailand	0	0	0	1	0	1
Brunei Darussalam	0	1				1
Indonesia	5	0	2	14	1	22
Malaysia	1	0	0	0	0	1
North-east Asia						
China (excludes SARs and Taiwan)	0	0	0	1	0	1
Korea, Republic of (South)		0	0	1	0	1
Southern and Central Asia						
India	0	0	0	35	4	39
Pakistan	0	0	0	12	2	14
Afghanistan	0	0	0	1	0	1
Peru	1	0	0	1	0	2
Sub-Saharan Africa						
Sub-Saharan Africa, nfd	5	2	0	0	1	8
Central and West Africa, nfd	1	0	0	0	0	1
Burkina Faso	0	0	1	0	0	1
Cameroon	1	0	0	0	0	1
Chad	1	0	0	0	0	1
Congo, Republic of	5	0	0	0	0	5
Congo, Democratic Republic of	1	0	0	0	0	1
Cote d'Ivoire	1	0	1	0	0	2
Gabon	1	0	0	0	0	1
Ghana	13	0	1	1	0	15
Guinea	1	0	0	0	0	1
Guinea–Bissau	1	0	0	0	0	1
Liberia	3	1	0	0	0	4
Mali	3	0	0	0	0	3
Nigeria	5	0	0	0	0	5
Sierra Leone	12	0	3	0		15
Togo	2	0	0	0	0	2

Table 23 *continued*: Notified cases of malaria, Australia 2014, by infecting species and region and country of acquisition

Country	*P. falciparum*	*P. malariae*	*P. ovale*	*P. vivax*	*P.* species	Total
Southern and East Africa						
Southern and East Africa, nfd	1	0	0	0	0	1
Burundi	1	0	0	0	0	1
Eritrea	0	0	2	1	0	3
Ethiopia	2	0	0	3	0	5
Kenya	12	2	2	0	1	17
Malawi	1	0	0	0	0	1
Mozambique	2	0	1	0	0	3
Rwanda	1	0	0	0	0	1
South Africa	2	0	0	0	0	2
Tanzania	11	0	0	0	1	12
Uganda	10	3	2	4	0	19
Zambia	7	0	3	0	0	10
Zimbabwe	3	0	0	0	0	3
Southern and East Africa, nec	4	0	0	0	1	5
Overseas acquired – country and region not stated						
Unknown/invalid	2	0	1	6	0	9
Overseas-country unknown	1	0	0	1	1	3
Total	158	11	22	116	15	322

nfd Not further defined.

human infectious diseases are zoonoses[133–135] and more than 70% of emerging zoonoses originate from wildlife.[134] An emerging zoonosis is defined by WHO as "a zoonosis that is newly recognised or newly evolved, or that has occurred previously but shows an increase in incidence or expansion in geographical, host or vector range".[136]

The zoonoses notifiable to the NNDSS included in this chapter are: anthrax, Australian bat lyssavirus (ABLV) or lyssavirus (unspecified) infection, brucellosis, leptospirosis, ornithosis, Q fever, and tularaemia.

Several zoonoses notifiable to the NNDSS are included under other headings in this report. For example, salmonellosis and campylobacteriosis are typically acquired from contaminated food and are listed under the gastrointestinal diseases section. Rabies is listed under Quarantinable diseases.

Anthrax

- There were no cases of anthrax notified in 2014.

Anthrax is caused by the bacterium *Bacillus anthracis* and most frequently causes cutaneous infection. However, it can also cause gastrointestinal and respiratory infections. Anthrax is primarily a disease of herbivores; humans and carnivores are incidental hosts. It can be an occupational hazard for veterinarians, and agriculture, wildlife and livestock workers who handle infected animals or by-products.

In Australia, the areas of anthrax risk are well defined and include the northern and north-eastern districts of Victoria and central New South Wales.[137] Anthrax occurs only sporadically in livestock in the at-risk areas. Rare or isolated incidents or cases in animals have historically occurred in Queensland, South Australia, Tasmania and Western Australia.[137]

Epidemiological situation in 2014

In 2014, there were no notified cases of anthrax in Australia. Over the previous 10 years, only 3 human cases of anthrax were reported in Australia in 2006, 2007 and 2010.[138–140] All had domestic farm or animal related exposures and all were cutaneous anthrax. Australia has never recorded a human case of inhalational or gastrointestinal anthrax.

There was 1 anthrax incident reported in livestock in Australia in 2014. It occurred on a property located within the known New South Wales anthrax endemic area.[137]

Australian bat lyssavirus and lyssavirus (unspecified)

- No cases of ABLV notified in 2014.

ABLV belongs to the genus lyssavirus, which also includes the rabies virus. Both invariably result in progressive, fatal encephalomyelitis in humans.[141] ABLV was first identified in Australia in 1996[142,143] and is present in several Australian species of bats (including flying foxes and microbats). Australia is free of terrestrial rabies.

The best way to prevent ABLV infection is to avoid contact with bats. For people whose occupation (including volunteer work) or recreational activities place them at increased risk of being exposed to ABLV, rabies virus vaccine is effective in preventing infection. Pre-exposure vaccination with rabies virus vaccine is recommended for bat handlers, veterinarians and laboratory personnel working with live lyssaviruses.[144] Post-exposure prophylaxis for ABLV consists of wound care and administration of a combination of rabies virus vaccine and human rabies virus immunoglobulin, depending on exposure category and prior vaccination or antibody status.[32,144]

Epidemiological situation in 2014

In 2014 there were no notified cases of ABLV or lyssavirus (unspecified) infection in Australia.

There have been 3 cases of ABLV infection recognised in humans in Australia, with single cases notified in each of 1996, 1998 and 2013. All 3 cases occurred following close contact with an infected bat in Queensland and all were fatal.[145–147] In 2013, the Queensland Department of Agriculture, Fisheries and Forestry confirmed the first known equine cases of ABLV infection in 2 horses on a Queensland property.[148,149]

The bat health focus group of Wildlife Health Australia (formerly the Australian Wildlife Health Network) gathers and collates information from a range of organisations on opportunistic testing of bats for ABLV. In 2014 there were 32 ABLV detections in bats compared with 14 detections during 2013.[150]

Brucellosis

- In 2014, 17 cases of brucellosis were notified to the NNDSS.

Brucellosis is characterised by a fever of variable duration with a range of other symptoms including headache, weakness, profuse sweating, chills, arthralgia, depression, weight loss and generalised aching.[22] *Brucella* species that can cause illness in humans include *Brucella melitensis* acquired from sheep and goats, *Brucella suis* from pigs and *Brucella abortus* from cattle. *B. abortus* was eradicated from Australian cattle herds in 1989 and *B. melitensis* has never been reported in Australian sheep or goats.[137] Therefore, all cases of *B. melitensis* or *B. abortus* in Australia are related to overseas travel. *B. suis* is confined to some areas of Queensland, where it is known to occur in feral pigs. Eales et al (2010)[151] found that feral pig hunting was the most common risk factor for brucellosis in Townsville during 1996 to 2009.

Internationally, brucellosis is mainly an occupational disease of farm workers, veterinarians, and abattoir workers who work with infected animals or their tissues.[22]

Epidemiological situation in 2014

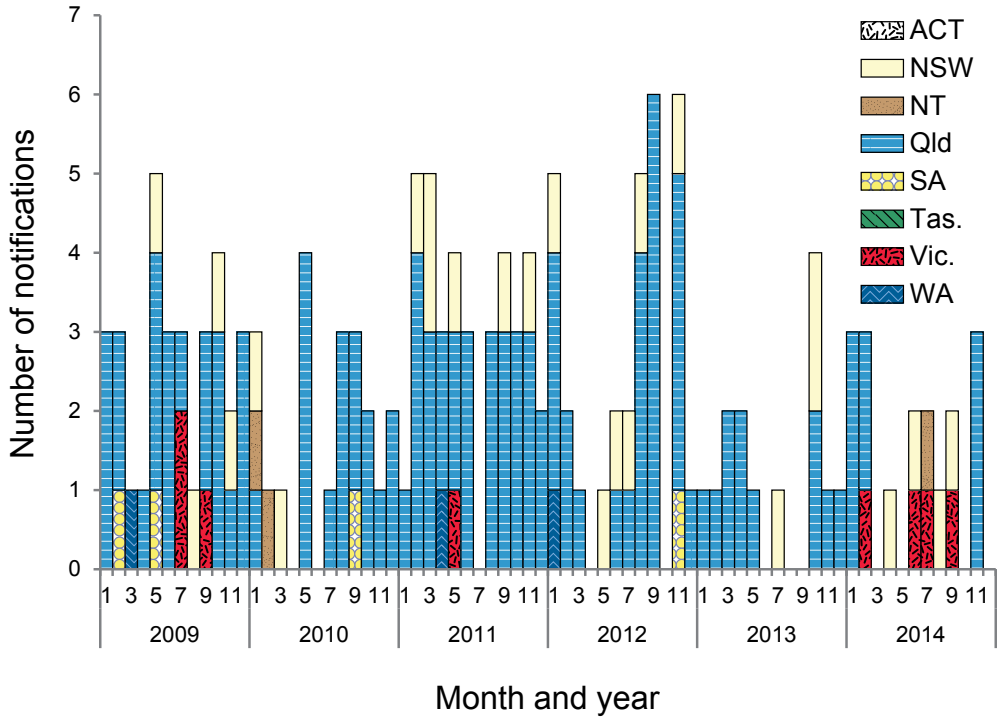
In 2014, there were 17 notified cases of brucellosis in Australia (0.1 per 100,000), representing a 37% decrease compared with the 5-year (2009 to 2013) mean (n=27).

Geographical distribution

Just under half of notified cases (47%, 8) were Queensland residents (Figure 94), with a state-specific notification rate of 0.2 per 100,000 and since 1991, 82% of notifications have been Queensland residents.

The species of the infecting organism was available for 71% (12/17) of notified cases in 2014. There were 3 cases of *B. suis,* all from Queensland, and all males aged between 30 and 46 years. There were 9 cases of *B. melitensis*, with the countries of acquisition listed as India (n=4), Iraq (n=2), Lebanon (n=1), Pakistan (n=1) and Sudan (n=1). The 5 remaining cases where the infecting organism was not specified, were all acquired in Australia.

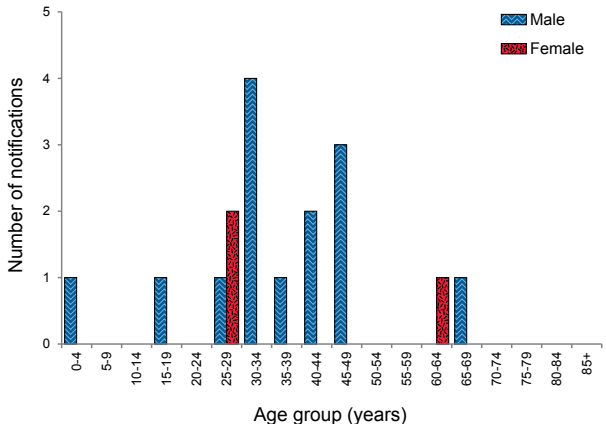
Figure 94: Notifications of brucellosis, Australia, 2009 to 2014, by month and year of diagnosis and state or territory



Age and sex distribution

The majority of notified cases (76%, 13/17) were aged between 25 and 49 years (Figure 95). In 2014, the median age of notified brucellosis cases was 34 years (range 3 to 66 years) and 82% (14) were male.

Figure 95: Notifications of brucellosis, Australia, 2014, by age group and sex



Leptospirosis

- In 2014, 88 cases of leptospirosis were notified to the NNDSS.

Leptospirosis can cause a variety of illnesses varying in severity from a mild influenza-like illness to Weil's syndrome, meningitis or pulmonary haemorrhage with respiratory failure possibly leading to death.[22] Leptospirosis is caused by spirochaetes of the genus *Leptospira,* which is found in the genital tract and renal tubules of domestic and wild animals. In affected areas, where there is exposure to infected urine of domestic and wild animals, this disease can be an occupational and recreational hazard (such as in certain agricultural sectors and swimming or wading in contaminated water).[152,153] The last reported death in Australia attributed to leptospirosis was in 2002.[154]

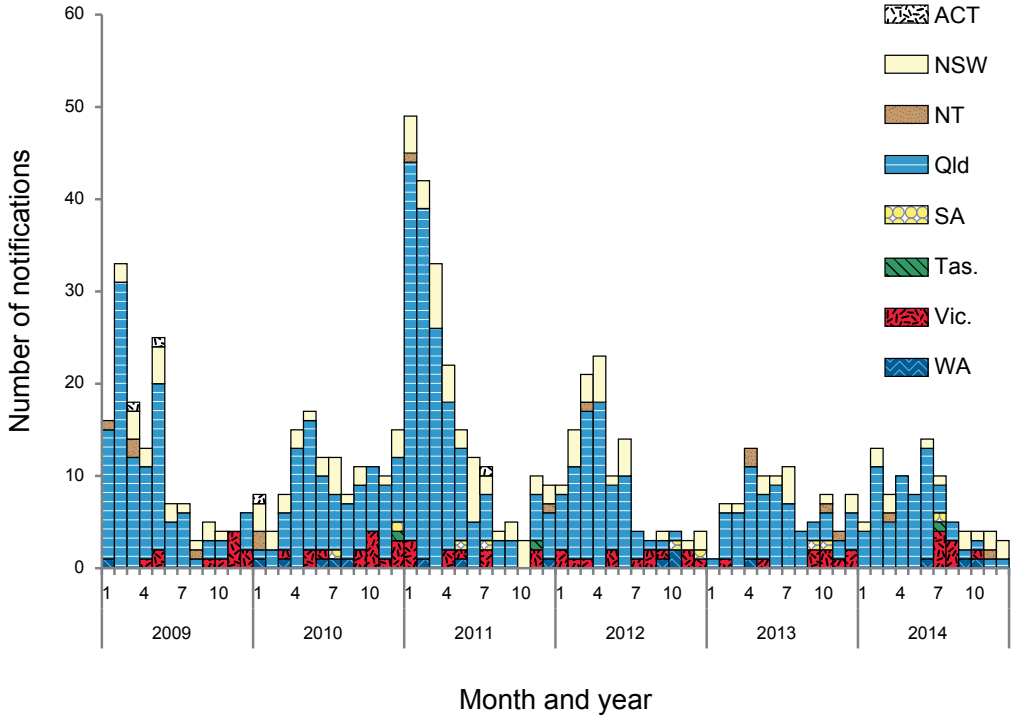
Epidemiological situation in 2014

In 2014, there were 88 notified cases of leptospirosis in Australia (0.4 per 100,000), which was a 36% decrease compared with the 5-year mean (2009 to 2013) (n=138).

Geographical distribution

Over two-thirds (67%, 59) of notified cases were Queensland residents (Figure 96), with a state-specific notification rate of 1.2 per 100,000.

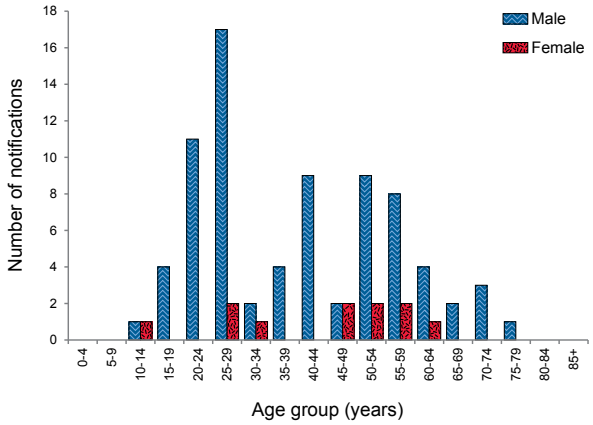
Figure 96: Notifications of leptospirosis, Australia, 2009 to 2014, by month and year of diagnosis and state or territory



Age and sex distribution

The highest counts were observed in males in the 25–29 years age group (n=17) (Figure 97). In 2014, the median age of notified leptospirosis cases was 40 years (range 13 to 78 years) and 82% (72) were male.

Figure 97: Notifications of leptospirosis, Australia, 2014, by age group and sex



Microbiological trends

The WHO/Food and Agriculture Organization/ World Organisation for Animal Health Collaborating Centre for Reference and Research on Leptospirosis (Leptospirosis Reference Laboratory, Queensland) routinely conducts PCR-based serotyping for leptospirosis cases from Queensland (from whence the majority of cases are reported), and collates national data that may be submitted to the laboratory from other states or territories. At the time of compiling this report, data for 2014 were not publicly available.

In Australia, serotyping is only conducted on pathogenic *Leptospira* species of which typing information was available for 72% (56/78) of these cases. The most frequently reported serovars were *L. interrogans* serovar Zanoni (23%, n=18), *L. borgpetersenii* serovar Arborea (15%, n=12) and *L. interrogans* serovar Australis (15%, n=12). In 2013, *L. interrogans* serovar Arborea was the most frequently reported serovar (13/78).

Ornithosis

- In 2014, 41 cases of ornithosis were notified to the NNDSS.

Ornithosis (or psittacosis) is a pneumonia-like illness caused by infection with the bacterium *Chlamydophila psittaci*.[22] It is transmitted to humans primarily from infected psittacines, but transmission to humans has also been known to occur from poultry and a range of other birds.[155]

Transmission to humans occurs via the inhalation of contaminated dried faeces, nasal or eye secretions and dust from the feathers. Individuals at risk of contracting ornithosis include bird owners and those with occupational exposure to birds.[156]

Epidemiological situation in 2014

In 2014 there were 41 notified cases of ornithosis in Australia (0.2 per 100,000), which was a 34% decrease compared with the 5-year mean (2009 to 2013) (n=62).

Geographical distribution

Similar to previous years, more than half of the 2014 notifications were Victorian residents (51%, 21) (Figure 98).

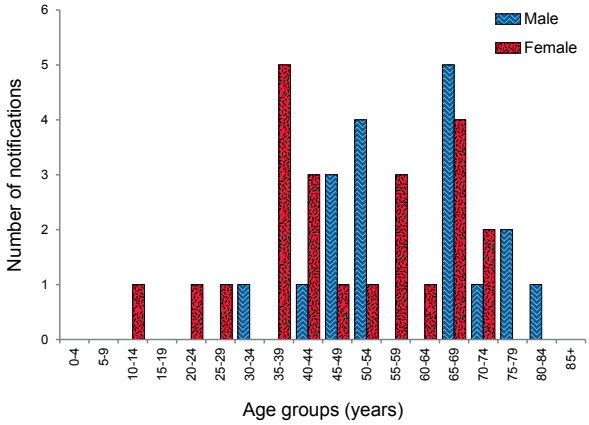
Age and sex distribution

In 2014, the median age of ornithosis notifications was 53 years (range 11 to 80 years) and 56% (23) were female (Figure 99).

Q fever

- In 2014, 469 cases of Q fever were notified to the NNDSS.
- 75% of notified cases were male.

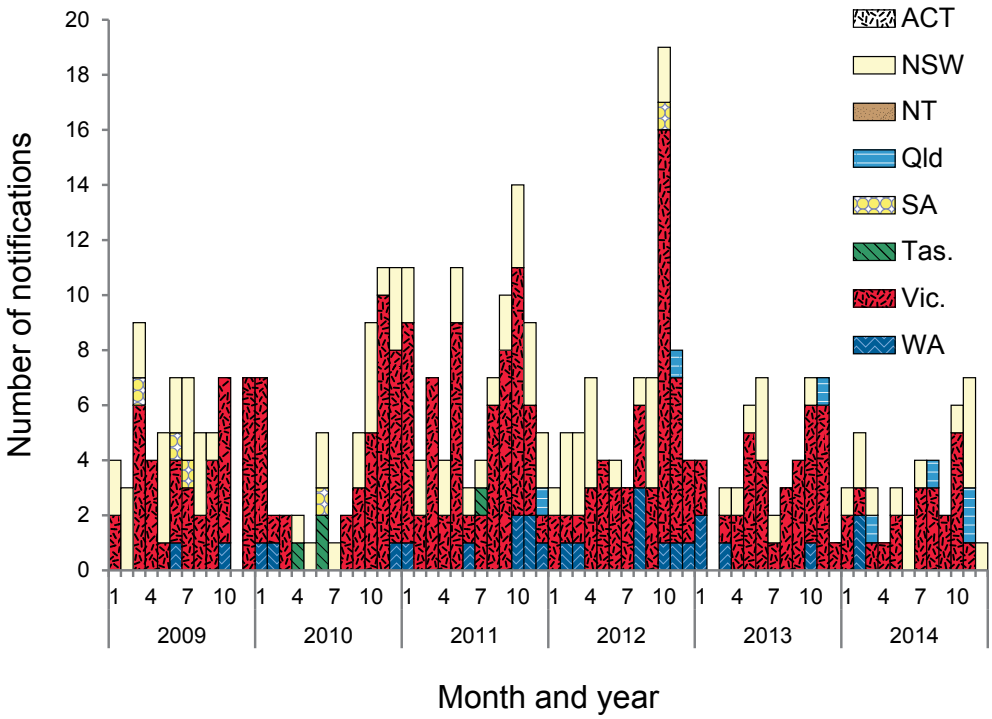
Figure 99: Notifications of ornithosis, Australia, 2014, by age group and sex



Q fever is caused by infection with the bacterium *Coxiella burnetii*. The primary reservoirs of these bacteria are cattle, sheep and goats. *C. burnetii* is resistant to environmental conditions and many common disinfectants.[157] Q fever is most commonly transmitted via the airborne route, where the organism is carried in dust contaminated with tissue, birth fluids or excreta from infected animals.[158] Prior to the commencement of vaccination programs in Australia, approximately half of all cases in New South Wales, Queensland and Victoria were among abattoir workers.[159,160]

The Australian Government funded the National Q Fever Management Program (NQFMP)

Figure 98: Notifications of ornithosis, Australia, 2009 to 2014, by month and year of diagnosis and state or territory



between 2001 and 2006 for states and territories to provide free vaccine to at-risk occupational groups (such as abattoir workers).[161]

Adults at risk of Q fever infection, including abattoir workers, farmers, veterinarians, stockyard workers, shearers and animal transporters should be considered for vaccination. The administration of the Q fever vaccine requires a pre-vaccination screening test to exclude those recipients with a previous (possibly unrecognised) exposure to the organism, including previous vaccination. A Q fever vaccine may cause an adverse reaction in a person who has already been exposed to the bacterium. Vaccination is not recommended for children under 15 years of age or pregnant females.[32]

Epidemiological situation in 2014

In 2014, there were 469 notified cases of Q fever in Australia (2.0 per 100,000), which was a 26% increase compared with the 5-year mean (2009 to 2013) (n=373).

Geographical distribution

Between 1991 and 2001, and prior to the introduction of the NQFMP, Q fever notification rates ranged between 2.5 and 4.9 cases per 100,000.[161] In 2014, the highest notification rate was in Queensland (5.1 per 100,000, n=240). Cases were reported in all jurisdictions except Tasmania (Figure 100).

'Hot spots' for Q fever occur in central Queensland and in the areas that border Queensland and New South Wales, with rates in those areas reaching as high as 142.2 per 100,000 (Figure 101).

Age and sex distribution

The median age of Q fever cases was 46 years (range 5 to 87 years) and 75% (352) were male. Almost a third (30%, 143) of notified cases were males aged between 40 to 59 years (Figure 102). This was consistent with a report that found higher rates of Q fever in men aged 50 to 59 years, and that agriculture-related occupations (including farming) are the most commonly reported occupation.[158]

Tularaemia

- There were no cases of tularaemia notified in 2014.

Tularaemia is a non-specific disease with diverse manifestations, often with an influenza-like onset, caused by infection with the bacterium *Francisella tularensis*.[22] The most common modes of transmission are through arthropod bites, handling infected animals, inhalation of infectious aerosols or exposure to contaminated food or water. Small mammals such as rodents, rabbits and hares are often the reservoir.[162]

Figure 100: Notifications of Q fever, Australia, 2009 to 2014, by month and year of diagnosis and state or territory

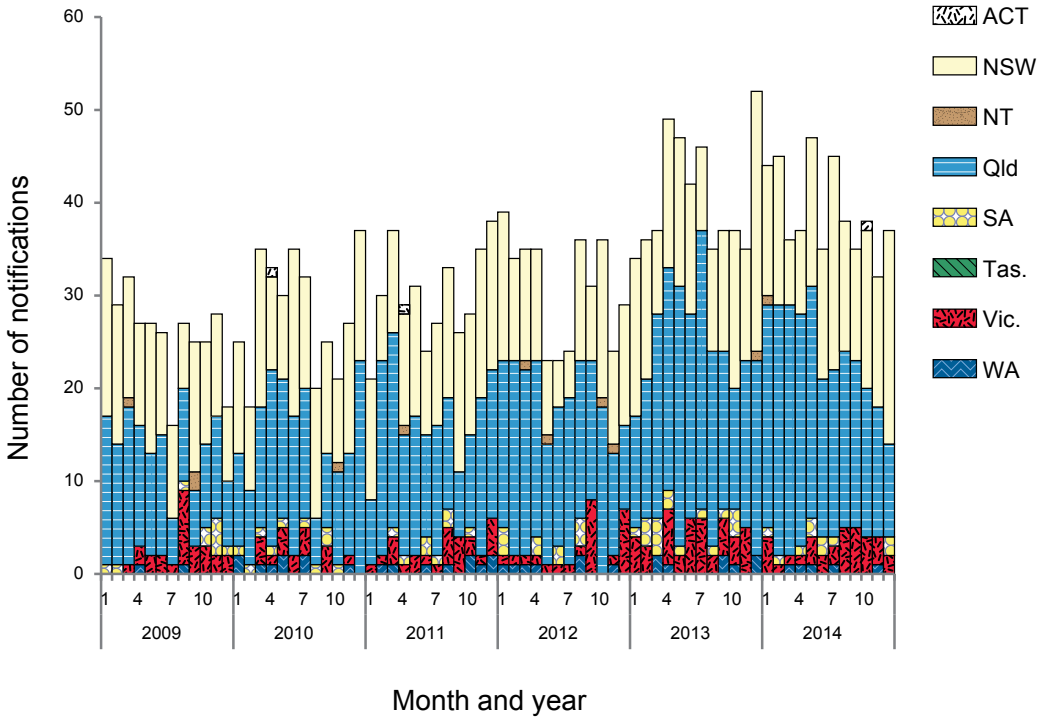


Figure 101: Notification rate for Q fever, Australia, 2014, by Statistical Area Level 3

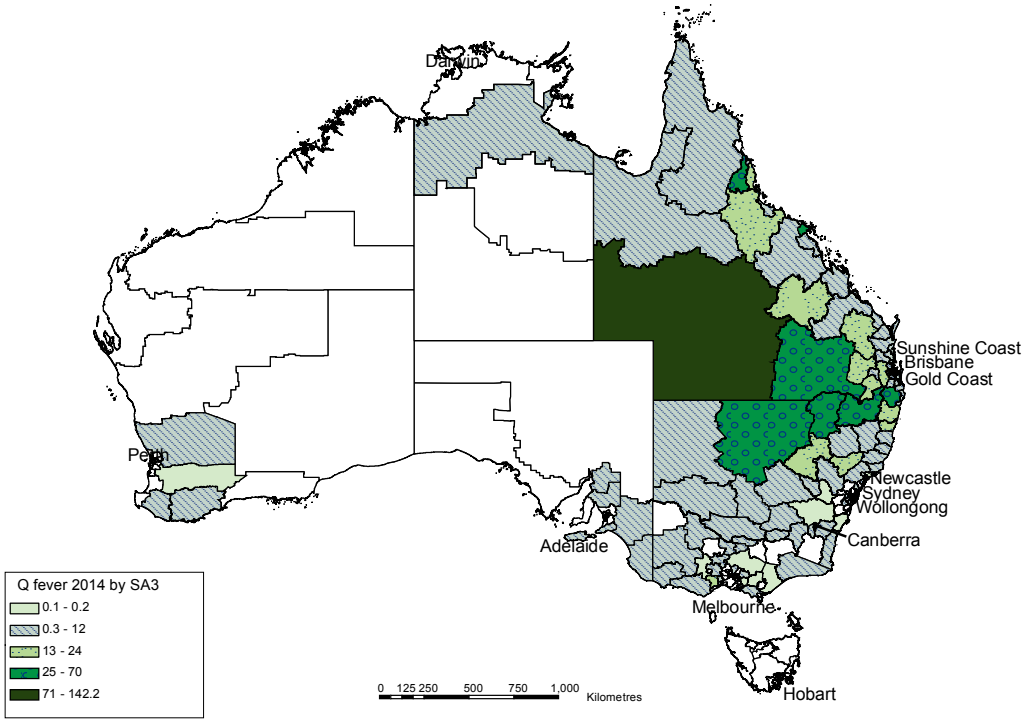
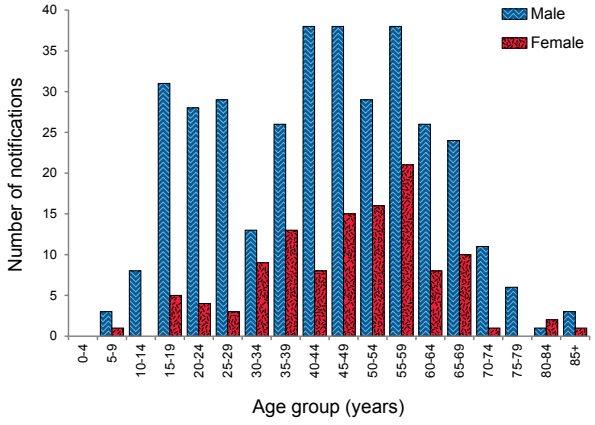


Figure 102: Notifications of Q fever, Australia, 2014, by age group and sex



Epidemiological situation in 2014

In 2014, there were no notified cases of tularaemia in Australia. Tularaemia was last notified in 2011, with 2 cases in Tasmanian residents. This was the first time that *F. tularensis* type B had been detected in the Southern Hemisphere.[163–165]

Other bacterial infections

Other bacterial diseases in the national notifiable disease list are legionellosis, leprosy, invasive meningococcal disease (IMD) and tuberculosis (TB). In 2014, there were 1,942 cases of other bacterial infections notified to the NNDSS, representing less than 1% of all reported cases and similar to the number notified in 2013 (n=1,932). Common objectives for the surveillance of diseases in this section are to monitor their epidemiology and to identify risk groups to accurately target control strategies.

Legionellosis

- In 2014, 424 cases of legionellosis were notified to the NNDSS.
- Compared with 2013, notifications of legionellosis declined by 17% in 2014.
- *Legionella pneumophila,* commonly associated with man-made water systems, was the most frequently reported causative species in 2014.

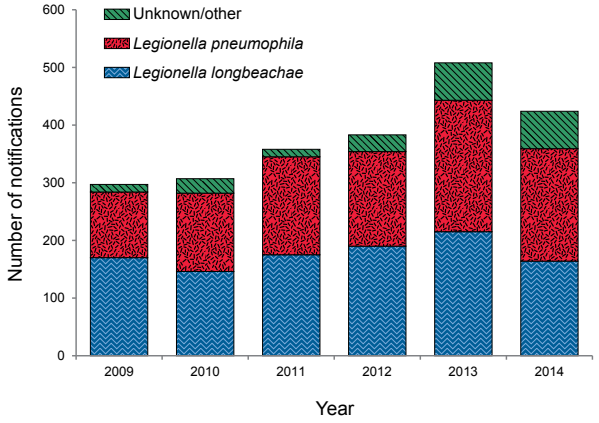
Legionellosis is an environmentally acquired pneumonia caused by the bacteria *Legionella*. It can take the form of either Legionnaires' disease, a severe form of infection of the lungs, or Pontiac fever, a milder influenza-like illness.[22] The species most commonly associated with human disease in Australia are *Legionella pneumophila* and *L. longbeachae*. *Legionella* bacteria are found naturally in low levels in the environment. In the absence of effective environmental treatments *Legionella* organisms can proliferate in air conditioning cooling towers, hot water systems, showerheads, spa pools, fountains, commercial potting

mix and other decomposing material such as bark and sawdust.[166–169] *Legionella* is generally transmitted to humans through contaminated water or dust aerosols.

Epidemiological situation in 2014

In 2014, there were 424 notifications of legionellosis, representing a rate of 1.8 per 100,000. Notifications declined by 17% following an outbreak-related peak in 2013 (n=508) (Figure 103).

Figure 103: Notifications of legionellosis, Australia, 2009 to 2014, by species and year



In 2014, data on the causative species were available for 85% (n=362) of notifications reported. Of those with a known causative, the most frequently reported causative species were *L. pneumophila* (54%, 195), followed by *L. longbeachae* (45%, 164). A single notification of *L. sainthelensi* and 2 notifications of *L. micdadei* were also reported (Table 24). Serogroup information was reported for 62% (120/195) of *L. pneumophila* notifications and 11% (18/164) of *L. longbeachae* notifications. Of these, 98% (117/120) of *L. pneumophila* notifications were typed to *L. pneumophila* serogroup 1, 1 notification was serogroup 2 and 2 were mixed. All *L. longbeachae* notifications were typed to *L. longbeachae* serogroup 1.

Over the period of 2009 to 2014, the number of notified cases of *L. pneumophila* ranged from 114 to 228 per year, whilst notified cases of *L. longbeachae* ranged from 144 to 215 per year (Figure 103). When compared with 2013, notifications of *L. pneumophila* declined by 14% and *L. longbeachae* by 24%.

In 2014, mortality data were available for 78% (n=329) of notifications. Of these, 4% (13/329) were reported to have died due to legionellosis and this was similar to the number of deaths reported in previous years. The majority of deaths were attributed to infection with *L. pneumophila* (46%, 6/13) (Table 24). Over the last 6 years (2009 to 2014) the mortality data of legionellosis notification has improved with the proportion of cases reported with death information increasing from 49% in 2009 to 78% in 2014.

Geographic distribution

In 2014, jurisdictional-specific rates of legionellosis varied from 0.5 per 100,000 in the Australian Capital Territory to 4.5 per 100,000 in Western Australia (Table 5).

In 2014, *L. pneumophila* was the most notified causative species in New South Wales, Queensland, South Australia and Victoria, while *L. longbeachae* was more frequently notified in the Northern Territory, Western Australian and Tasmania. The Australian Capital Territory reported and an equal number of notifications of both species. The most frequent species annually reported by each jurisdiction can vary between

Table 24: Notifications, notification rates and deaths for legionellosis, Australia, 2014, by species and state or territory

Species	State or territory									
	ACT	NSW	NT	Qld	SA	Tas.	Vic.	WA	Aust.	Deaths
L. longbeachae	1	17†	4	16	19	5	9	93*	164	5
L. pneumophila	1	41	3‡	45‡	20†	2	60	23†	195	6
L. micdadei	0	1‡	0	0	0	0	1	0	2	1
L. sainthelensi	0	0	0	0	1	0	0	0	1	0
Unknown species	0	11	0	33	0	1	17‡	0	62	1
Total	2	70	7	94	40	8	87	116	424	13

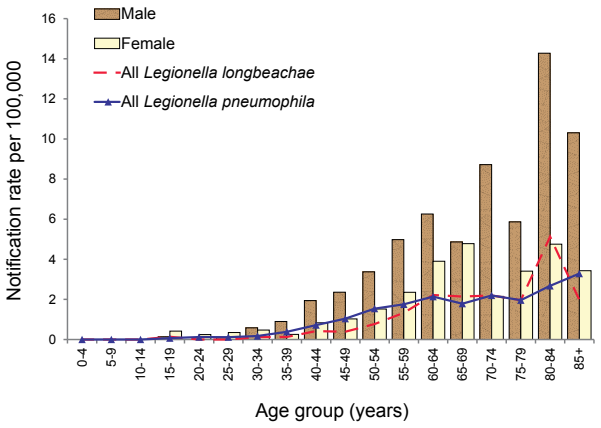
* 3 deaths.
† 2 deaths.
‡ 1 death.

L. pneumophila and *L. longbeachae*. However, generally Western Australia and Northern Territory tend to report more *L. longbeachae* notifications, while New South Wales, Queensland, South Australia and Victoria tend to report more *L. pneumophila* notifications. The Australian Capital Territory and Tasmania tend to report only a small number of notifications each year; therefore, they have no obvious trend in the most frequent causative species.

Age and sex distribution

In 2014, males accounted for the majority (65%) of the notifications resulting in a male to female ratio of 1.8:1. There were no notifications in people under the age of 15 years. In males, the highest notification rates were observed in those aged 80–84 years (14.3 per 100,000) and 85 years and over (10.3 per 100,000). While in females, the highest notification rates were observed in those aged 65–69 years and 80–84 years age groups (both 4.8 per 100,000) (Figure 104).

Figure 104: Notification rate for legionellosis, Australia, 2014, by age group, sex and species

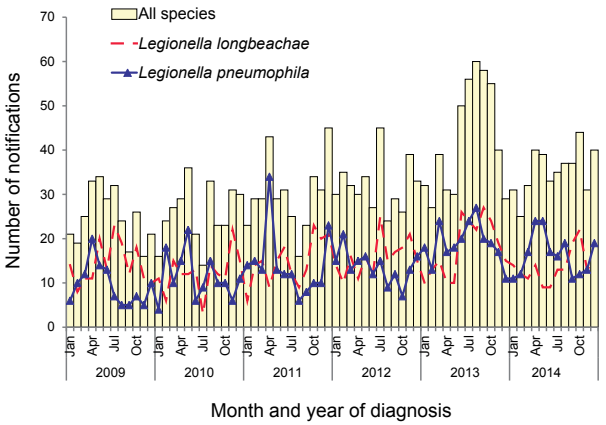


The ages of the 13 cases reported to have died due to legionellosis in 2014 ranged between 57 to 83 years (median 75 years); 12 deaths were male and 1 was female. In 2014, the demographic profile of legionellosis remained consistent with the recognised epidemiology of the disease.[22,170,171]

Seasonality

In 2014, diagnoses of legionellosis were highest in October, with 44 notified cases. The diagnosis of *L. pneumophila* peaked in April–May (24 cases each month), and the diagnosis of *L. longbeachae* peaked in October (n=22). From 2009 to 2013, the diagnosis of *L. pneumophila* commonly occurred in the autumn and summer months, except for 2013 when diagnoses peaked at the end of winter. In the same period, the diagnosis of *L. longbeachae* more commonly occurred in winter and spring. (Figure 105).

Figure 105: Notifications of legionellosis, Australia, 2009 to 2014, by month and year of diagnosis and species



Place of acquisition

In 2014, a place of acquisition was reported for 86% (n=363) of legionellosis notifications. Of these, 92% (334) were reported as acquired within Australia and 8% (29) were reported as acquired overseas. Of the overseas acquired notifications, Indonesia (17%, 5/29), the United States of America (10%, 3/29) and Thailand (10%, 3/29) were the most commonly reported places of acquisition.

Outbreaks

In 2014, there were 5 outbreaks of *L. pneumophila* reported to the NNDSS.

In South Australia, there was an outbreak of 6 cases of legionellosis caused by *L. pneumophila* serogroup 1. All cases lived or worked within the Adelaide central business district (CBD). During the environmental investigation several cooling towers in the Adelaide CBD were identified as reservoirs of *L. pneumophila* serogroup 1 and were subsequently decontaminated.

In Victoria, there were 4 outbreaks, involving a total of 17 cases. None of these outbreaks were linked to overseas sources and none of the cases linked to outbreaks died due to their infection.

One of the outbreaks involved a total of 10 cases and included 4 separate clusters. There were 3 environmental detections from cooling towers

associated with the outbreak; however, none of the environmental samples matched the *Legionella* strain isolated from the clinical samples.

Another outbreak involved 3 linked cases, all of whom had either visited or worked at the Melbourne Airport. Despite extensive sampling and testing, no environmental detections were found for this outbreak.

The remaining 2 outbreaks involved 2 cases in each outbreak. One of these outbreaks was associated with a common geographical link, but no environmental detections were found. The other outbreak was linked to a workplace at the port of Melbourne. An environmental detection was found in a cooling tower in the port of Melbourne area but this environmental sample did not contain the same *Legionella* strain as isolated from the clinical samples.

Leprosy

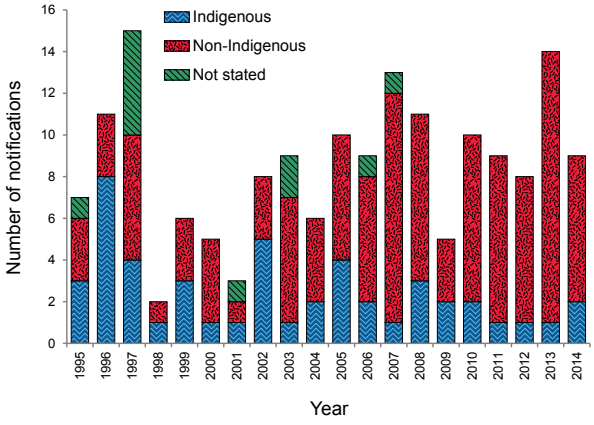
- A total of 9 cases of leprosy were notified in 2014, maintaining a notification rate of less than 0.1 per 100,000.
- Most cases of leprosy notified in 2014 were acquired overseas.

Leprosy is a chronic infection of the skin and peripheral nerves with the bacterium *Mycobacterium leprae*. Leprosy is an uncommon disease in Australia with the majority of cases occurring in migrants from leprosy-endemic countries and Indigenous populations. The incidence of leprosy worldwide is declining due to various factors including economic development, bacille Calmette Guérin (BCG) immunisation and high coverage with multi-drug therapy.[22] Leprosy is not a highly infectious disease and is typically slow to progress to a symptomatic stage. The incubation period for leprosy is about 5 years; however, it can take as long as 20 years for symptoms to appear.[172] People at-risk are generally in close and frequent contact with leprosy patients or living in countries where the disease is more common. Leprosy is curable and once a person with leprosy begins appropriate treatment, they quickly become non-infectious.

Epidemiological situation in 2014

In 2014, a total of 9 cases of leprosy were notified (4 female, 5 male), representing a rate of less than 0.1 per 100,000. There were 5 cases notified in Western Australia, and 1 each in Victoria, South Australia, Queensland and New South Wales. Cases ranged in age from 17 to 75 years, with a median age of 45 years. Two cases were reported as being Indigenous. The remaining 7 cases were reported as being non-Indigenous and as having acquired the infection overseas. Cases were reported as being from India (n=2), Sri Lanka (n=1), the Philippines (n=1), Samoa (n=1) and the overseas country of acquisition was unknown for 2 cases. Since 1995, annual notifications of leprosy have ranged from 2 to 15 cases per year (Figure 106).

Figure 106: Notifications of leprosy, Australia, 1995 to 2014, by year and Indigenous status



Meningococcal disease (invasive)

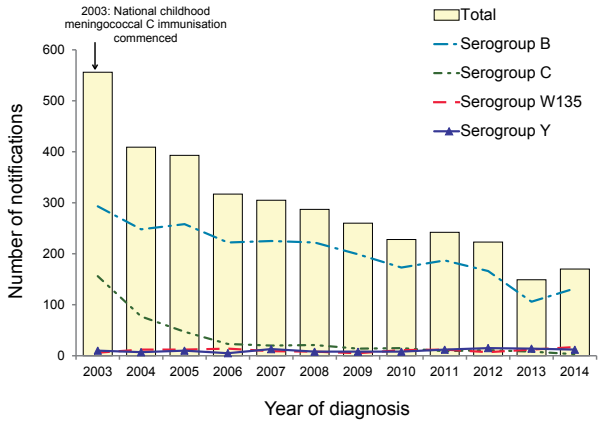
- There were 170 cases and 8 deaths related to IMD reported in 2014.
- The majority of IMD cases were caused by serogroup B organisms.
- Infections with serogroup Y account for a small but increasing proportion of IMD notifications.
- Seventy-two per cent of IMD cases reported in 2014 were less than 25 years of age.

IMD is caused when the bacterium *Neisseria meningitidis* enters a normally sterile site, usually the blood (septicaemia), cerebrospinal fluid (meningitis) or both. Asymptomatic respiratory tract carriage of meningococci is present in 5% to 10% of the population and prevalence may be higher when groups of people occupy small areas of any space.[22,32] The disease is transmitted via respiratory droplets and has an incubation period of between 2 and 10 days, commonly 3 to 4 days. [22,173] It occasionally causes a rapidly progressive serious illness, most commonly in previously healthy children and young adults. Globally, serogroups A, B, C, X, W135 and Y commonly cause invasive disease.[174] Historically, *N. meningitidis* serogroups B and C have been the major cause of IMD in Australia.

Epidemiological situation in 2014

In 2014, there were 170 cases of IMD reported to the NNDSS, representing a rate of 0.7 per 100,000 population. This was an increase of 14% compared with 2013 (n=149), but less than the number of cases notified between 2003 and 2012 (range 556 to 223 cases) (Figure 107). This rise in IMD cases was due to infections caused by serogroup B.

Figure 107: Notifications of invasive meningococcal disease, Australia, 2003 to 2014, by selected serogroup and year



The majority of cases notified in 2014 (99%, n=168) met the case definition as a confirmed case, being diagnosed based on laboratory definitive evidence, or laboratory suggestive evidence and clinical evidence.[173] A small number of cases (n=2) were reported as probable and diagnosed based on clinical evidence only.

In 2014, all states and territories reported cases of IMD (Table 1), with notification rates ranging from 0.4 per 100,000 in Tasmania to 2 per 100,000 in South Australia (Table 25). Mortality data were available for 74% (126/170) of cases. Of these, 8 cases were reported as having died from IMD, including 7 from infection with serogroup B organisms, and 1 from infection with serogroup Y organisms (Table 25). Six of the deaths associated with IMD infection caused by serogroup B organisms occurred in children less than 5 years of age and 1 in a teenager 17 years of age. The 1 death caused by a serogroup Y organism occurred in an adult over 85 years of age.

Age and sex distribution

More males (53%, n=90) than females (47%, n=80) were notified with IMD in 2014. Proportionally, 72% (n=122) of all cases reported were less than 25 years of age, of which half were children less than 5 years of age (n=61). The highest notification rate in 2014 for both males and females was in the 0–4 years age group (3.4 per 100,000) with a second peak in adolescents (15–19 years of age) (Figure 108).

Figure 108: Notification rate for invasive meningococcal disease, Australia, 2014, by age group and sex (n=170)

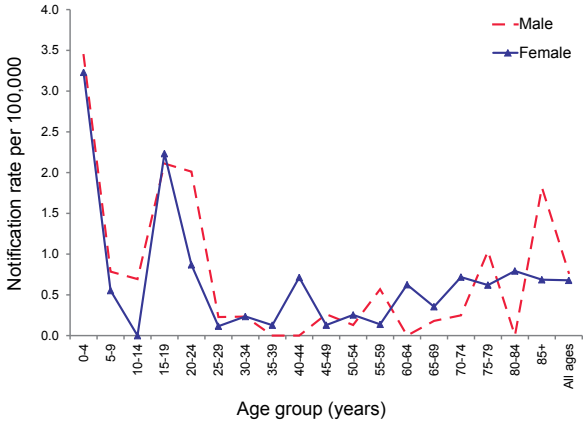


Table 25: Notifications of invasive meningococcal disease and deaths due to invasive meningococcal disease, Australia, 2014, by serogroup and state or territory

Serogroup	State or territory									
	ACT†	NSW†	NT	Qld	SA	Tas.	Vic.	WA	Aust.	Deaths
B	1	23	3	30	34	1	27	13	132	7
C	0	0	0	1	0	0	0	2	3	0
W135	0	7	0	3	0	1	4	2	17	0
Y	1	7	0	2	0	0	1	1	12	1
Unknown*	0	1	0	4	0	0	1	0	6	0
Total	2	38	3	40	34	2	33	18	170	8
Rate per 100,000	0.5	0.5	1.2	0.8	2.0	0.4	0.6	0.7	0.7	0

* Unknown includes notifications where serogroup was non-groupable or not grouped. Not grouped is when no serogroup is available and non-groupable is where the serogroup is reported by the reference laboratory as a non-groupable strain.

† Conjunctival IMD cases are also reported under the local case definition, and reported to the national dataset by the jurisdiction. Conjunctival cases cannot be distinguished from invasive cases in the national dataset.

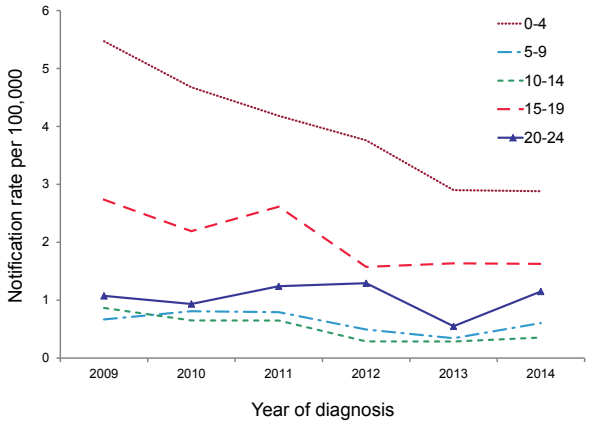
Serogroup analysis

Data on serogroup were available for 96% (n=164) of cases in 2014, of which 80% (132) were caused by serogroup B organisms, 10% (17) by serogroup W135, 7% (12/164) by serogroup Y and 2% (3) by serogroup C (Table 25). Cases caused by serogroup Y (n=12) were slightly lower compared with 2013 (n=14), but were higher than the average of 10 cases seen in the previous 10 years (2004–2013). Notifications of IMD caused by serogroup C organisms continue to decrease with 3 cases notified in 2014 compared with 8 in 2013 (Table 25) and representing a 98% decrease since the introduction of the meningococcal C vaccine on the NIP in 2003.

All 3 cases of IMD due to serogroup C organisms notified in 2014 were under the age of 25 years. Two cases were in the 0–4 years age group, and 1 was in the 20–24 years age group. Age-specific rates of serogroup C infections have remained below 0.2 cases per 100,000 since 2010.

Serogroup B accounted for the majority of cases across all age groups including those aged less than 25 years. Compared with 2013, serogroup B rates were relatively stable in all age groups except the 5–9 years and 20–24 years age groups, which displayed a 2-fold and 2.4-fold increase respectively (Figure 109).

Figure 109: Notification rate for serogroup B invasive meningococcal disease, Australia, 2009 to 2014, by year and selected age groups



An increase in notifications of serogroup W135 was evident in 2014 (n=17) with notifications nearly 2 times higher than the annual average of the previous 5 years. There were 17 cases of W135 reported in 2014 compared with 12 cases in 2013 and 7 cases in 2012.

Vaccination status

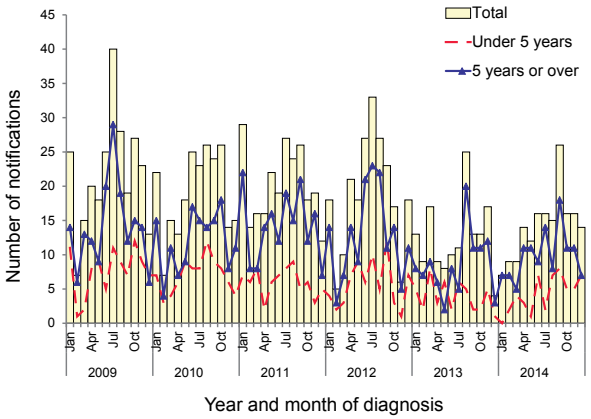
From 2003, the meningococcal C vaccine has been available for infants aged 12 months as a part of the childhood immunisation schedule funded under the NIP. A catch-up program provided access to the meningococcal C vaccine for children and adolescents born between 1984 and 2001.

Of the 3 cases of IMD caused by serogroup C organisms reported in 2014, 2 were less than 12 months of age and therefore not eligible for vaccination, and 1 was eligible for vaccination (21 years) but was reported with an unknown vaccination status.

Seasonality

In 2014, an average of 14 cases of IMD were reported monthly, with a range of 7 to 26 cases. A clear seasonal pattern was apparent in 2014, with the highest number of notifications reported in spring. The 2014 season peaked in September with 26 cases reported and was later than the seasonal pattern displayed in the previous 5 years (2009 to 2013), in which notifications peaked in mid to late winter (Figure 110). Consistent with the previous 5 years, the 2014 seasonal trend was more obvious in cases 5 years of age or over compared with those less than 5 years of age.

Figure 110: Notifications of invasive meningococcal disease, Australia, 2009 to 2014, by month and year and age group



Susceptibility

The Australian Meningococcal Surveillance Program (AMSP) was established in 1994 for the purpose of monitoring and analysing isolates of *N. meningitidis* from cases of IMD in Australia. The program is undertaken by a network of reference laboratories in each state and territory, using

standardised methodology to determine the phenotype (serogroup, serotype and serosubtype) and the susceptibility of *N. meningitidis* to a core group of antibiotics.

Annual reports of the AMSP are published in CDI, with the most recent report published for 2014.[175] The latest data from AMSP show that 12% of isolates tested were fully sensitive and 88% demonstrated decreased susceptibility to the penicillin group of antibiotics. No isolates tested in 2014 exhibited resistance to penicillin. All tested IMD isolates were susceptible to ceftriaxone and ciprofloxacin, and 2 isolates were resistant to rifampicin.

Discussion

In Australia, IMD has remained at its lowest levels since the national notification commenced in 1991. The reduction has been seen most considerably in disease caused by serogroup C, but declines in disease caused by serogroup B are also evident.

Tuberculosis

- A total of 1,339 cases of TB were notified in 2014.
- In 2014, the notification rate of TB increased slightly from 5.5 per 100,000 in 2013 to 5.9 per 100,000.

TB is an infection caused by the bacterium *Mycobacterium tuberculosis*. TB is transmitted by airborne droplets produced by people with pulmonary or respiratory tract TB when coughing or sneezing. While Australia has one of the lowest rates of TB in the world, the disease remains a public health issue, particularly in Australia's overseas-born and Indigenous communities. [176]

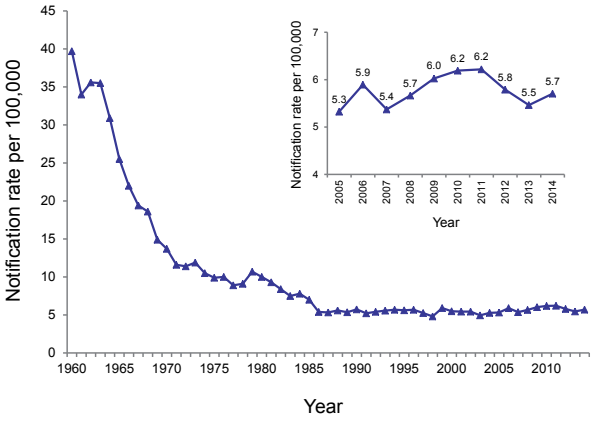
Epidemiological situation in 2014

In 2014, a total of 1,339 cases of TB were notified to the NNDSS representing a rate of 5.7 per 100,000. This was an increase on the rate of 5.5 per 100,000 (n=1,263) reported in 2013, but less than the preceding 5-year mean (2009 to 2013) of 5.9 per 100,000. Australia has achieved good TB control and has maintained low rates of TB since the mid 1980s (Figure 111).

Geographic distribution

New South Wales (n=472), Victoria (n=448), Queensland (n=165) and Western Australia (n=139) accounted for 91% of all cases of TB diagnosed in Australia. The Northern Territory (11.4 per 100,000), the Australian Capital Territory (7.8 per 100,000), Victoria (7.7 per 100,000) and New South Wales (6.3 per 100,000) all reported a rate higher than the national notification rate. In 2014, the Northern Territory, South Australia and Western Australia reported lower notification rates than the previous year. All the other states and territories reported an increase on the previous year. Notifications and rates of TB by state or territory are presented in Table 6.

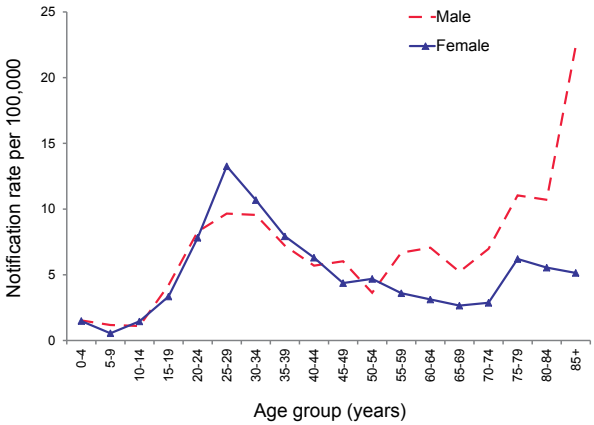
Figure 111: Notification rate for tuberculosis, Australia, 1960 to 2014, by year



Age and sex distribution

Overall, the age groups with the highest notification rates were in the 25–29 years and 85 years or over age groups (both 11.4 per 100,000), followed by the 30–34 years age group (10.1 per 100,000). The highest age and sex specific rates were observed in men in the 85 years or over (22.4 per 100,000) and women in the 25–29 years age groups (13.3 per 100,000) (Figure 112). Males accounted for 53% of the TB notifications in 2014.

Figure 112: Notification rate for tuberculosis, Australia, 2014, by age group and sex



Vaccination

The BCG vaccine was first introduced for protection against tuberculosis in the 1920s and despite variable evidence on the efficacy of the vaccine, it remains the only vaccine in use for TB today.[177,178]

According to national guidelines developed by Australia's National Tuberculosis Advisory Committee, BCG vaccination is recommended for Aboriginal and Torres Strait Islander neonates in communities with a high incidence of TB; neonates and children under 5 years of age who will be travelling to or living in countries or areas with a high prevalence of TB for extended periods; and neonates born to parents with leprosy or a family history of leprosy.

BCG vaccination is not recommended for general use in the Australian population or for most health care workers. It is contraindicated in HIV infected persons.[179] Note that BCG immunisation practices may vary between states and territories due to differences in jurisdiction specific TB vaccination policies and population demographics.

Enhanced surveillance data sets

Enhanced data are collected on all cases of TB. Further analyses, including identification of risk groups and reporting on treatment outcomes, can be found in the TB annual report series also published in CDI.

Appendices

Appendix 1: Estimate of Australian population, 2014, by state or territory

	State or territory								
	ACT	NSW	NT	Qld	SA	Tas.	Vic.	WA	Aus.
Males	191,566	3,729,091	129,421	2,351,996	835,151	256,418	2,888,381	1,296,737	11,680,860
Females	193,923	3,786,643	115,191	2,369,352	850,245	258,268	2,951,115	1,268,640	11,794,489
Total	385,489	7,515,734	244,612	4,721,348	1,685,396	514,686	5,839,496	2,565,377	23,475,349

Source: Australian Bureau of Statistics. Table 4, Estimated Resident Population, State and Territories. Australian Demographic Statistics. ABS Cat no. 3101.0 December 2014.

Appendix 2: Estimate of Australian population, 2014, by state or territory and age

Age group	State or territory								
	ACT	NSW	NT	Qld	SA	Tas.	Vic.	WA	Aus.
00–04	26,825	485,831	19,179	316,933	100,794	30,989	375,099	171,492	1,527,302
05–09	23,775	474,199	17,945	316,945	99,415	32,173	357,939	165,313	1,487,862
10–14	21,457	448,958	16,958	301,045	97,089	31,458	336,342	153,899	1,407,390
15–19	22,954	467,371	16,413	309,790	104,763	33,795	357,934	161,287	1,474,485
20–24	32,204	513,569	19,302	335,799	114,429	31,345	419,758	184,276	1,650,869
25–29	33,893	539,032	23,785	340,992	115,376	29,452	450,175	214,047	1,747,148
30–34	32,434	541,359	22,198	331,559	111,195	29,222	439,588	201,368	1,709,344
35–39	28,162	498,014	18,647	309,359	102,734	28,963	395,440	177,602	1,559,166
40–44	28,217	526,430	18,554	340,344	114,318	34,190	417,742	186,103	1,666,157
45–49	25,063	482,577	16,170	310,804	112,827	33,873	386,969	172,605	1,541,092
50–54	24,574	502,417	15,456	313,943	116,159	37,637	380,927	168,373	1,559,707
55–59	21,718	462,858	13,497	281,596	108,960	36,354	348,782	151,016	1,424,956
60–64	18,847	410,371	10,504	251,970	99,624	33,753	307,662	130,589	1,263,524
65–69	15,987	368,229	7,340	224,623	90,102	30,363	273,184	109,827	1,119,751
70–74	10,668	273,615	4,316	162,073	65,641	22,361	202,085	78,516	819,339
75–79	7,755	207,728	2,231	114,086	51,096	16,084	155,090	58,137	612,237
80–84	5,358	153,937	1,243	80,148	38,736	11,702	116,447	40,879	448,467
85+	5,598	159,239	874	79,339	42,138	10,972	118,333	40,048	456,553
Total	385,489	7,515,734	244,612	4,721,348	1,685,396	514,686	5,839,496	2,565,377	23,475,349

Source: Australian Bureau of Statistics. Estimated Resident Population, State and Territories. Australian Demographic Statistics. ABS Cat no. 3101.0 December 2014.

Appendix 3: Indigenous status, National Notifiable Diseases Surveillance System, Australia, 2014, by notifiable disease*

Disease name	Aboriginal but not TSI origin	TSI but not Aboriginal origin	Aboriginal and TSI origin	Not Indigenous	Not stated	Blank/ missing	Total	% complete	Number complete	Number incomplete
Arbovirus (NEC)	0	0	0	16	12	0	28	57	16	12
Barmah Forest virus infection	15	2	0	173	417	134	741	26	190	551
Botulism	0	0	0	1	0	0	1	100	1	0
Brucellosis	0	0	0	16	1	0	17	94	16	1
Campylobacteriosis	291	9	16	9,579	9,507	529	19,931	50	9,895	10,036
Chlamydial infection	5,508	757	354	25,371	31,682	22,436	86,108	37	31,990	54,118
Cholera	0	0	0	2	0	0	2	100	2	0
Cryptosporidiosis	148	3	4	1,153	971	126	2,405	54	1,308	1,097
Dengue virus infection	24	8	6	1,289	357	32	1,716	77	1,327	389
Diphtheria	0	0	0	2	0	0	2	100	2	0
Donovanosis	1	0	0	0	0	0	1	100	1	0
Gonococcal infection	3,233	194	95	6,875	3,694	1,584	15,675	66	10,397	5,278
Haemolytic uraemic syndrome	2	0	0	15	2	1	20	85	17	3
Haemophilus influenzae type b	5	0	0	16	0	0	21	100	21	0
Hepatitis A	3	1	0	218	9	0	231	96	222	9
Hepatitis B (newly acquired)	12	1	0	148	15	0	176	92	161	15
Hepatitis B (unspecified)	132	15	5	2,114	1,877	2,351	6,494	35	2,266	4,228
Hepatitis C (newly acquired)	117	1	0	305	10	0	433	98	423	10
Hepatitis C (unspecified)	745	8	18	3,085	3,386	3,007	10,249	38	3,856	6,393
Hepatitis D	0	0	0	39	9	11	59	66	39	20
Hepatitis E	0	0	0	53	3	0	56	95	53	3
Influenza (laboratory confirmed)	1,818	108	79	25,047	19,212	21,478	67,742	40	27,052	40,690
Japanese encephalitis virus infection	0	0	0	1	0	0	1	100	1	0
Kunjin virus infection	0	0	0	1	0	0	1	100	1	0
Legionellosis	7	0	0	390	23	4	424	94	397	27
Leprosy	2	0	0	7	0	0	9	100	9	0
Leptospirosis	0	0	0	70	18	0	88	80	70	18
Listeriosis	0	1	0	71	7	1	80	90	72	8
Malaria	2	0	0	272	48	0	322	85	274	48
Measles	11	0	0	316	11	2	340	96	327	13

Appendix 3 *continued*: Indigenous status, National Notifiable Diseases Surveillance System, Australia, 2014, by notifiable disease*

Disease name	Aboriginal but not TSI origin	TSI but not Aboriginal origin	Aboriginal and TSI origin	Not Indigenous	Not stated	Blank/ missing	Total	% complete	Number complete	Number incomplete
Meningococcal disease (invasive)	21	0	0	147	2	0	170	99	168	2
Mumps	2	1	0	144	33	10	190	77	147	43
Ornithosis	0	0	0	35	5	1	41	85	35	6
Pertussis	212	8	9	6,335	3,864	1,435	11,863	55	6,564	5,299
Pneumococcal disease (invasive)	179	7	7	1,205	131	35	1,564	89	1,398	166
Q fever	14	0	0	395	55	5	469	87	409	60
Ross River virus infection	129	15	6	2,466	1,990	710	5,316	49	2,616	2,700
Rubella	0	0	0	13	2	2	17	77	13	4
Shiga toxin-producing *Escherichia coli*	1	0	0	99	11	4	115	87	100	15
Salmonellosis	403	20	19	7,572	5,085	3,259	16,358	49	8,014	8,344
Shigellosis	138	3	3	709	178	20	1,051	81	853	198
Syphilis – congenital	5	0	0	0	0	0	5	100	5	0
Syphilis < 2 years	227	5	6	1,601	160	10	2,009	92	1,839	170
Syphilis > 2 years or unspecified duration	212	15	2	1,119	469	104	1,921	70	1,348	573
Tetanus	0	0	0	2	1	0	3	67	2	1
Tuberculosis	25	15	1	1,298	0	0	1,339	100	1,339	0
Typhoid fever	0	0	0	116	3	0	119	98	116	3
Varicella zoster (chickenpox)	99	3	6	1,771	180	29	2,088	90	1,879	209
Varicella zoster (shingles)	137	1	6	4,828	421	80	5,473	91	4,972	501
Varicella zoster (unspecified)	140	28	7	2,500	9,065	357	12,097	22	2,675	9,422
Total	14,020	1,229	649	109,001	92,927	57,757	275,581	45	124,899	150,682

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

TSI Torres Strait Islander.

Abbreviations

13vPCV	13 valent pneumococcal conjugate vaccine
23vPPV	23 valent pneumococcal polysaccharide vaccine
ABLV	Australian bat lyssavirus
AFP	acute flaccid paralysis
AGSP	Australian Gonococcal Surveillance Programme
AIDS	acquired immune deficiency syndrome
AMSP	Australian Meningococcal Surveillance Programme
ANCJDR	Australian National Creutzfeldt-Jakob Disease Registry
BCG	bacille Calmette–Guérin
BFV	Barmah Forest virus
CDI	Communicable Diseases Intelligence
CDNA	Communicable Diseases Network Australia
CDWG	Case Definitions Working Group
CIDT	culture-independent diagnostic testing
CJD	Creutzfeldt-Jakob disease
CRS	congenital rubella syndrome
DENV	dengue virus
Hib	*Haemophilus influenzae* type b
HIV	human immunodeficiency virus
HPAIH	highly pathogenic avian influenza in humans
HUS	haemolytic uraemic syndrome
ILI	influenza like illness
IMD	invasive meningococcal disease
IPD	invasive pneumococcal disease
JEV	Japanese encephalitis virus
KUNV	Kunjin virus
MMR	measles-mumps-rubella
MVEV	Murray Valley encephalitis virus
NAMAC	National Arbovirus and Malaria Advisory Committee
NDP	no data provided
NEC	not elsewhere classified
NIP	National Immunisation Program
NN	not notifiable
NNDSS	National Notifiable Diseases Surveillance System
NQFMP	National Q fever Management Program
NSC	National Surveillance Committee
NS1	non-structural protein 1
PCR	polymerase chain reaction
RRV	Ross River virus
SACC	Standard Australian Classification of Countries
SARS	severe acute respiratory syndrome
STEC	Shiga toxin-producing *Escherichia coli*
STI(s)	sexually transmissible infections(s)
TB	tuberculosis
VPD(s)	vaccine preventable disease(s)
VZV	varicella zoster virus
WHO	World Health Organization
WHOCC	World Health Organization Collaborating Centre for Reference and Research on Influenza
ZIKV	Zika virus

Acknowledgements

The authors wish to thank the following people for their contribution to this report.

Members of the National Surveillance Committee

The National Centre for Immunisation Research and Surveillance of Vaccine Preventable Diseases

Author details

Coordinating author: Timothy Sloan-Gardner

Data management: Mark Trungove, Rachael Corvisy

Bloodborne diseases: Amy Bright, Carolyn Konrad

Gastrointestinal diseases: Timothy Sloan-Gardner

Quarantinable diseases: Katrina Knope

Sexually transmissible infections: Amy Bright, Amy Burroughs, Carolyn Konrad

Vaccine preventable diseases: Nicolee Martin, Anna Glynn-Robinson, Cindy Toms, Natasha Wood

Vectorborne diseases: Katrina Knope

Zoonoses: Timothy Sloan-Gardner

Other bacterial infections: Cindy Toms, Natasha Wood

With contributions from:

National organisations

Communicable Diseases Network Australia and subcommittees

Australian Childhood Immunisation Register

Australian Gonococcal Surveillance Programme

Australian Meningococcal Surveillance Programme

Australian Sentinel Practice Research Network

Australian Quarantine Inspection Service

The Kirby Institute for Infection and Immunity in Society

National Centre for Immunisation Research and Surveillance of Vaccine Preventable Diseases

National Enteric Pathogens Surveillance Scheme

OzFoodNet Working Group

World Health Organization Collaborating Centre for Reference and Research on Influenza

State and territory health departments

Communicable Diseases Control, ACT Health, Australian Capital Territory

Communicable Diseases Surveillance and Control Unit, NSW Ministry of Health, New South Wales

Centre for Disease Control, Northern Territory Department of Health and Community Services, Northern Territory

Communicable Diseases Branch, Queensland Health, Queensland

Communicable Disease Control, South Australian Department of Health, South Australia

Communicable Diseases Prevention Unit, Department of Health and Human Services, Tasmania

Health Protection Branch, Department of Health, Victoria

Communicable Diseases Control Directorate, Department of Health, Western Australia

References

1. *National Health Security Act, 2007*. Accessed March 2015. Available from: http://www.comlaw.gov.au/Details/C2007A00174
2. National Health Security (National Notifiable Disease List) Instrument 2008. Accessed March 2015. Available from: http://www.comlaw.gov.au/ComLaw/legislation/LegislativeInstrument1.nsf/0/7162D634C6DD1BAACA25740B0079D6B8?OpenDocument
3. National Health Security Agreement 2008. Accessed March 2015. Available from: http://www.health.gov.au/internet/main/publishing.nsf/Content/ohp-nhs-agreement.htm
4. The Kirby Institute. *HIV, viral hepatitis and sexually transmissible infections in Australia. Annual Surveillance Report 2015*. UNSW Australia, Sydney NSW: The Kirby Institute; 2015.
5. Klug GM, Boyd A, Sarros S, Stehmann C, Simpson M, McLean CA, et al. Creutzfeldt-Jakob disease surveillance in Australia, update to December 2013. *Commun Dis Intell* 2014;38(4):E348–355.
6. Communicable Diseases Network Australia. National Notifiable Diseases Surveillance System. 2015. Accessed March 2015. Available from: www.health.gov.au/nndssdata
7. Australian Bureau of Statistics. Australian Demographic Statistics, December 2014. Canberra: Australian Bureau of Statistics; 2015. Report No.: ABS Catalogue: 3101.0.
8. Australian Institute of Health and Welfare. Age-standardised rate – Identifying and definitional attributes. 2005. Accessed on 17 March 2015. Available from: http://meteor.aihw.gov.au/content/index.phtml/itemId/327276
9. Graham S, Guy RJ, Donovan B, McManus H, El-Hayek C, Kwan K, et al. Epidemiology of chlamydia and gonorrhea among Indigenous and non-Indigenous Australians, 2000–2009. *Med J Aust* 2012;197(11):642–646.
10. Hammerschlag M. Sexually transmitted diseases in sexually abused children: medical and legal implications. *Sex Transm Infect* 1998;74(3):167–174.
11. Australian Institute of Health and Welfare. National Health Data Dictionary 13.3; 2008.
12. Australian Bureau of Statistics. Standard Australian Classification of Countries (SACC). Canberra: Australian Bureau of Statistics; 2011. Report No.: 1269.0.
13. Chen MY, Fairley CK, Donovan B. Nowhere near the point of diminishing returns: correlations between chlamydia testing and notification rates in New South Wales. *Aust N Z J Public Health* 2005;29(3):249–253.
14. Hocking J, Fairley C, Counahan M, Crofts N. The pattern of notification and testing for genital *Chlamydia trachomatis* infection in Victoria, 1998–2000: an ecological analysis. *Aust N Z J Public Health* 2003;27(4):405–408.
15. Burckhardt F, Warner P, Young H. What is the impact of change in diagnostic test method on surveillance data trends in *Chlamydia trachomatis* infection? *Sex Transm Infect* 2006;82(1):24–30.
16. Chen MY, Karvelas M, Sundararajan V, Hocking JS, Fairley CK. Evidence for the effectiveness of a chlamydia awareness campaign: increased population rates of chlamydia testing and detection. *Int J STD AIDS* 2007;18(4):239–243.
17. Hammad A, Guy RJ, Fairley C, Wand H, Chen MY, Dickson B, et al. Understanding trends in genital *Chlamydia trachomatis* can benefit from enhanced surveillance: findings from Australia. *Sex Transm Infect* 2012;88(7):552–557.
18. Stephens N, O'Sullivan M, Coleman D, Shaw K. *Chlamydia trachomatis* in Tasmania 2001–2007: rising notification trends. *Aust N Z J Public Health* 2010;34(2):120–125.
19. Communicable Diseases Network Australia. Australian national notifiable diseases case definitions. 2014. Accessed on 5 September 2014. Available from: http://www.health.gov.au/casedefinitions
20. National HBV Testing Policy Expert Reference Committee. *National hepatitis B testing policy 2012 v1.1*. Australasian Society for HIV, Viral Hepatitis and Sexual Health Medicine, Darlinghurst, NSW: Commonwealth of Australia; 2012.
21. National HCV Testing Policy Expert Reference Committee. *National hepatitis C testing policy 2012 v1.1*. Australasian Society for HIV, Viral Hepatitis and Sexual Health Medicine, Darlinghurst, NSW: Commonwealth of Australia; 2012.
22. Heymann DL. *Control of Communicable Diseases Manual*. 19th edn. Washington: American Public Health Association, USA; 2008.
23. National Centre for Immunisation Research and Surveillance of Vaccine Preventable Diseases. Hepatitis B Vaccines for Australians: Information for Immunisation Providers. In; 2015.
24. Medicare Australia. Australian Childhood Immunisation Register – Statistics. 2014. Accessed March 2015. Available from: http://www.medicareaustralia.gov.au/provider/patients/acir/statistics.jsp
25. Gidding HF, Warlow M, MacIntyre CR, Backhouse J, Gilbert GL, Quinn HE, et al. The impact of a new universal infant and school-based adolescent hepatitis B vaccination program in Australia. *Vaccine* 2007;25(51):8637–8641.
26. National Centre for Immunisation Research and Surveillance of Vaccine Preventable Diseases. Significant events in hepatitis B vaccination practice in Australia. 2014. Accessed July 2014. Available from: http://ncirs.edu.au/immunisation/history/Hepatitis-B-history-July-2014.pdf
27. National Centre for Immunisation Research and Surveillance of Vaccine Preventable Diseases. Hepatitis vaccines for Australians, NCIRS fact sheet: June 2012. 2012. Accessed March 2015. Available from: http://www.ncirs.edu.au/immunisation/fact-sheets/hepatitis-B-fact-sheet.pdf
28. Chiu C, Dey A, Wang H, Menzies R, Deeks S, Mahajan D, et al. Vaccine preventable diseases in Australia, 2005 to 2007. *Commun Dis Intell* 2010;34(Suppl):S1–S172.
29. Maclachlan JH, Towell T, Cowie BC. The burden of chronic hepatitis B virus infection in Australia, 2011. *Aust N Z J Public Health* 2013;37(5):416–422.

30. Naidu L, Chiu C, Habig A, Lowbridge C, Jayasinghe S, Wang H, et al. Vaccine preventable diseases and vaccination coverage in Aboriginal and Torres Strait Islander people, Australia 2006–2010. *Commun Dis Intell* 2013;37(Suppl):S1–S95.

31. Australian Bureau of Statistics. Census of Population and Housing: Characteristics of Aboriginal and Torres Strait Islander Australians, 2011, ABS. cat no 2076.0. Canberra: ABS; 2011.

32. Australian Technical Advisory Group on Immunisation. *The Australian Immunisation Handbook*. 10th edn. Canberra, Australia: National Health and Medical Research Council and the Department of Health; 2013.

33. The Kirby Institute. *HIV, viral hepatitis and sexually transmissible infections in Australia Annual Surveillance Report 2014*; 2014.

34. Butler T, Lim D, Callander D. National Prison Entrants' Bloodborne Virus and Risk Behaviour Survey Report 2004, 2007 and 2010. Prevalence of HIV, hepatitis C, hepatitis B, sexually transmissible infections, and risk behaviours among Australian prison entrants; 2011.

35. Razali K, Thein HH, Bell J, Cooper-Stanbury M, Dolan K, Dore G, et al. Modelling the hepatitis C virus epidemic in Australia. *Drug Alcohol Depend* 2007;91(2–3):228–235.

36. OzFoodNet Working Group. Monitoring the incidence and causes of diseases potentially transmitted by food in Australia: Annual report of the OzFoodNet network, 2011. *Commun Dis Intell* 2015;39(2):E236–264.

37. NNDSS Annual Report Writing Group. Australia's notifiable disease status, 2013: Annual report of the National Notifiable Diseases Surveillance System. *Commun Dis Intell* 2015;39(3):E387–E477.

38. Donnan EJ, Fielding JE, Gregory JE, Lalor K, Rowe S, Goldsmith P, et al. A multistate outbreak of hepatitis A associated with semidried tomatoes in Australia, 2009. *Clin Infect Dis* 2012;54(6):775–781.

39. New South Wales Ministry of Health. Health Protection Report, NSW – 2014. Accessed on 8 October 2015. Available from: http://www.health.nsw.gov.au/hpr/Pages/201502-diseases.aspx

40. Lamont RF, Sobel J, Mazaki-Tovi S, Kusanovic JP, Vaisbuch E, Kim SK, et al. Listeriosis in human pregnancy: a systematic review. *J Perinat Med* 2011;39(3):227–236.

41. OzFoodNet Working Group. Monitoring the incidence and causes of diseases potentially transmitted by food in Australia: Annual report of the OzFoodNet Network, 2010. *Commun Dis Intell* 2012;36(3):E213–E241.

42. van den Beld MJ, Reubsaet FA. Differentiation between Shigella, enteroinvasive *Escherichia coli* (EIEC) and noninvasive *Escherichia coli*. (1435–4373 (Electronic)).

43. Queensland Health. Statewide Weekly Communicable Diseases Surveillance Report 30 September 2013,. 2013. Accessed on 7 October 2015. Available from: https://www.health.qld.gov.au/ph/documents/cdb/weeklyrprt-130930.pdf

44. Cumpston JHL. *Health and disease in Australia*. Canberra: Australian Government Publishing Service; 1989.

45. Grattan-Smith PJ O'Regan WJ, Ellis PS, O'Flaherty SJ, McIntyre PB, Barnes CJ. Rabies. A second Australian case with a long incubation period. *Med J Aust* 1992;156(9):651–654.

46. Fenner F, Henderson D, Arita I, Jezek Z, Ladnyi I. Smallpox and its eradication. Geneva, Switzerland; 1988.

47. Australian Government Department of Health and Ageing. Guidelines for smallpox outbreak, preparedness, response and management. 2004. Available from: http://www.health.gov.au/internet/main/publishing.nsf/Content/FD73F6A81331E729CA256F190004427D/$File/smallpox.pdf

48. Miller M, Roche P, Yohannes K, Spencer J, Bartlett M, Brotherton J, et al. Australia's notifiable diseases status, 2003: Annual report of the National Notifiable Diseases Surveillance System. *Commun Dis Intell* 2005;29(1):1–61.

49. World Health Organization. Cumulative number of confirmed human cases of avian influenza A(H5N1) reported to WHO. 2013. Available from: http://www.who.int/influenza/human_animal_interface/H5N1_cumulative_table_archives/en/index.html

50. Curran M, Harvey B, Crerar S. Annual report of the National Notifiable Diseases Surveillance System, 1996. *Commun Dis Intell* 1997;21:281–307.

51. Forssman B, Mannes T, Musto J, Gottlieb T, Robertson G, Natoli JD, et al. *Vibrio cholerae* O1 El Tor cluster in Sydney linked to imported whitebait. *Med J Aust* 2007;187(6):345–347.

52. Geisler W, Lensing S, Press C, Hook EW, 3rd. Spontaneous resolution of genital *Chlamydia trachomatis* infection in women and protection from reinfection. *J Infect Dis* 2013;207(12):1850–1856.

53. Victoria Department of Health. Blue book: Guidelines for the control of infectious diseases. 2009. Accessed on 3 April 2014. Available from: http://ideas.health.vic.gov.au/bluebook/

54. Australasian Society for HIV Medicine. *HIV, viral hepatitis and STIs: a guide for primary care providers*. Darlinghurst: Australasian Society for HIV Medicine; 2014.

55. Bowden FJ, on behalf of the National Donovanosis Eradication Advisory Committee. Donovanosis in Australia: going, going... *Sex Transm Infect* 2005;81(5):365–366.

56. Roberts-Witteveen A, Pennington K, Higgins N, Lang C, Lahra M, Waddell R, et al. Epidemiology of gonorrhoea notifications in Australia, 2007–12. *Sex Health* 2014;11(4):324–331.

57. Lahra M. Australian Gonococcal Surveillance Programme annual report, 2014. *Commun Dis Intell* 2015;39(3):E347–E354.

58. Prestage G, Ferris J, Grierson J, Thorpe R, Zablotska I, Imrie J, et al. Homosexual men in Australia: population, distribution and HIV prevalence. *Sex Health* 2008;5(2):97–102.

59. Allen K, Guy R, Leslie D, Goller J, Medland N, Roth N, et al. The rise of infectious syphilis in Victoria and the impact of enhanced clinical testing. *Aust N Z J Public Health* 2008;32(1):38–42.

60. Australian Broadcasting Corporation. Mount Isa health unit battles rise in syphilis rates. 2014. Accessed on 27 July 2015. Available from: http://www.abc.net.au/news/2014–12–03/mount-isa-health-unit-not-expecting-short-term/5936434

61. Centre for Disease Control Northern Territory. Syphilis outbreak in the NT: 2015 update. *The Northern Territory Disease Control Bulletin* 2015;22(1):1–30.

62. Oaten J. Syphilis outbreak among Northern Territory Indigenous youth prompts fears for unborn children. 2015. Accessed on 28 July 2015. Available from: http://www.abc.net.au/news/2015–07–12/syphilis-outbreak-nt-indigenous-youth-prompts-fears-for-unborn/6613514

63. Baskin B. Queensland remote area health staff are struggling with a syphilis outbreak. *The Daily Telegraph* 7 August 2012.

64. Ward J, Guy RJ, Akre S, Middleton M, Giele C, Su J, et al. Epidemiology of syphilis in Australia: moving toward elimination of infectious syphilis from Aboriginal and Torres Strait Islander communities? *Med J Aust* 2011;194(10):525–529.

65. Australian Government Department of Health. *Fourth National Aboriginal and Torres Strait Islander Blood-borne Viruses and Sexually Transmissible Infections Strategy, 2014–2014*. Canberra: Australian Government Department of Health; 2014.

66. Australian Government Department of Health. *Third National Sexually Transmissible Infections Strategy 2014–2017*. Canberra: Australian Government Department of Health; 2014.

67. Impact of vaccines universally recommended for children. 1900–1998. *MMWR Morb Mortal Wkly Rep* 1999;48(12):243–248.

68. Andre FE, Booy R, Bock HL, Clemens J, Datta SK, John TJ, et al. Vaccination greatly reduces disease, disability, death and inequity worldwide. *Bull World Health Organ* 2008;86(2):81–60.

69. Ehreth J. The global value of vaccination. *Vaccine* 2003;21(7–8):596–600.

70. Préziosi M, Halloran ME. Effects of pertussis vaccination on disease: Vaccine efficacy in reducing clinical severity. *Clin Infect Dis* 2003;37(6):772–779.

71. Schlenker TL, Bain C, Baughman AL, Hadler SC. Measles herd immunity: the association of attack rates with immunization rates in preschool children. *JAMA* 1992;267(6):823–826.

72. Punpanich W, Chotpitayasunondh T. A review on the clinical spectrum and natural history of human influenza. *Int J Infect Dis* 2012;16(10):e714–e723.

73. Carrat F, Vergu E, Ferguson NM, Lemaitre M, Cauchemez S, Leach S, et al. Time lines of infection and disease in human influenza: a review of volunteer challenge studies. *Am J Epidemiol* 2008;167:775–785.

74. Mauskopf J, Klesse M, Lee S, Herrera-Taracena G. The burden of influenza complications in different high-risk groups: a targeted literature review. *J Med Econ* 2013;16(2):264–277.

75. Bennett JE, Dolin R, Blaser MJ. *Principles and Practice of Infectious Diseases*. 8th edn: Elsevier Health Sciences; 2014.

76. de Jong MD, Thanh TT, Khanh TH, Hien VM, Smith GJD, Chau NV, et al. Oseltamivir resistance during treatment of influenza A (H5N1) infection. *N Engl J Med* 2005;353(25):2667–2672.

77. National Centre for Immunisation Research and Surveillance of Vaccine Preventable Diseases. *Haemophilus influenzae* type b (Hib) vaccines for Australian children, NCIRS Fact sheet: October 2009. Sydney: National Centre for Immunisation Research and Surveillance for Vaccine Preventable Diseases; 2009.

78. Chandran A, Watt J, Santosham M. *Haemophilus influenzae*. In: Plotkin SA, Orenstein WA, Offit PA, editors. *Vaccines*. Philadelphia, PA, USA: Saunders Elsevier; 2008.

79. National Centre for Immunisation Research and Surveillance of Vaccine Preventable Diseases. Significant events in pneumococcal vaccination practice in Australia. 2015. Accessed on 26 May 2015. Available from: http://ncirs.edu.au/immunisation/history/Pneumococcal-history-March-2015.pdf

80. Centers for Disease Control and Prevention. Measles. In: Atkinson W, Wolfe C, Hamborsky J, eds. *Epidemiology and prevention of vaccine preventable diseases*. 12th edn. Washington, D.C.: Public Health Foundation; 2011.

81. World Health Organization. Measles Fact Sheet Fact sheet N°286. Updated November 2015. Accessed November 2015. Available from: http://www.who.int/mediacentre/factsheets/fs286/en/

82. Strebel P, Papania M, Dayan G, Halsey N. Vaccines. In: Plotkin SA, Orenstein WA, Offit PA, editors. Philadelphia: Saunders Elsevier; 2008.

83. Heywood AE, Gidding HF, Riddell MA, McIntyre PB, MacIntyre CR, Kelly HA. Elimination of endemic measles transmission in Australia. *Bull World Health Organ* 2009;87(1):64–71.

84. Gidding H, Wood J, MacIntyre CR, Kelly H, Lambert SB, Gilbert GL, et al. Sustained measles elimination in Australia and priorities for long-term maintenance. *Vaccine* 2007;25(18):3574–3580.

85. Gidding HF, Gilbert GL. Measles immunity in young Australian adults. *Commun Dis Intell* 2001;25(3):133–136.

86. de Serres G, Gay NJ, Farrington CP. Epidemiology of transmissible diseases after elimination. *Am J Epidemiol* 2000;151(11):1039–1048.

87. World Health Organization. Four Western Pacific countries and areas are the first in their Region to be measles-free. 2014. Available from: http://www.wpro.who.int/mediacentre/releases/2014/20140320/en/

88. Plotkin SA, Rubin S. *Mumps vaccine*. 5th edn. Philadelphia, PA: Saunders Elsevier; 2008.

89. Deeks S, Lim G, Simpson M, Gagné L, Kristjanson E, Fung C, et al. An assessment of mumps vaccine effectiveness by dose during an outbreak in Canada. *Can Med Assoc J* 2011;183(9):1014–1020.

90. Demicheli V, Rivetti A, Debalini M, Di Pietrantonj C. Vaccines for measles, mumps and rubella in children. *Cochrane Database Sys Rev* 2012;2:CD004407.

91. Gupta R, Best J, MacMahon E. Mumps and the UK epidemic 2005. *BMJ* 2005;330:1132–1135.

92. Dayan GH, Quinlisk MP, Parker AA, Barskey AE, Harris ML, Schwartz JM, et al. Recent resurgence of mumps in the United States. *N Engl J Med* 2008;358(15):1580–1589.

93. Whelan J, Van Binnendijk R, Greenland K, Fanoy E, Kharqi M, Yap K, et al. Ongoing mumps outbreak in a student population with high vaccination coverage, Netherlands. *Euro Surveill* 2010;15(17):pii:19554.

94. Cohen C, White JM, Savage EJ, Glynn JR, Choi Y, Andrews N, et al. Vaccine effectiveness estimates, 2004–2005 mumps outbreak, England. *Emerg Infect Dis* 2007;13(1):12–17.

95. Pertussis. In: Atkinson W, Wolfe C, Hamborsky J, editors. *Epidemiology and prevention of vaccine preventable diseases*. 12th edn. Washington, DC: Public Health Foundation, Centers for Disease Control and Prevention; 2011.

96. Communicable Diseases Network Australia. Series of National Guidelines – Pertussis. 2013. Accessed March 2015. Available from: https://www.health.gov.au/internet/main/publishing.nsf/Content/cdna-song-pertussis.htm

97. Quinn H, McIntyre PB. Impact of removal of the 18 month DTPa dose on pertussis vaccine effectiveness. *12th National Immunisation Conference*. Adelaide, South Australia; 2010.

98. Quinn HE, Snelling TL, Macartney KK, McIntyre PB. Duration of protection after first dose of acellular pertussis vaccine in infants. *Pediatrics* 2014;133(3):e513–e519.

99. Sheridan SL, McCall BJ, Davis CA, Robson JM, Hull BP, Selvey CE, et al. Acellular pertussis vaccine effectiveness for children during the 2009–2010 pertussis epidemic in Queensland. *Med J Aust* 2014;200(6):334–338.

100. Wendelboe AM, van Rie A, Salmaso S, Englund JA. Duration of immunity against pertussis after natural infection or vaccination. *Pediatr Infect Dis J* 2005;24(5 Suppl):S58–S61.

101. Munoz FM. Pertussis in infants, children, and adolescents: diagnosis, treatment, and prevention. *Semin Pediatr Infect Dis* 2006;17(1):14–19.

102. McLean H, Redd S, Abernathy E, Icenogle J, Wallace G. Chapter 15: Congenital rubella syndrome. In: *Manual for the Surveillance of Vaccine-Preventable Diseases*. Atlanta: Centers for Disease Control and Prevention; 2012.

103. Song N, Gao Z, Wood J, Hueston L, Gilbert C, MacIntyre R, et al. Current epidemiology of rubella and congenital rubella syndrome in Australia: Progress towards elimination. *Vaccine* 2012;30(27):4073–4078.

104. Western Pacific Regional Office. *Rubella and congenital rubella syndrome* Geneva: World Health Organization; 2012.

105. Khandaker G, Zyrynski Y, Jones C. Surveillance for congenital rubella syndrome in Australia since 1993: cases reported between 2004 and 2013. *Vaccine* 2014;32(50):6746–6751.

106. Quinn H, McIntyre P. Tetanus in the elderly – An important preventable disease in Australia. *Vaccine* 2007;25(7):1304–1309.

107. Mackenzie JS, Lindsay MD, Coelen RJ, Broom AK, Hall RA, Smith DW. Arboviruses causing human disease in the Australasian zoogeographic region. *Arch Virol* 1994;136(3–4):447–467.

108. Russell RC, Dwyer DE. Arboviruses associated with human disease in Australia. *Microbes Infect* 2000;2(14):1693–1704.

109. Viennet E, Knope K, Faddy H, Williams C, Harley D. Assessing the threat of chikungunya emergence in Australia. *Commun Dis Intell* 2013;37(2):E136–E143.

110. Knope K, Kurucz N, Doggett S, Muller M, Johansen C, Feldman R, et al. Arboviral diseases and malaria in Australia, 2012–13: annual report of the National Arbovirus and Malaria Advisory Committee. *Commun Dis Intell* 2016;40(1):E17–E47.

111. Communicable Diseases Network Australia. Australian National Notifiable Diseases case definitions. 2014. Accessed on 5 September 2015. Available from: http://www.health.gov.au/casedefinitions

112. Therapeutic Goods Administration. Product recall, Panbio Barmah Forest virus IgM ELISA. An *in vitro* diagnostic medical device (IVD). Recall no. RC-2013-RN-00967-1,13/09/2013. 2013. Accessed on 6 May 2014. Available from: http://www.tga.gov.au/SARA/arn-detail.aspx?k=RC-2013-RN-00967-1

113. Rich G, McKechnie J, McPhan I, Richards B. Laboratory diagnosis of Ross River virus infection. *Commun Dis Intell* 1993;17(10):208–209.

114. Selvey LA, Donnelly JA, Lindsay MD, Pottumarthy Boddu S, D'Abrera VC, Smith DW. Ross River virus infection surveillance in the Greater Perth Metropolitan Area – has there been an increase in cases in the winter months? *Commun Dis Intell* 2014;38(2):E114–E122.

115. Guzman MG, Kouri G, Martinez E, Bravo J, Riveron R, Soler M, et al. Clinical and serologic study of Cuban children with dengue hemorrhagic fever/dengue shock syndrome (DHF/DSS). *Bull Pan Am Health Organ* 1987;21(3):270–279.

116. Vaughn DW, Green S, Kalayanarooj S, Innis BL, Nimmannitya S, Suntayakorn S, et al. Dengue viremia titer, antibody response pattern, and virus serotype correlate with disease severity. *J Infect Dis* 2000;181(1):2–9.

117. Mackenzie JS, Gubler DJ, Petersen LR. Emerging flaviviruses: the spread and resurgence of Japanese encephalitis, West Nile and dengue viruses. *Nat Med* 2004;10(12 Suppl):S98–S109.

118. Souarès Y, Pacific Public Health Surveillance Network. Telehealth and outbreak prevention and control: the foundations and advances of the Pacific Public Health Surveillance Network. *Pac Health Dialog* 2000;7(2):11–28.

119. Hanna JN, Ritchie SA, Richards AR, Humphreys JL, Montgomery BL, Ehlers GJ, et al. Dengue in north Queensland, 2005–2008. *Commun Dis Intell* 2009;33(2):198–203.

120. Queensland Health. *Queensland Dengue Management Plan 2010–2015, 2011*. Queensland: Queensland Health.

121. Knope K, National Arbovirus and Malaria Advisory Committee, Giele C. Increasing notifications of dengue related to overseas travel, 1991 to 2012. *Commun Dis Intell* 2013;37(1):E55–E59.

122. Hanna JN, Ritchie SA, Phillips DA, Lee JM, Hills SL, van den Hurk AF, et al. Japanese encephalitis in north Queensland, Australia, 1998. *Med J Aust* 1999;170(11):533–536.

123. Knope K, Whelan P, Smith D, Johansen C, Moran R, Doggett S, et al. Arboviral diseases and malaria in Australia, 2010–11: annual report of the National Arbovirus and Malaria Advisory Committee. *Commun Dis Intell* 2013;37(1):E1–E20.

124. Knox J, Cowan R, Doyle J, Ligtermoet M, Archer J, Burrow J, et al. Murray Valley encephalitis: a review of clinical features, diagnosis and treatment. *Med J Aust* 2012;196(5):322–326.

125. Roche S, Wicks R, Garner M, East IJ, Paskin R, Moloney BJ, et al. Descriptive overview of the 2011 epidemic of arboviral disease in horses in Australia. *Aust Vet J* 2013;91(1–2):5–13.

126. Frost MJ, Zhang J, Edmonds JH, Prow NA, Gu X, Davis R, et al. Characterization of virulent West Nile virus Kunjin strain, Australia, 2011. *Emerg Infect Dis* 2012;18(5):792–800.

127. *Plasmodium knowlesi* malaria in humans is widely distributed and potentially life threatening. *Clin Infect Dis* 2008;46(2):165–171. doi: 110.1086/524888.

128. World Health Organization. Synopsis of the world malaria situation in 1981. *Wkly Epidemiol Rec* 1983;58(26):197–199.

129. Leder K, Torresi J, Brownstein JS, Wilson ME, Keystone JS, Barnett E, et al. Travel-associated illness trends and clusters, 2000–2010. *Emerg Infect Dis* 2013;19(7):1049–1073.

130. Gray TJ, Trauer JM, Fairley M, Krause VL, Markey PG. Imported malaria in the Northern Territory, Australia—428 consecutive cases. *Commun Dis Intell* 2012;36(1):107–113.

131. Hanna JN, Ritchie SA, Eisen DP, Cooper RD, Brookes DL, Montgomery BL. An outbreak of *Plasmodium vivax* malaria in Far North Queensland, 2002. *Med J Aust* 2004;180(1):24–28.

132. World Health Organization. Zoonoses. Technical report series no. 169. Geneva, Switzerland: World Health Organization; 1959.

133. Taylor LH, Latham SM, Woolhouse ME. Risk factors for human disease emergence. *Philos Trans R Soc Lond B Biol Sci* 2001;356(1411):983–989.

134. Jones KE, Patel NG, Levy MA. Global trends in emerging infectious diseases. *Nature* 2008(451):990–994.

135. Woolhouse MEJ, Gowtage-Sequeria S. Host range and emerging and re-emerging pathogens. *Emerg Infect Dis* 2005;11(12):1842–1847.

136. World Health Organization. Report of the WHO/FAO/OIE joint consultation on emerging zoonotic diseases. Geneva, Switzerland: World Health Organization; 2004.

137. Animal Health Australia. Animal Health in Australia, 2014. Canberra: Animal Health Australia; 2015.

138. Kolbe A, Yuen M, Doyle B. A case of human cutaneous anthrax. *Med J Aust* 2006;185(5):281–282.

139. Fielding J. Zoonoses: Anthrax. *Victorian Infectious Diseases Bulletin* 2007;10(2):47.

140. NSW Department of Health. Communicable Diseases Report, NSW, January and February 2010. *N S W Public Health Bull* 2010;21(3–4):103–107.

141. Calisher CH, Ellison JA. The other rabies viruses: The emergence and importance of lyssaviruses from bats and other vertebrates. *Travel Med Infect Dis* 2012;10(2):69–79.

142. Fraser GC, Hooper PT, Lunt RA, Gould AR, Gleeson LJ, Hyatt AD, et al. Encephalitis caused by a lyssavirus in fruit bats in Australia. *Emerg Infect Dis* 1996;2(4):327–331.

143. Hooper PT, Lunt RA, Gould AR, Samaratunga H, Hyatt AD, Gleeson LJ, et al. A new lyssavirus — the first endemic rabies-related virus recognized in Australia. *Bulletin de l'Institut Pasteur* 1997;95(4):209–218.

144. Communicable Diseases Network Australia. Series of National Guidelines: Rabies virus and other lyssavirus including Australian bat lyssavirus) exposures and infections. 2013. Accessed on 7 June 2015. Available from: http://www.health.gov.au/internet/main/publishing.nsf/Content/cdna-song-abvl-rabies.htm

145. Allworth A, Murray K, Morgan J. A human case of encephalitis due to a lyssavirus recently identified in fruit bats. *Commun Dis Intell* 1996;20(24):504.

146. Hanna JN, Carney IK, Smith GA, Tannenberg AEG, Deverill JE, Botha JA, et al. Australian bat lyssavirus infection: a second human case, with long incubation period. *Med J Aust* 2000;172(12):597–599.

147. Francis JR, Nourse C, Vaska VL, Calvert S, Northill JA, McCall B, et al. Australian bat lyssavirus in a child: The first reported case. *Pediatrics* 2014;133(4):e1063–1067.

148. Queensland Department of Agriculture. Australian bat lyssavirus overview. In: *Animal Health and Diseases*: Queensland Government; 2013.

149. Crook A. Australian bat lyssavirus communiqué. Department of Agriculture, Fisheries and Forestry; 2013.

150. Bat Health Focus Group. ABLV Bat Stats, Australian Bat Lyssavirus report – December 2014. *Wildlife Health Australia* 2015.

151. Eales KM, Norton RE, Ketheesan N. Brucellosis in northern Australia. *Am J Trop Med Hyg* 2010;83(4):876–878.

152. Levett PN. Leptospirosis. *Clin Microbiol Rev* 2001;14(2):296–326.

153. World Health Organization, Society IL. Human Leptospirosis: Guidance for Diagnosis, Surveillance and Control; 2003.

154. O'Leary FM, Hunjan JS, Bradbury R, Thanakrishnan G. Fatal leptospirosis presenting as musculoskeletal chest pain. *Med J Aust* 2004;180(1):29–31.

155. Beeckman DS, Vanrompay DC. Zoonotic *Chlamydophila psittaci* infections from a clinical perspective. *Clin Microbiol Infect* 2009;15(1):11–17.

156. Deschuyffeleer TP, Tyberghien LF, Dickx VL, Geens T, Saelen JM, Vanrompay DC, et al. Risk assessment and management of *Chlamydia psittaci* in poultry processing plants. *Ann Occup Hyg* 2012;56(3):340–349.

157. McCaul TF, Williams JC. Developmental cycle of *Coxiella burnetii:* structure and morphogenesis of vegetative and sporogenic differentiations. *J Bacteriol* 1981;147(3):1063–1076.

158. Lowbridge CP, Tobin S, Seale H, Ferson MJ. Notifications of Q fever in NSW, 2001–2010. *N S W Public Health Bull* 2012;23(1–2):31–35.

159. Bell M, Patel M, Sheridan J. Q fever vaccination in Queensland abattoirs. *Commun Dis Intell* 1997;21(3):29–31.

160. Lin M, Delpech V, McAnulty J, Campbell-Lloyd S. Notifications of Q fever in New South Wales, 1991–2000: EpiReview. *N S W Public Health Bull* 2001;12(6):172–175.

161. Gidding HF, Wallace C, Lawrence GL, McIntyre PB. Australia's National Q Fever Vaccination Program. *Vaccine* 2009;27(14):2037–2041.

162. Ellis J, Oyston PC, Green M, Titball RW. Tularemia. *Clin Microbiol Rev* 2002;15(4):631–646.

163. Jackson J, McGregor A, Cooley L, Ng J, Brown M, Ong CW, et al. *Francisella tularensis* subspecies holarctica, Tasmania, Australia, 2011. *Emerg Infect Dis* 2012;18(9):1484–1486.

164. Veitch M. The public health response to tularaemia in Tasmania. *Communicable Disease Control Conference 2013*. Canberra, Australia; 2013.

165. NNDSS Annual Report Writing Group. Australia's notifiable disease status, 2011: Annual report of the National Notifiable Diseases Surveillance System. *Commun Dis Intell* 2013;37(4):E313–E393.

166. de Jong B. *Legionella,* springtime and potting soils. *Euro Surveill* 2010;15(8):19497.

167. O'Connor BA, Carman J, Eckert K, Tucker G, Givney R, Cameron S. Does potting mix make you sick? Results for a *Legionella longbeachae* case–control study in South Australia. *Epidemiol Infect* 2007;135(1):34–39.

168. Pravinkumar SJ, Edwards G, Lindsay D, Redmond S, Stirling J, House R, et al. A cluster of Legionnaires' disease caused by *Legionella longbeachae* linked to potting compost in Scotland, 2008–2009. *Euro Surveill* 2010;15(8):19496.

169. Whiley H, Bentham R. *Legionella longbeachae* and legionellosis. *Emerg Infect Dis* 2011;17(4):579–583.

170. Li J, O'Brien ED, Guest C. A review of national legionellosis surveillance in Australia, 1991 to 2000. *Commun Dis Intell* 2002;26:462–470.

171. Rota MC, Caporal MG, Bella A, Ricci ML, Napoli C. Legionnaires disease in Italy: results of the epidemiological surveillance from 2000 to 2011. *Euro Surveill* 2013;18(23).

172. World Health Organization. Leprosy – Fact Sheet No. 101. 2015. Accessed on 19 June 2015. Available from: http://www.who.int/mediacentre/factsheets/fs101/en/

173. Communicable Diseases Network Australia. Meningococcal disease (invasive) surveillance case definition – V1.4. 2009. Accessed on 6 July 2015. Available from: http://www.health.gov.au/internet/main/publishing.nsf/Content/cda-surveil-nndss-case-defs-cd_mening.htm

174. Jafri RZ, Ali A, Messonnier NE, Tevi-Benissan C, Durrheim D, Eskola J, et al. Global Epidemiology of invasive meningococcal disease. *Population Health Metrics* 2013;11(17).

175. Lahra MM, Enriquez R. Annual Report of the Australian Meningococcal Surveillance Programme, 2014. *Commun Dis Intell* 2015:In press.

176. Bareja C, Waring J, Stapledon R, National Tuberculosis Advisory Committee for the Communicable Diseases Network Australia. Tuberculosis notifications in Australia, 2010. *Commun Dis Intell* 2014;38(1):E36–E48.

177. Luca S, Mihaescu T. History of BCG Vaccine. *Maedica (Burchar)* 2013;8(1):53–58.

178. Public Health England. Tuberculosis: the green book, chapter 32. In: *Immunisation against infectious disease* London: Department of Health; 2013. p. 394.

179. National Tuberculosis Advisory Committee. The BCG vaccine: Information and recommendations for use in Australia. *Commun Dis Intell* 2013;37(1):E65–E72.

Immunisation coverage annual report, 2013

Brynley P Hull, Aditi Dey, Frank H Beard, Robert I Menzies, Julia M Brotherton, Peter B McIntyre

Executive summary

This 7th annual immunisation coverage report shows data for 2013 from the Australian Childhood Immunisation Register (ACIR) and National Human Papillomavirus (HPV) Vaccination Program Register. From July 2013, 2 new combined vaccines were included on the National Immunisation Program (NIP): *Haemophilus influenzae* type b-meningococcal C conjugate and measles-mumps-rubella-varicella (MMRV), the latter involving moving the 2nd dose of MMR vaccine from 48 months to 18 months of age. For the first time, from December 2013, the definition of 'fully immunised' at 12 months of age included pneumococcal conjugate vaccine. This report includes coverage in 2013 for 'fully immunised' and individual vaccines by Indigenous status at standard age milestones, and assessment of timeliness of vaccination.

Immunisation coverage

The proportion of Australian children classified as 'fully immunised' at 12 months of age was 90.8%, at 24 months 92.1% and at 60 months 91.2%. Vaccines available on the NIP but not assessed during 2013 for 'fully immunised' status or eligibility for incentive payments included pneumococcal conjugate and rotavirus vaccines at 12 months of age and meningococcal C conjugate and varicella vaccines at 24 months of age. Coverage for pneumococcal conjugate (90.9%) and meningococcal C conjugate (93.2%) vaccines was similar to that for other vaccines administered at the same schedule points. Coverage was lower for rotavirus vaccine (for which upper age limits apply) at 12 months of age (83.6%) and for varicella vaccine at 24 months of age (84.8%). However, varicella coverage increased 3.5 percentage points (jurisdictional range 0.7 to 5.1) in the first 6 months following the introduction of MMRV. For HPV vaccine at the national level, 71.0% of females aged 14–15 years had 3 documented doses (jurisdictional range 62.8% to 80.0%). Coverage for the 1st dose was 82.0%. Recorded coverage among women in their 20s vaccinated largely outside of school (64.0% for 1 dose, 48.0% for 3 doses) was lower.

Indigenous immunisation coverage

Coverage for 'fully immunised' at 12 months of age among Indigenous children was lower than for non-Indigenous children in all jurisdictions, with the differential varying from 11.6 percentage points in South Australia to 1.4 percentage points in the Northern Territory. However, coverage was similar for Indigenous children at 24 months of age and higher at 60 months of age. Hepatitis A vaccine and the additional pneumococcal vaccine booster dose, recommended for Indigenous children only, had sub-optimal recorded coverage of 60.1% and 59.9%, respectively.

Timeliness

Disparity in on-time vaccination between Indigenous and non-Indigenous children remained similar to previous years. It declined progressively from 21.6% at 12 months to 12.9% at 24 months of age and 5.8% at 60 months of age. By late 2013, the percentage of children who received the 1st dose of diphtheria-tetanus-pertussis acellular (child formulation) (DTPa)-containing vaccine at less than 8 weeks of age was greater than 50.0% in all but 1 jurisdiction and greater than 70.0% in 5 jurisdictions. By late 2013, the percentage of children who received the 4th dose of DTPa-containing vaccine dose at less than 4 years of age was greater than 35.0% in 3 jurisdictions.

Vaccination objection

In 2013, the proportion of children whose parents were registered objectors to vaccination was 1.9% at the national level, with large regional variations. This percentage has increased slowly over time.

Keywords: immunisation coverage, immunisation delay, Indigenous immunisation coverage, vaccination objection, human papillomavirus vaccine coverage

Introduction

This is the 7th annual immunisation coverage report, with the 1st report having focused on 2007 data.[1–6] It complements other reports providing data on immunisation coverage in Australia[7] and highlights important trends and significant issues. It follows the format of the previous reports, providing a detailed summary for 2013 that includes vaccination coverage at standard milestone ages, coverage for vaccines not included in standard coverage assessments, timeliness of vaccination, coverage for Indigenous children, analysis of 'partially immunised' children, and data for small geographic areas on the prevalence of vaccination objectors. This report also includes data

on adolescents outside the Australian Childhood Immunisation Register (ACIR) age group from previously published sources. Readers are referred to the 1st report for a more detailed explanation of the background to this series of annual reports and the range of analyses presented.[1] This report uses the long-standing international practice of reporting at key milestone ages to measure coverage against national targets and to track trends over time. Table 1 shows the Australian National Immunisation Program Schedule for 2013.

High levels of reporting to the ACIR are maintained by a system of incentive payments for immunisation providers and carers. These have been discussed in detail elsewhere.[6,8] Some recent changes to immunisation policy, the incentive payment system and changes to the 'fully immunised' coverage algorithms are highlighted in the Box and also referred to in this report.

Table 1: Australian National Immunisation Program Schedule for children, adolescents and adults in 2013

Age	Vaccine									
Childhood vaccines										
Birth	Hep B									
2 months	Hep B	DTPa	Hib	Polio				13vPCV	Rotavirus	
4 months	Hep B	DTPa	Hib	Polio				13vPCV	Rotavirus	
6 months	Hep B	DTPa	Hib	Polio				13vPCV	Rotavirus*	
12 months			Hib-MenCCV†		MMR		Hep A‡			
18 months						MMRV§	Hep A‡‖	13vPCV‡		
24 months							Hep A‖	13vPCV‖		
48 months		DTPa		Polio	MMR¶					
Adolescent vaccines										
12 years	Hep B**	dTpa				VZV**				HPV††
15 years		dTpa							Flu‡‡,§§	23vPPV¶¶
Adult vaccines										
≥50 years									Flu‡‡,§§	23vPPV‡‡
65 years									Flu§§	23vPPV

* 3rd dose of rotavirus vaccine at 6 months of age is dependent on vaccine brand used in each state or territory.

† In July 2013, the combined *Haemophilus influenzae* type b (Hib) and meningococcal serogroup C conjugate (MenCCV) vaccine, Menitorix®, was added to the National Immunisation Program (NIP) Schedule at 12 months of age. This combination vaccine replaces the single dose of monovalent MenCCV vaccine and booster dose of monovalent Hib vaccine previously scheduled at 12 months of age.

‡ Aboriginal and Torres Strait Islander children in the Northern Territory and Western Australia.

§ Measles-mumps-rubella-varicella vaccine introduced onto the NIP Schedule on 1 July 2013.

‖ Aboriginal and Torres Strait Islander children in Queensland and South Australia. The hepatitis A vaccination schedule for Indigenous children changed in July 2013 so that now dose 1 is administered to Indigenous children at 12 months of age and dose 2 at 18 months of age in all 4 jurisdictions (the Northern Territory, Queensland, South Australia and Western Australia).

¶ The dose of measles-mumps-rubella vaccine at 4 years of age will cease on 1 January 2016.

** Catch-up only.

†† From February 2013, males and females aged 12–13 years received the human papillomavirus vaccine at school. Males aged 14–15 years also received the vaccine as part of a catch-up program until the end of the 2014 school year.

‡‡ For Aboriginal and Torres Strait Islander people only.

§§ Annual vaccination, all aged ≥6 months with medical risk factors, non-Indigenous adults aged ≥65 years.

¶¶ Aboriginal and Torres Strait Islander adults with medical risk factors.

Box: Recent significant changes in immunisation policy, immunisation incentives and coverage calculation algorithms

- July 2013: The combined *Haemophilus influenzae* type b (Hib)-meningococcal serogroup C conjugate vaccine, (MenCCV) Menitorix®, was added to the National Immunisation Program (NIP) Schedule at 12 months of age. This combination vaccine replaces the single dose of monovalent MenCCV and booster dose of monovalent Hib vaccine previously scheduled at 12 months of age.
- A combination measles-mumps-rubella-varicella (MMRV) vaccine for children aged 18 months was added to the NIP, replacing the separate measles-mumps-rubella (MMR) vaccine previously given at 4 years, and the varicella vaccine (for chickenpox) previously given at 18 months. MMR vaccination at 4 years of age is to continue in parallel until the first cohort eligible for MMRV vaccine reaches 4 years of age.
- Pneumococcal vaccine was added to the list of immunisations that children need to receive to be assessed as 'fully immunised' at 12 months of age. The expansion of the definition of 'fully immunised' reinforces the importance of vaccines by linking them to payments to families and immunisation providers.[9]
- The hepatitis A vaccination schedule for Indigenous children changed so that now dose 1 is administered to Indigenous children at 12 months of age and dose 2 at 18 months of age in all 4 relevant jurisdictions (the Northern Territory, Western Australia, Queensland and South Australia).
- February 2013: The human papillomavirus vaccine was funded under the NIP for males aged 12–13 years, delivered in school-based programs.
- July 2012: Eligibility for the Family Tax Benefit Part A supplement requires that children are assessed as fully immunised, replacing the Maternity Immunisation Allowance. To meet the immunisation requirements for the Family Tax Benefit Part A supplement parents need to have their children immunised during the financial years that each child turns 1, 2 and 5 years of age. Children need to be up-to-date with immunisation or have an approved exemption.
- October 2011: Prevenar 13® (13-valent pneumococcal conjugate vaccine, 13vPCV) replaced 23vPPV as a booster dose in Indigenous children living in the Northern Territory, Western Australia, Queensland and South Australia.
- July 2011: Prevenar 13® (13-valent pneumococcal conjugate vaccine, 13vPCV) replaced Prevenar® (7-valent pneumococcal conjugate vaccine, 7vPCV) on the NIP for children at 2, 4 and 6 months of age in all states and territories except the Northern Territory (adopted 13vPCV from 1 October 2011).
- September 2009: Changes in the coverage calculation algorithms occurred that tightened the rules regarding receipt of Hib and hepatitis B vaccines for children aged 12 and 24 months.
- October 2009: Recommendation by the Australian Technical Advisory Group on Immunisation (ATAGI) that the 4th dose of diphtheria-tetanus-pertussis acellular (child formulation) (DTPa)-containing vaccine can be given from 3.5 years of age instead of the previously recommended 4 years of age.
- March 2009: Recommendation by ATAGI to parents and immunisation providers to consider bringing the 1st dose of DTPa forward to 6 weeks of age to provide earlier protection.
- January 2009: Changes to the overdue rules so that children were classified as overdue for preschool boosters at 4 years and 1 month instead of the previous 5 years of age. This applied to parental and provider incentive payments.
- December 2007: The coverage algorithm for immunisations due at 48 months of age was changed to assess children at 60 months rather than 72 months of age.

Methods

The Australian Childhood Immunisation Register

The ACIR was established on 1 January 1996 by incorporating demographic data from Medicare on all enrolled children under the age of 7 years.[10] Participation in the ACIR is opt-out so it constitutes a nearly complete population register, as approximately 99.0% of children are registered with Medicare by 12 months of age.[10] Children not enrolled in Medicare can also be added to the ACIR via a supplementary number. Since 2001, immunisations given overseas may be recorded if a provider endorses their validity. Data are transferred to the ACIR when a recognised immunisation provider supplies details of an eligible immunisation either automatically from medical practice software or through the internet using the Medicare Australia website or by submitting paper encounter forms. The existence of medical contraindications and vaccination objection to immunisation is also recorded on the ACIR. All vaccination records for a child remain on the register indefinitely, but no new immunisation encounter records are added after the 7th birthday.

Immunisations recorded on the ACIR must be rendered in accordance with the guidelines issued by the Australian Technical Advisory Group on Immunisation (ATAGI).[11] Notifications falling outside these guidelines or duplicate notifications prompt an enquiry with the provider and, if their validity cannot be established, they are rejected.

Measuring immunisation coverage using the Australian Childhood Immunisation Register

The cohort method has been used for calculating coverage at the population level (national and state or territory)[12] since the ACIR's inception. Cohort immunisation status is assessed at 12 months of age (for vaccines due at 6 months), 24 months of age (for vaccines due at 12 months), and 60 months of age (for vaccines due at 48 months). A minimum 3-month lag period is allowed for late notification of immunisations to the ACIR, but only immunisations given on or before a child's 1st, 2nd or 5th respective birthdays are considered.[12] If a child's records indicate receipt of the last dose of a vaccine that requires more than 1 dose to complete the series, it is assumed that earlier vaccinations in the sequence have been given. This assumption has been shown to be valid.[13,14]

Three-month birth cohorts are used for time trend analyses, while 12-month wide cohorts are used for other analyses in this report such as for small area coverage. The 12-month wide cohorts used in this report are children born between 1 January and 31 December 2012 for the 12-month milestone age; children born between 1 January and 31 December 2011 for the 24-month milestone age; and children born between 1 January and 31 December 2008 for the 5-year (60-month) milestone age.

The proportion of children designated as 'fully immunised' is calculated using the number of children completely immunised with the vaccines of interest by the designated age as the numerator, and the total number of Medicare-registered children in the age cohort as the denominator. 'Fully immunised' at 12 months of age was defined as a child having a record on the ACIR of a 3rd dose of the combined DTPa-hepB-IPV-Hib vaccine (and a 3rd dose of pneumococcal conjugate vaccine since the December 2013 quarterly coverage report). 'Fully immunised' at 24 months of age was defined as a child having a record on the ACIR of a 3rd dose of the combined DTPa-hepB-IPV-Hib vaccine, a 4th dose of *Haemophilus influenzae* type b (Hib) (PRP-T) vaccine, and a 1st dose of a measles-mumps-rubella (MMR)-containing vaccine. 'Fully immunised' at 60 months of age was defined as a child having a record on the ACIR of a 4th dose of combined DTPa-IPV vaccine and a 2nd dose of an MMR-containing vaccine.

Immunisation coverage estimates were also calculated for individual National Immunisation Program (NIP) vaccines, including the 6 NIP vaccines not routinely reported in the quarterly coverage reports published in *Communicable Diseases Intelligence*[15] and not part of 'fully immunised' calculations at 12 and 24 months of age. They were: a 2nd or 3rd dose of rotavirus vaccine by 12 months of age; a 1st dose of varicella vaccine and a 1st dose of meningococcal C conjugate vaccine by 24 months of age; a 2nd dose of hepatitis A vaccine in Indigenous children by 30 or 36 months of age; and a dose of pneumococcal vaccine in Indigenous children by 30 months of age. In July 2013, the hepatitis A vaccination schedule for Indigenous children changed so that now dose 1 is administered to Indigenous children at 12 months of age and dose 2 at 18 months of age in all 4 jurisdictions (the Northern Territory, Queensland, South Australia and Western Australia). However, hepatitis A coverage data presented in this report uses the old calculation with assessment by 30 or 36 months of age as the new assessment ages do not apply to the cohort analysed in this report.

Changes to immunisation policy and changes to the 'fully immunised' coverage algorithms have had an impact on vaccination coverage presented in this report. In December 2007, the coverage algorithm for immunisations due at 48 months of age was changed to assess children at 60 months

rather than 72 months of age. In January 2009, changes were made to the overdue rules so that children were classified as overdue for pre-school boosters at 49 months instead of the previous 60 months of age. In March 2009, a recommendation was made by ATAGI to parents and immunisation providers to consider bringing the 1st dose of diphtheria-tetanus-pertussis acellular (child formulation) (DTPa) forward to 6 weeks of age to provide earlier protection against pertussis infection. From the September 2009 coverage assessment date onwards, changes were made in the coverage calculation algorithms that tightened the rules regarding receipt of Hib and hepatitis B vaccines for children aged 12 and 24 months of age. Prior to September 2009, if a child aged 12 months had a record on the ACIR of a 2nd or 3rd dose of any Hib vaccine, he or she was considered 'fully immunised'. From September 2009, a child needed a record on the ACIR of a 3rd dose of any Hib vaccine or a 2nd dose of either PedvaxHIB or Comvax to be assessed as 'fully immunised'. Prior to September 2009, if a child aged 12 months had a record on the ACIR of a 2nd or 3rd dose of any hepatitis B vaccine, he or she was considered 'fully immunised'. From September 2009, a child needed a record on the ACIR of a 3rd dose of any hepatitis B vaccine or a 2nd dose of either Engerix-B (paediatric), Comvax or H-B-VAX II (paediatric) to be assessed as 'fully immunised' at 12 months of age. In October 2009, a recommendation was made by ATAGI that the 4th dose of DTPa-containing vaccine can be given from 42 months (3.5 years) of age instead of the previously recommended 48 months of age. From the December 2013 quarterly coverage report, the 3rd dose of pneumococcal conjugate vaccine was included in coverage requirements for 'fully immunised' at the 12-month milestone.

Timeliness

Age-appropriate immunisation was defined as receipt of a scheduled vaccine dose within 30 days of the recommended age. For example, a child who received the 1st dose of DTPa (due at 60 days of age) when he or she was more than 90 days of age was classified as late for that dose. For descriptive purposes, we categorised the outcome measure for each dose as either 'no delay', 'delay of between 1 and 6 months' or 'delay greater than 6 months'. Doses received 'too early' (greater than 30 days prior to when it was due) and doses never administered or recorded were excluded. Timeliness is measured in 12-month birth cohorts. Children included in the timeliness analysis were assessed at 1–3 years after doses were due, to allow time for late vaccinations to be recorded. Therefore, cohorts assessed for timeliness are not the same as those assessed for coverage milestones. The interval between doses was not evaluated. Timeliness of different vaccines and doses was also compared by plotting the cumulative percentage receiving each vaccine dose by age, with the proportion ever immunised set as 100%.

Remoteness status

The area of residence of children was defined as 'Major cities', 'Inner regional', 'Outer regional', 'Remote', and 'Very remote' using the Accessibility/Remoteness Index of Australia (ARIA),[16] which was developed by the National Centre for Social Applications of GIS (now the Australian Population and Migration Research Centre) as a joint project with the Australian Government Department of Health in 1997–1998. ARIA is an unambiguously geographical approach to defining remoteness. The most widely used ARIA product is ARIA+. ARIA+ is a continuous varying index with values ranging from 0 (high accessibility) to 15 (high remoteness), and is based on road distance measurements from over 12,000 populated localities to the nearest Service Centres in 5 categories based on population size. For the timeliness analysis, we combined the 2 'Regional' categories ('Inner Regional' and 'Outer Regional') into 1 category and the 2 'Remote' categories ('Remote' and 'Very Remote') into 1 category.

Indigenous status

Indigenous status on the ACIR is recorded as 'Indigenous', 'non-Indigenous' or 'unknown', as reported by the child's carer to Medicare or by the immunisation provider to the ACIR. For this report we considered 2 categories of children: 'Indigenous' and 'non-Indigenous'; children with unknown Indigenous status were presumed to be 'non-Indigenous'. Coverage estimate time trends are presented from 2002 only, due to poor rates of reporting Indigenous status prior to then.[17]

Small area analysis

Analysis for small areas was done by Australian Bureau of Statistics (ABS)-defined Statistical Area 3 (SA3) areas,[18] chosen because each is small enough to show differences within jurisdictions but not too small to render maps unreadable. Maps were created using version 12 of the MapInfo mapping software[19] and the ABS Census Boundary Information. As postcode is the only geographical indicator available from the ACIR, the ABS Postal Area to SA3 Concordance 2011 was used to match ACIR postcodes to SA3s, in order to create an SA3 field for each child in the relevant study cohort.[20]

Vaccination objection and incomplete immunisation

A child must be registered with Medicare before its parent(s) can lodge an objection to vaccination. Registered vaccination objectors are eligible for parent incentive payments even if their children are unvaccinated. However, some parents may object to vaccination but not lodge any registered objection to the ACIR. Such 'silent' vaccination objectors may have either some or no vaccines recorded on the ACIR. We calculated the proportions of children with vaccination objector status and no vaccines recorded on the ACIR, vaccination objector status and at least 1 vaccine recorded on the ACIR, no vaccination objector status and no vaccines recorded on the ACIR, and no vaccination objector status and not fully immunised by 24 months of age, from the cohort of children registered with Medicare and born between 1 January and 31 December 2011. Some of the children in the latter 2 groups may be 'silent objectors'. We chose this cohort when calculating proportions so that children under the age of 12 months were not included, to allow sufficient time for registration of objection and to exclude infants late for vaccination.

Human papillomavirus vaccine coverage

The National Human Papillomavirus (HPV) Vaccination Program is listed on the NIP Schedule, funded under the Immunise Australia Program, and delivered to females and, since 2013, males through an on-going school-based program usually in the 1st year of secondary school. From 2007 to 2009, there was a time-limited catch-up program delivered through schools, general practices and community immunisation services for females up to age 26. Males were offered a time-limited catch-up program in 2013–2014, at the age of 14–15 years. A full course of HPV immunisation was defined as 3 doses of quadrivalent HPV vaccine. Data on the National HPV Vaccination Program are provided by the National HPV Vaccination Program Register, which is operated by the Victorian Cytology Services Incorporated. Data for males were not available during preparation of this report. The purpose of this legislated register is to support the implementation of the vaccination program and to provide data for monitoring and evaluation. States and territories provide data to the HPV Register from their school-based programs. Doses administered in general practice or by community providers outside of the school program are notified on a voluntary basis, with a notification payment provided only to general practitioners (GPs) during the 2007–2009 catch-up program. The World Health Organization proposes using 15 years as the reference age for HPV vaccination coverage for the purposes of international comparison.

Coverage in the elderly

As there has not been an Adult Vaccination Survey[21] undertaken in Australia since 2009, no data are presented in this 2013 report on influenza or pneumococcal (23vPPV) vaccination coverage over the age of 65 years.

Results

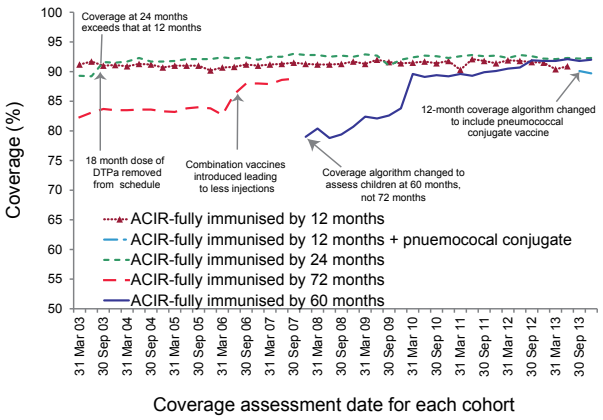
Coverage estimates

Overall

Coverage estimates in 2013 for full-year birth cohorts at the 3 milestone ages of 12 months, 24 months and 60 months are provided in Tables 2, 3 and 4. Nationally and for almost all jurisdictions, 'fully immunised' coverage and coverage for all individual vaccines (except rotavirus and varicella vaccines) for the 12-month, 24-month and 60-month age groups exceeded the target of 90%. However, coverage at 60 months of age in Western Australia was below this target for almost all vaccines and 'fully immunised'.

Figure 1 shows time trends in 'fully immunised' childhood vaccination coverage in Australia, assessed at 12 months, 24 months and 60 months of age, for 3-month cohorts born from 1 January 1999 to 31 December 2012. Coverage has been stable for the 12– and 24-month milestone age groups since late 2003. However, during 2013, coverage for the 12-month age group for vaccines due at 6 months of age declined by 1.8 percentage points. Half

Figure 1: Trends in 'fully immunised' vaccination coverage estimates, Australia, 2000 to 2013*



* By 3-month birth cohorts born between 1 January 1999 and 31 December 2012. Coverage assessment date was 12 months after the last birth date of each cohort.

Source: Australian Childhood Immunisation Register.

A list of vaccine abbreviations is contained in the glossary.

Table 2: Percentage of children in 2013 immunised by 12 months of age, by vaccine and state or territory*

Vaccine	State or territory								
	ACT	NSW	NT	Qld	SA	Tas.	Vic.	WA	Aust.
Total number of children	5,484	100,605	3,757	62,985	20,043	5,860	75,921	33,480	308,495
Diphtheria, tetanus, acellular pertussis	94.4	91.2	91.5	91.9	91.4	90.8	91.9	91.3	91.6
Poliomyelitis	94.3	91.1	91.5	91.9	91.3	90.7	91.8	91.2	91.5
Haemophilus influenzae type b	94.0	90.7	91.2	91.7	91.0	90.4	91.4	90.8	91.2
Hepatitis B	93.7	90.7	91.3	91.5	90.9	90.7	91.3	90.5	91.1
Fully immunised†	93.3	90.4	91.0	91.4	90.7	90.3	91.0	90.1	90.8
Rotavirus	88.2	86.4	86.1	81.2	83.3	86.1	82.4	78.3	83.6
Pneumococcal conjugate vaccine	93.7	90.6	91.1	91.4	90.8	90.3	91.1	90.4	90.9
Fully immunised† + pneumococcal conjugate vaccine	93.0	89.9	90.5	91.1	90.3	89.6	90.5	89.7	90.3

* Cohort born in 2012.

† 'Fully immunised' – 3 doses of a DTPa-containing vaccine, 3 doses of polio vaccine, 2 or 3 doses of PRP-OMP-containing Hib vaccine or 3 doses of any other Hib vaccine, and 3 doses of any hepatitis B vaccine or 2 doses of either Engerix-B (paediatric), Comvax or H-B-VAX II (paediatric). From July 2013, the 3rd dose of pneumococcal conjugate vaccine was included in the coverage algorithm for the 12-month milestone.

Source: Australian Childhood Immunisation Register.

A list of vaccine abbreviations is contained in the glossary.

Table 3: Percentage of children in 2013 immunised by 24 months of age, by vaccine and state or territory*

Vaccine	State or territory								
	ACT	NSW	NT	Qld	SA	Tas.	Vic.	WA	Aust.
Total number of children	5,473	99,099	3,687	62,498	19,847	6,219	73,885	33,415	304,123
Diphtheria, tetanus, acellular pertussis	95.1	94.6	95.3	94.9	94.7	95.8	95.2	93.6	94.8
Poliomyelitis	95.1	94.6	95.4	94.8	94.7	95.8	95.2	93.6	94.7
Haemophilus influenzae type b	95.2	94.8	95.8	94.7	94.5	95.7	95.1	93.5	94.7
Hepatitis B	94.5	94.0	95.1	94.4	94.2	95.4	94.6	95.4	94.1
Measles, mumps, rubella†§	94.4	93.7	94.9	94.2	93.9	94.6	94.1	92.7	93.9
Fully immunised‡	92.8	91.8	93.5	92.8	92.1	93.4	92.4	90.4	92.1
Varicella§	88.4	84.4	86.9	87.4	83.4	84.2	84.2	82.5	84.8
Meningococcal C	93.6	93.0	94.4	93.7	93.4	94.2	93.4	91.9	93.2

* Cohort born in 2011.

† A combination MMRV vaccine for children aged 18 months was added to the National Immunisation Program. The MMRV vaccine replaces the separate MMR vaccine previously given at 4 years, and the varicella vaccine (for chickenpox) previously given at 18-months.

‡ Fully immunised: 3 or 4 doses of a DTPa-containing vaccine, 3 doses of polio vaccine, 3 or 4 doses of PRP-OMP-containing Hib vaccine or 4 doses of any other Hib vaccine, 3 or 4 doses of Engerix-B (paediatric), Comvax or H-B-VAX II (paediatric) hepatitis B vaccine or 4 doses of all other hepatitis B vaccines, and 1 dose of an MMR-containing vaccine.

§ MMRV coverage for children born July to December 2011 not shown due to data system issues.

Source: Australian Childhood Immunisation Register.

A list of vaccine abbreviations is contained in the glossary.

Table 4: Percentage of children in 2013 immunised by 60 months of age, by vaccine and state or territory*

Vaccine	State or territory								
	ACT	NSW	NT	Qld	SA	Tas.	Vic.	WA	Aust.
Total number of children	5,211	100,460	3,514	65,215	20,346	6,556	74,384	33,834	309,970
Diphtheria, tetanus, acellular pertussis	92.6	92.5	91.4	92.3	91.5	93.4	92.9	89.9	92.2
Poliomyelitis	92.6	92.5	91.4	92.3	91.5	93.3	92.9	90.0	92.2
Measles, mumps, rubella	92.3	92.4	91.7	92.3	91.5	93.7	92.8	89.8	92.2
Fully immunised †	92.3	91.2	90.5	91.4	90.2	92.2	92.4	88.9	91.2

* Cohort born in 2008.

† Fully immunised: 4 or 5 doses of a DTPa-containing vaccine, 4 doses of polio vaccine, and 2 doses of an MMR-containing vaccine.

Source: Australian Childhood Immunisation Register.

A list of vaccine abbreviations is contained in the glossary.

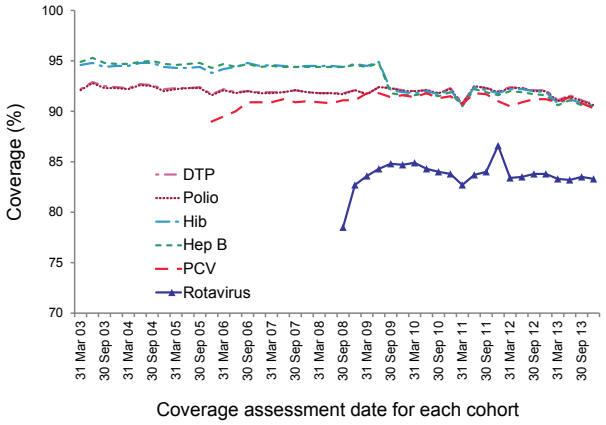
a percentage point of this decrease is due to the recent inclusion of 13-valent pneumococcal conjugate vaccine in the coverage calculation algorithm for 'fully immunised' at 12 months of age, as seen by comparison of coverage calculated according to the new and old algorithms (Table 2). For vaccines due at 48 months of age, 'fully immunised' coverage dropped to 80.4% in January 2008, following the change in assessment age from 72 months to 60 months. However, 'fully immunised' coverage for the 60-month age group then rose substantially in 2009 and 2010 and kept increasing throughout 2011–2013 to a level comparable with that in the 24-month age group (92.0%).

Individual vaccines

Coverage at 12 months of age for individual antigens in the relevant combination vaccine (DTPa-hepB-IPV-Hib) also decreased in 2013 (Figure 2). Coverage for 3 doses of pneumococcal conjugate vaccine (PCV) by 12 months of age rose slightly from 89.0% in late 2005 to 90.3% in late 2013, just below that for all other vaccines due at this age, except for rotavirus vaccine. Rotavirus vaccine coverage rose steeply from late 2008 from 78.5% to 83.4% in late 2011 and then decreased from early 2012 to 83.4%. Rotavirus coverage remained at below 84.0% during 2013.

At 24 months of age, hepatitis B coverage was higher than for all other vaccines, at just under 95.0%, until the change in the coverage algorithm in late 2009 (Figure 3). In previous years, coverage has been lowest for MMR and Hib vaccines, the only vaccines that have a 12-month dose used in calculations, but in 2013 coverage was similar for all vaccines (except varicella) at just below 95.0%.

Figure 2: Trends in vaccination coverage estimates for individual vaccines* at 12 months of age, Australia, 2000 to 2013



By 3-month birth cohorts born between 1 January 1999 and 31 December 2012. Coverage assessment date was 12 months after the last birth date of each cohort.

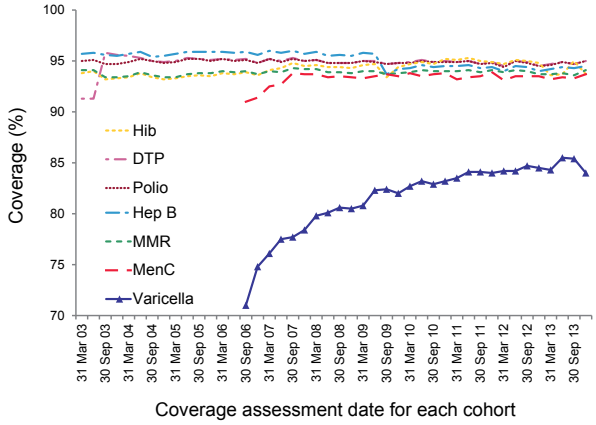
* 3rd dose of DTP, polio and 7-valent pneumococcal, 2nd or 3rd dose of Hib, hepatitis B and rotavirus.

Source: Australian Childhood Immunisation Register.

A list of vaccine abbreviations is contained in the glossary.

For vaccines due at 48 months of age, trends in individual vaccine coverage were similar to that seen for 'fully immunised coverage' i.e. a marked drop in January 2008 following the change in assessment age from 72 months to 60 months, followed by a marked increase in 2009 and 2010 and on-going increase to a level higher than when coverage was assessed at 72 months of age (Figure 4). Coverage for both vaccines due at 48 months (DTPa and MMR) was greater than 92.0% in 2013.

Figure 3: Trends in vaccination coverage estimates for individual vaccines* at 24 months of age, Australia, 2000 to 2013



By 3-month birth cohorts born between 1 January 1998 and 31 December 2011. Coverage assessment date was 24 months after the last birth date of each cohort.

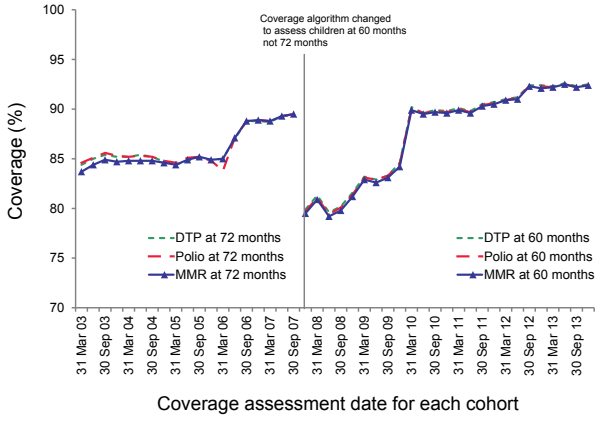
MMRV coverage for children born July to December 2011 not shown due to data system issues.

* 3rd or 4th dose of DTP, 3rd dose of polio, 3rd or 4th dose of Hib, 2nd or 3rd dose of hepatitis B, 1 dose of MMR, meningococcal C conjugate and varicella.

Source: Australian Childhood Immunisation Register.

A list of vaccine abbreviations is contained in the glossary.

Figure 4: Trends in vaccination coverage estimates for individual vaccines* at 60 months of age prior to December 2007), Australia, 2002 to 2013



By 3-month birth cohorts born between 1 January 1996 and 31 December 2008. Coverage assessment date was 72 months after the last birth date of each cohort up to December 2007 and then 60 months after the last birth date of each cohort.

* 4th dose of DTP and polio, 2nd dose of MMR.

Source: Australian Childhood Immunisation Register.

A list of vaccine abbreviations is contained in the glossary.

Coverage estimates for Indigenous children

Immunisation coverage in 2013 continued to be lower for Indigenous children than for non-Indigenous children in the 12 months age group. Coverage was similar for Indigenous children at 24 months of age and higher at 60 months of age (Tables 5 and 6). The coverage differential between Indigenous and non-Indigenous children for individual vaccines varied, with coverage lower for Indigenous children for all vaccines at 12 months of age, but higher at 24 months of age for hepatitis B, Hib, MMR and meningococcal C conjugate vaccines, and at 60 months of age for DTP, polio, and MMR.

Table 5: Vaccination coverage estimates percentages, Australia, 2013, by age, vaccine and Indigenous status

Vaccine	Milestone age	Indigenous	Non-Indigenous
DTPa	12 months*	86.5	91.9
	24 months†	94.3	94.8
	60 months‡	93.4	92.2
Polio	12 months*	86.4	91.8
	24 months†	94.3	94.7
	60 months‡	93.3	92.2
Hib	12 months*	86.3	91.4
	24 months†	94.8	94.7
	60 months‡	N/I	N/I
Hep B	12 months*	86.4	91.3
	24 months†	94.3	93.8
	60 months‡	N/I	N/I
MMR	12 months*	N/I	N/I
	24 months†	94.3	93.9
	60 months‡	93.7	92.1
Varicella	12 months*	N/I	N/I
	24 months†	82.4	84.9
	60 months‡	N/I	N/I
MenC	12 months*	N/I	N/I
	24 months†	93.7	93.2
	60 months‡	N/I	N/I
PCV	12 months*	86.4	91.2
	24 months†	N/I	N/I
	60 months‡	N/I	N/I
Rotavirus	12 months*	72.2	83.9
	24 months†	N/I	N/I
	60 months‡	N/I	N/I

* Cohort born 1 January 2012 to 31 December 2012.

† Cohort born 1 January 2011 to 31 December 2011.

‡ Cohort born 1 January 2008 to 31 December 2008.

N/I Not included in coverage estimates for that group.

Source: Australian Childhood Immunisation Register.

A list of vaccine abbreviations is contained in the glossary.

Table 6: Percentage of children fully immunised by 12 months, 24 months and 60 months of age, Australia, 2013, by Indigenous status and state or territory

	State or territory								
	ACT	NSW	NT	Qld	SA	Tas.	Vic.	WA	Aust.
12 months – fully immunised*									
Indigenous	91.7	88.1	90.1	85.7	79.5	87.8	86.6	82.7	86.3
Non-Indigenous	93.4	90.5	91.5	91.9	91.1	90.5	91.0	90.6	91.0
12 months – fully immunised (including pneumococcal conjugate vaccine)									
Indigenous	90.8	88.0	89.7	85.5	79.4	87.3	86.3	82.4	86.0
Non-Indigenous	93.0	90.0	91.0	91.5	90.7	89.7	90.5	90.1	90.6
24 months – fully immunised†									
Indigenous	93.2	91.6	95.9	93.0	85.8	93.7	90.6	90.4	92.0
Non-Indigenous	92.8	91.8	92.0	92.8	92.3	93.4	92.5	90.4	92.1
60 months – fully immunised‡									
Indigenous	95.1	93.8	95.3	93.9	87.4	94.2	91.5	90.3	93.0
Non-Indigenous	91.7	91.9	88.4	91.7	91.2	92.7	92.4	89.2	91.6

* 'Fully immunised' – 3 doses of a DTPa-containing vaccine, 3 doses of polio vaccine, 2 or 3 doses of PRP-OMP-containing Hib vaccine or 3 doses of any other Hib vaccine, and 2 or 3 doses of Comvax hepatitis B vaccine or 3 doses of all other hepatitis B vaccines.

† 'Fully immunised' – 3 or 4 doses of a DTPa-containing vaccine, 3 doses of polio vaccine, 3 or 4 doses of PRP-OMP-containing Hib vaccine or 4 doses of any other Hib vaccine, 3 or 4 doses of Engerix-B (paediatric), Comvax or H-B-VAX II (paediatric) or 4 doses of all other hepatitis B vaccines, and 1 dose of an MMR-containing vaccine.

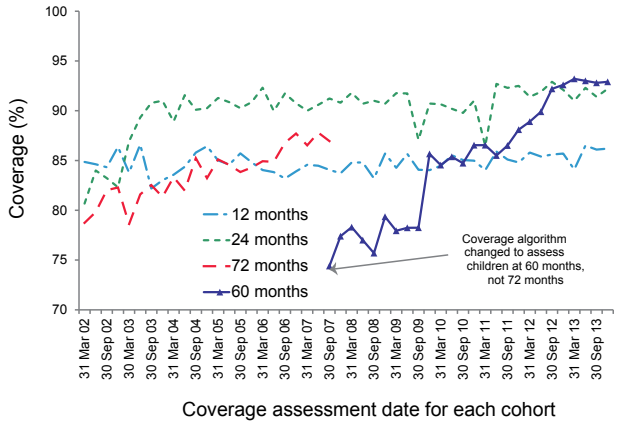
‡ 'Fully immunised' – 4 or 5 doses of a DTPa-containing vaccine, 4 doses of polio vaccine, and 2 doses of an MMR-containing vaccine.

Source: Australian Childhood Immunisation Register.

A list of vaccine abbreviations is contained in the glossary.

The proportion of Indigenous children 'fully immunised' by 24 months of age was consistently higher than at 12 and 60 months of age until 2012, when coverage at 60 months rose to levels comparable with those at 24 months (Figure 5).

Figure 5: Trends in 'fully immunised' vaccination coverage estimates for Indigenous children in Australia, 2002 to 2013, by age cohorts



Source: Australian Childhood Immunisation Register.

Coverage at 12 months of age was lower among Indigenous versus non-Indigenous children in all jurisdictions. Differentials reached 11.6 and 8 percentage points in South Australia and Western Australia, respectively, but in the Northern Territory, with the highest proportion of Indigenous children, the differential was only 1.4 percentage points (Table 6). At the age of 24 months, the coverage in Indigenous compared with non-Indigenous children varied from 3.9 percentage points higher in the Northern Territory to 6.5 percentage points lower in South Australia (Table 6).

At 60 months of age, coverage at the national level was 1.4 percentage points higher in Indigenous versus non-Indigenous children but there was variation between individual jurisdictions, ranging from coverage of 6.9 percentage points higher in the Northern Territory to 3.8 percentage points lower for Indigenous children in South Australia (Table 6).

Coverage for other National Immunisation Program vaccines not routinely reported in quarterly coverage reports

Pneumococcal conjugate vaccine and rotavirus

The 7vPCV was first added to the NIP in January 2005 and was replaced in July 2011 by 13vPCV for all Australian children at 2, 4 and 6 months of age. Since coverage was first calculated for this vaccine in late 2006, it has remained high, although slightly lower than for DTPa, polio, Hib and hepatitis B vaccines. Coverage has increased slightly from 89.0% in 2006 to 90.3% in late 2013 (Figure 2) and is greater than the 1993 Immunise Australia Program target of 90% in all jurisdictions (Table 2). In July 2013, the 3rd dose of pneumococcal conjugate vaccine was included in the coverage algorithm for the 12-month milestone.

Rotavirus vaccine was added to the NIP in July 2007, so coverage for 2 or 3 doses (depending on vaccine brand) at 12 months of age could be calculated only from the December 2008 quarter onwards. Rotavirus coverage was lower nationally (Figure 2) and had greater variation between jurisdictions than other vaccines given at 2, 4 and 6 months of age. Reported coverage in 2013 at 12 months of age varied from 88.2% and 86.4% in the Australian Capital Territory and New South Wales, respectively, for 2 doses of Rotarix® vaccine, to 78.3% in Western Australia for 3 doses of RotaTeq® vaccine (Table 2).

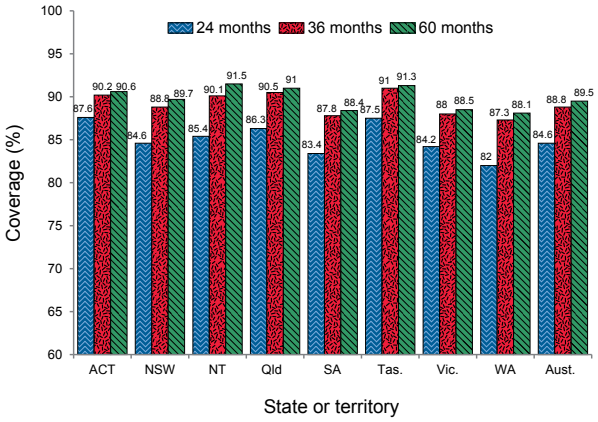
Meningococcal C and varicella

Meningococcal C conjugate vaccine was added to the NIP in January 2003. Since coverage was first calculated for this vaccine in mid-2006, it has remained at high levels, increasing from 91.0% in late 2006 to 93.7% in late 2013 (Figure 3). There was little variation in 2013 by jurisdiction with all jurisdictions experiencing coverage levels greater than 91% and the Northern Territory and Tasmania approaching 95% (Table 3).

Varicella vaccine was added to the NIP in November 2005. Reported coverage for this vaccine has consistently been 10 to 15 percentage points lower than for all the other vaccines assessed at the 24-month milestone, being 84.0% for the latest assessment in 2013 (Figure 3). Reported varicella vaccine coverage in 2013 also showed considerable variation by jurisdiction from 82.5% in Western Australia to 88.4% in the Australian Capital Territory (Table 3).

As the 18-month schedule point was historically associated with lower coverage, as there is only a 6-month time period to catch-up for varicella vaccination, we compared varicella coverage assessed at 36 months and 60 months to that assessed at 24 months by jurisdiction (Figure 6). Coverage by jurisdiction varied between 2.6 and 5.3 percentage points higher at 36 months and 3.0 to 6.1 percentage points higher at 60 months, with 4 jurisdictions reaching over 90.0% varicella coverage when assessed at 60 months.

Figure 6: Comparison of 1-dose varicella vaccine coverage assessed in 2013 at 24 months of age versus 36 months of age and 60 months of age, by state or territory



Cohort born October to December 2010.

Source: Australian Childhood Immunisation Register.

A comparison of varicella coverage before and after introduction of measles-mumps-rubella-varicella vaccine

In July 2013, the combination varicella and MMR vaccine (MMRV) was recommended and funded for the single dose of varicella vaccine scheduled at 18 months of age, at the same time lowering the age of administration of the 2nd dose of a measles-containing vaccine from 48 to 18 months of age.

Table 7 provides varicella coverage for two 3-month wide birth cohorts before and after introduction of MMRV vaccine. For Australia as a whole, varicella coverage increased by 3.5 percentage points from pre– to post-introduction of MMRV. Increases occurred in all jurisdictions except the Northern Territory, ranging from a 0.7 percentage point increase in Tasmania to a 5.1 percentage point increase in South Australia.

Data are also available from the ACIR on the number of reports from GPs stating that children, born since May 2004, have natural immunity to varicella and do not require varicella vaccination. Reports of natural immunity to varicella total

Table 7: Comparison of varicella coverage (%) before and after introduction of measles-mumps-rubella-varicella vaccine, by state or territory

	State or territory								
	ACT	NSW	NT	Qld.	SA	Tas.	Vic.	WA	Aust.
Before MMRV* introduction	87.2	85.2	89.2	87.9	83.7	87.1	84.9	83.9	85.6
After MMRV† introduction	92.0	89.1	88.4	90.2	88.8	87.8	89.1	87.0	89.1

* Cohort born 1 April to 30 June 2011, assessed at 24 months.

† Cohort born 1 April to 30 June 2012, assessed at 24 months.

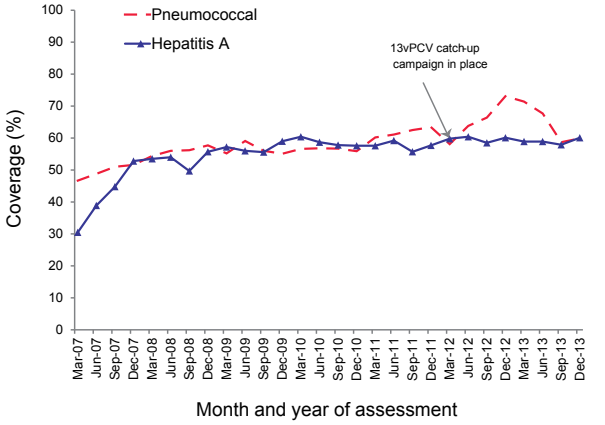
Source: Australian Childhood Immunisation Register.

greater than 30,533 since May 2004 (not shown), corresponding to approximately 1.1% of the entire cohort, a percentage that has remained stable since 2004. It is likely that there is some under-reporting of presumed natural immunity by GPs who consequently recommend that the varicella vaccine is not needed.

Pneumococcal booster and hepatitis A for Indigenous children in some jurisdictions

Hepatitis A vaccine was available in Australia prior to the development of the ACIR in 1996. It has been included on the NIP since November 2005 for Indigenous children in the Northern Territory, Queensland, South Australia and Western Australia, but was used earlier than this in North Queensland. Since March 2007, coverage of 2 doses of hepatitis A vaccine for Indigenous children by 30 months of age in Western Australia and the Northern Territory and 36 months of age in Queensland and South Australia has increased from 30.5% in early 2007 to 60.1% in December 2013 (Figure 7). An additional 10.4% of children had received 1 dose of hepatitis A vaccine by 18 or 24 months of age, putting national coverage for at least 1 dose of hepatitis A vaccine for 2013 at 70.5% in Indigenous children compared with 60.1% for 2 doses (Table 8). There was variation in reported hepatitis A vaccine coverage by jurisdiction, from a low of 42.2% in South Australia to a high of 85.9% in the Northern Territory (Table 8).

Figure 7: Trends in coverage estimates for hepatitis A* and pneumococcal† vaccines for Indigenous children, Australia,‡ 2007 to 2013



* 2 doses by 30 months of age for the Northern Territory and Western Australia (1 dose by 18 months of age), 2 doses by 36 months of age for Queensland and South Australia (1 dose by 24 months of age). In July 2013, the hepatitis A vaccination schedule for Indigenous children changed so that now dose 1 is administered to Indigenous children at 12 months of age and dose 2 at 18 months of age in all 4 jurisdictions. However, hepatitis A coverage data presented in this report uses the old calculation with assessment by 30 or 36 months of age as the new assessment ages apply only to half the year.

† 18-month dose assessed at 30 months of age.

‡ Northern Territory, Queensland, South Australia and Western Australia only.

Source: Australian Childhood Immunisation Register.

A pneumococcal booster dose at 18 to 24 months of age has been recommended and funded for Indigenous children in 4 jurisdictions (the Northern Territory, Queensland, South Australia and Western Australia) since 2001; firstly as 23-valent pneumococcal polysaccharide vaccine then as 13-valent pneumococcal conjugate vaccine, from July 2013 in Queensland, South Australia and Western Australia, and from October 2013 in the Northern Territory. Coverage gradually increased from 46.7% in March 2007 to 59.9% in December 2013 (Figure 7). From 2011 to 2012, coverage increased by almost 10 percentage points to 73.1%, due mainly to the 13vPCV catch-up campaign that took place in 2012. There was a large variation in coverage for a 4th dose of pneumococcal vaccine by jurisdiction, from a low of 29.5% in South Australia to a high of 86.2% in the Northern Territory (Table 8).

Hepatitis B birth dose

Hepatitis B birth dose coverage is not included in this report due to substantial under-reporting. Most doses are given in maternity hospitals, many of which lack mechanisms for reporting

Table 8: Vaccination coverage* percentage for hepatitis A and pneumococcal for Indigenous children, Australia,† 2013, by state or territory

State or territory	Hepatitis A‡		Pneumococcal§	13vPCV‖
	2 doses	1 dose		
Northern Territory	85.9	87.5	86.2	85.9
Queensland	57.1	66.7	57.7	56.3
South Australia	42.2	54.1	29.5	21.4
Western Australia	67.0	71.1	59.9	54.8
Australia†	60.1	70.5	59.9	57.3

* For the last 3-month cohorts assessable in 2013.

† Northern Territory, Queensland, South Australia and Western Australia only.

‡ 2 doses by 30 months of age for the Northern Territory and Western Australia (1 dose by 18 months of age), 2 doses by 36 months of age for Queensland and South Australia (1 dose by 24 months of age). In July 2013, the hepatitis A vaccination schedule for Indigenous children changed so that now dose 1 is administered to Indigenous children at 12 months of age and dose 2 at 18 months of age in all 4 jurisdictions. However, hepatitis A coverage data presented in this report uses the old calculation with assessment by 30 or 36 months of age as the new assessment ages do not apply yet.

§ 1 dose of 13vPCV (4th dose) or 23vPPV (1st dose) or 10vPCV (4th dose in the Northern Territory) by 30 months of age.

‖ 1 dose of 13vPCV (4th dose) by 30 months of age.

Source: Australian Childhood Immunisation Register.

A list of vaccine abbreviations is contained in the glossary.

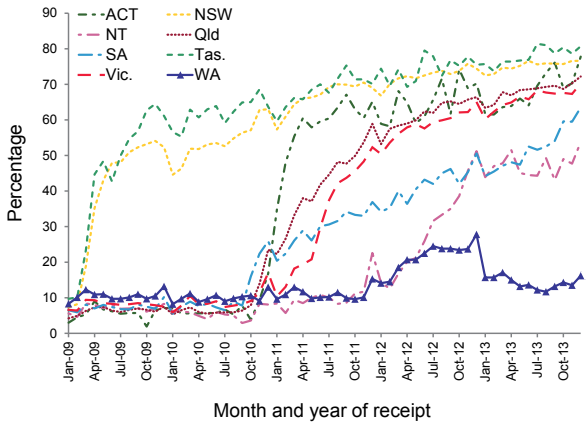
to the ACIR. A 2006 unpublished report found a minimum estimate for coverage of the birth dose of hepatitis B vaccine of 85%, based on provider-completed parent-held written records.[22]

Recommendations on 1st and 4th dose of diphtheria-tetanus-acellular pertussis (child formulation)

In response to a pertussis epidemic and to provide early protection to young infants, in March 2009 ATAGI recommended that immunisation providers give the 1st dose of DTPa vaccine at 6 weeks of age instead of 8 weeks (2 months) of age. This was promoted in that year during epidemics in New South Wales and Tasmania (and later in other jurisdictions). Prior to this, very few children received the vaccine dose at less than 8 weeks of age but over the next 4 years the percentage rose and in the Australian Capital Territory, New South Wales, Queensland, Tasmania and Victoria more than 70.0% of children were receiving the dose prior to 8 weeks of age by December 2013 (Figure 8). By late 2013, this percentage was greater than 50.0% in all jurisdictions except Western Australia.

ATAGI also recommended in October 2009 that the pre-school booster dose of DTPa-IPV could be given from 3.5 rather than 4 years of age. Take-up of this recommendation was slower with no jurisdiction giving the vaccine in any great numbers at 3.5 to 4 years of age until November 2010 (Figure 9). As at December 2013, more than 35.0% of children in 3 jurisdictions (the Australian Capital Territory, the Northern Territory and South Australia) were receiving the dose at 3.5 to 4 years of age (Figure 9).

Figure 8: Percentage of children who received the 1st dose of DTP/Hexa vaccine at age 6 to <8 weeks, by state or territory and month of receipt



* DTP/Hexa = Combined DTPa-IPV-Hib-Hep B vaccine.

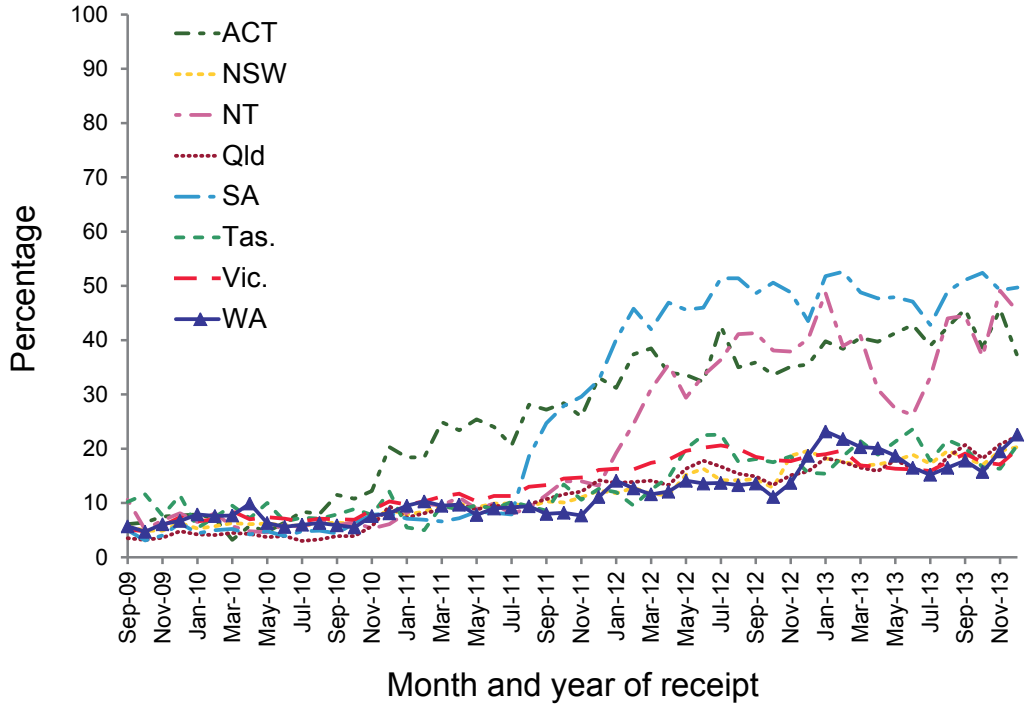
A list of vaccine abbreviations is contained in the glossary.

Source: Australian Childhood Immunisation Register.

Timeliness of immunisation

We examined the timeliness of immunisation in 2013, for both vaccines requiring multiple doses (DTP and MMR) and a single dose (MenCCV) at 12, 24 and 60 months of age.

Figure 9: Percentage of children who received the 4th dose of DTPa-IPV vaccine at age 3.5 years to <4 years, by state or territory and month of receipt

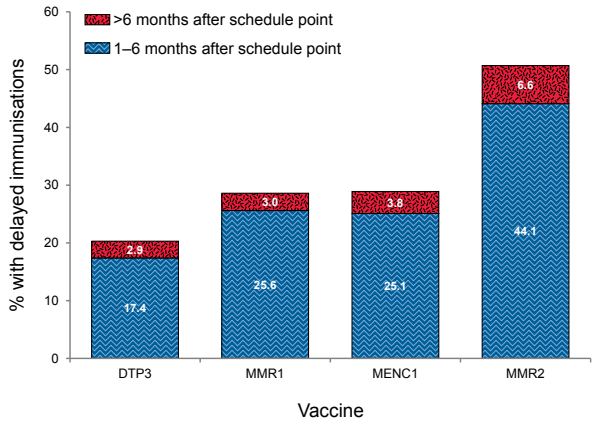


A list of vaccine abbreviations is contained in the glossary.

Source: Australian Childhood Immunisation Register.

As demonstrated in previous reports, the proportion with vaccination delay increased with older age (Figure 10). The greatest proportion with any delay was seen with the 2nd dose of MMR vaccine, with 50.7% of doses given late and 6.6% given more than 6 months late. These figures are an improvement from the 2012 report, which were 57.0% and 8.9%, respectively.

Figure 10: Vaccination delay for cohorts born in 2011 (DTP3, MMR1, MENC1) and 2007 (MMR2), Australia



DTP3 3rd dose of a diphtheria-tetanus-acellular pertussis-containing vaccine.

MMR1 1st dose of a measles-mumps-rubella vaccine.

MENC1 1st dose of a meningococcal C conjugate vaccine.

MMR2 2nd dose of a measles-mumps-rubella vaccine.

Source: Australian Childhood Immunisation Register.

For the 3rd dose of DTPa vaccine, there was greater delay for Indigenous children than for non-Indigenous children, with a 21.6% differential in on-time vaccination at less than 7 months of age (Figure 11). The same pattern was found for timeliness of the 1st dose of MMR vaccine, but with a smaller differential of 12.9% (Figure 12). Although Indigenous children had similar coverage to non-Indigenous children by 24 months of age, they were more likely to have delayed vaccination. This differential in on-time vaccination between Indigenous and non-Indigenous children remained high (the corresponding differentials for the 3rd dose of DTPa and 1st dose of MMR from the 2012 report were 21.5% and 13%, respectively). In contrast to the 3rd dose of DTPa and the 1st dose of MMR, analysis of timeliness of immunisation for a vaccine due at 48 months of age, the 2nd dose of MMR, showed a much smaller differential in delayed receipt of this vaccine between non-Indigenous children and Indigenous children of 5.8% at <49 months of age (Figure 13).

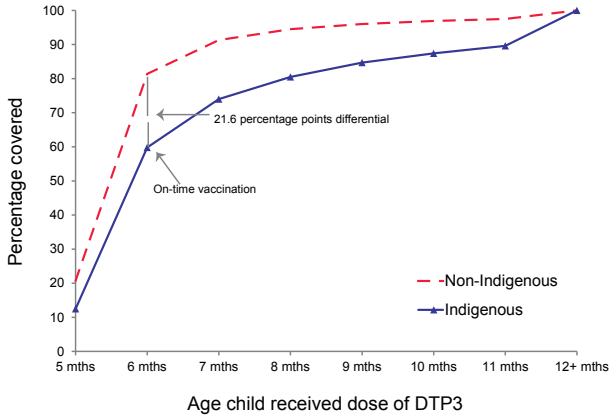
Delayed receipt of the 3rd dose of DTPa and the 1st dose of MMR by more than 1 month was found in 28.0% to 37.2% of Indigenous children and 16.5% to 26.3% of non-Indigenous children, depending

on remoteness status (Table 9). Vaccination delay was greater for Indigenous children than for non-Indigenous children for both vaccines across all categories (major cities, inner/outer regional and remote/very remote areas).

Vaccination delay for Indigenous children by jurisdiction was measured for the 3rd dose of PCV, with greater proportions experiencing delays of 1 to 6 months in Western Australia (37.4%) and South Australia (35.6%) (Figure 14). This is an increase from 2012 where the corresponding figures were 35.7% and 32.0%, respectively. The proportion of Indigenous children with long delays (>6 months) also increased from 2012 in South Australia (from 9.8% to 12.1%) and Western Australia (from 11.1% to 13.8%).

Trends in timeliness of the 3rd dose of pneumococcal vaccine and the 1st dose of MMR vaccine by Indigenous status are provided in Figures 15

Figure 11: Timeliness* of the 3rd dose of diphtheria-tetanus-acellular pertussis vaccine, Australia, by Indigenous status

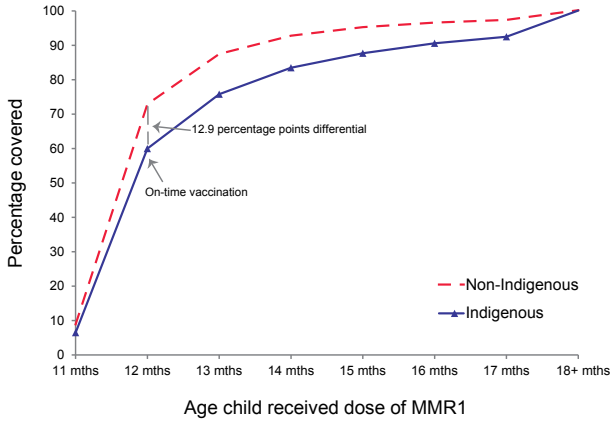


* Percentage covered = number of children who received vaccine dose at particular ages / the total number of children who received the vaccine dose, expressed as a percentage.

Cohort born in 2011.

Source: Australian Childhood Immunisation Register.

Figure 12: Timeliness* of the 1st dose of measles-mumps-rubella vaccine, Australia, by Indigenous status

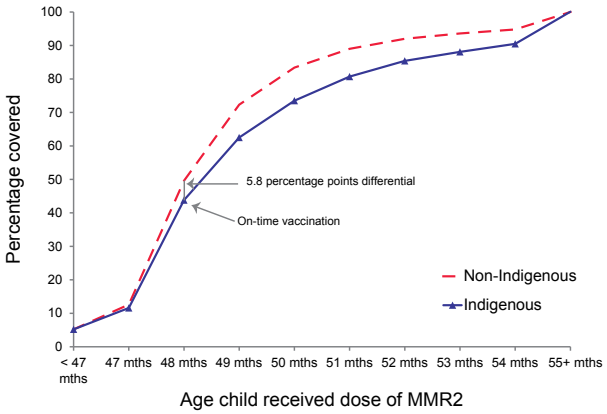


* Percentage covered = number of children who received vaccine dose at particular ages / the total number of children who received the vaccine dose, expressed as a percentage.

Cohort born in 2011.

Source: Australian Childhood Immunisation Register.

Figure 13: Timeliness* of the 2nd dose of measles-mumps-rubella vaccine, Australia, by Indigenous status

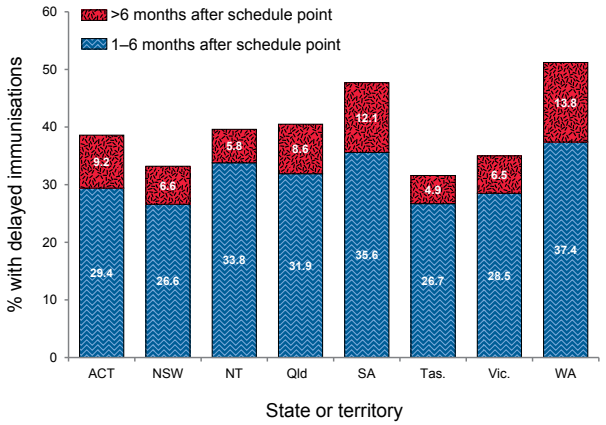


* Percentage covered = number of children who received vaccine dose at particular ages / the total number of children who received the vaccine dose, expressed as a percentage.

Cohort born in 2007.

Source: Australian Childhood Immunisation Register.

Figure 14: Vaccination delay for Indigenous children for the 3rd dose of pneumococcal conjugate vaccine, Australia, 2013, by state or territory



Cohort born in 2011.

Source: Australian Childhood Immunisation Register.

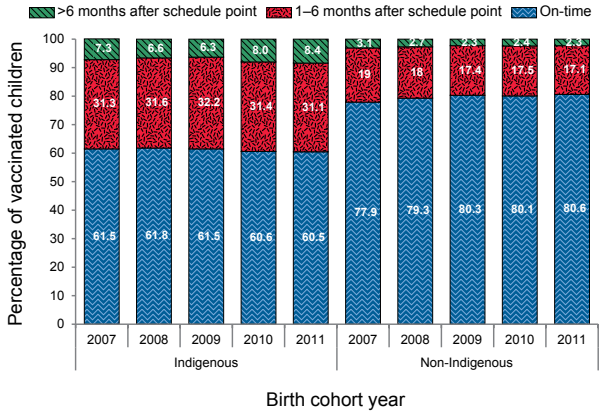
Table 9: Vaccination delay, for children aged 2 years, Australia, 2013, by Indigenous and remoteness status

Vaccine dose Indigenous status	Remoteness	1–6 months after schedule point %	>6 months after schedule point %
Diphtheria-tetanus-acellular pertussis dose 3			
Indigenous	Major cities	28.0	10.2
	Inner and Outer regional	29.4	10.9
	Remote and Very remote	37.2	9.8
Non-Indigenous	Major cities	16.5	2.4
	Inner and Outer regional	17.3	2.9
	Remote and Very remote	17.0	2.5
Measles-mumps-rubella dose 1			
Indigenous	Major cities	32.7	7.6
	Inner and Outer regional	34.1	7.8
	Remote and Very remote	32.8	6.5
Non-Indigenous	Major cities	25.3	2.8
	Inner and Outer regional	25.0	2.7
	Remote and Very remote	26.3	2.3

Source: Australian Childhood Immunisation Register.

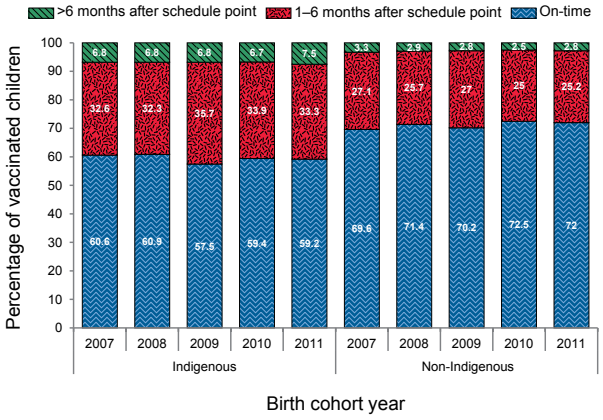
and 16. Timeliness for the 3rd dose of pneumococcal vaccine improved marginally over time for non-Indigenous children, from 77.9% in the 2007 birth cohort to 80.6% in the 2011 birth cohort; however, no improvements were seen for Indigenous children (Figure 15). Timeliness for the 1st dose of MMR vaccine improved marginally over time for non-Indigenous children, from 69.6% in the 2007 birth cohort to 72.0% in the 2011 birth cohort. In contrast, timeliness decreased marginally over time for Indigenous children, from 60.6% in the 2007 birth cohort to 59.2% in the 2011 birth cohort (Figure 16).

Figure 15: Timeliness of the 3rd dose of pneumococcal vaccine, Australia, 2007 to 2011, by Indigenous status and year of birth



Source: Australian Childhood Immunisation Register.

Figure 16: Timeliness of the 1st dose of measles-mumps-rubella vaccine, Australia, 2007 to 2011, by Indigenous status and year of birth



Source: Australian Childhood Immunisation Register.

Vaccination objection and incomplete immunisation

The percentage of registered vaccination objectors, with or without vaccines recorded on the ACIR, and children not registered as objectors but with either no vaccines recorded or some vaccines but not fully immunised, for all jurisdictions and Australia is shown in Table 10. Of the 4 groups, the largest is those parents who are not registered as objecting and whose children are not fully immunised by 24 months of age. Some of these children, and some of those children not registered as objectors but with no vaccines recorded, may be 'silent objectors'.

The rate of registered objection in 2013 for Australia was 1.9% and this varied by jurisdiction with a high of 2.5% in Queensland and a low of 1.0% in the Northern Territory.

The proportion of children whose parents are registered as objecting to vaccination are presented by Statistical Area 3 (SA3) in Figure 17. The map shows pockets of high levels of registered objection within jurisdictions in 2013, particularly in coastal areas of northern and south-east Queensland, northern New South Wales, inner Melbourne and Fremantle, with rates of objection reaching over 6.0% in many areas. These areas have had consistently high levels of registered objection over many years.

Partially immunised children

The percentage of 'partially immunised' children (excluding those whose parents have registered as vaccination objectors) who are up-to-date for specific vaccines due by 24 months of age is shown in Table 11. The vaccine that 'partially immu-

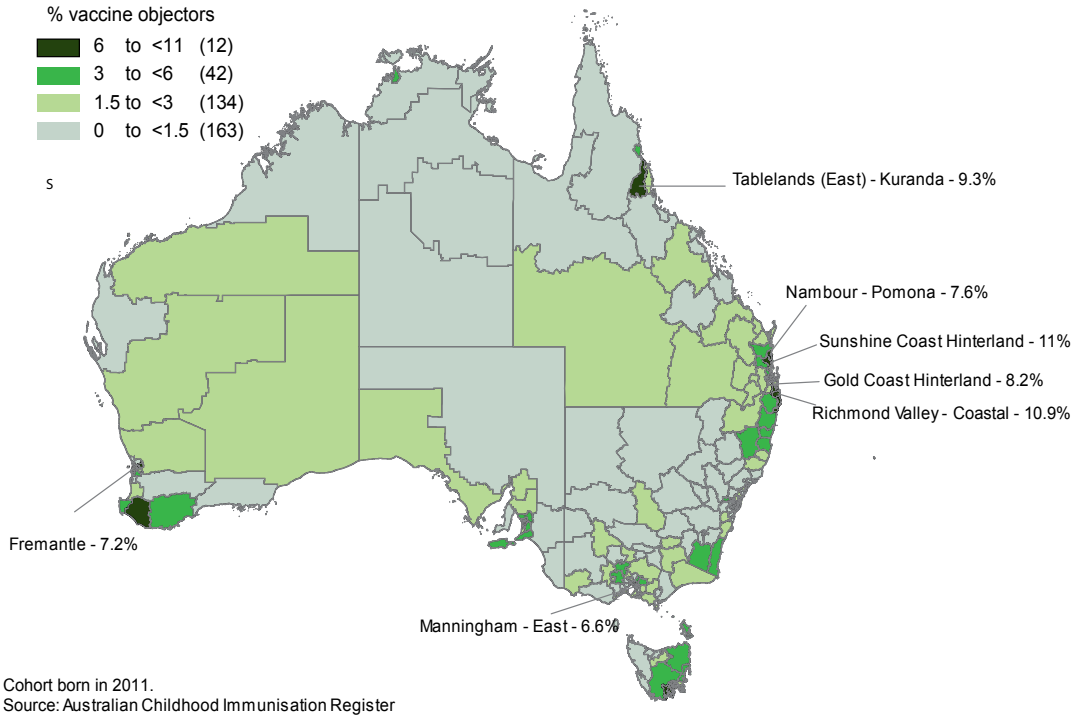
Table 10: Percentage of children aged 2 years with no or some vaccines recorded on the Australian Childhood Immunisation Register, Australia, 2013, by whether registered vaccination objection and state or territory

	State or territory								
	ACT	NSW	NT	Qld	SA	Tas.	Vic.	WA	Aust.
Total number of children	5,473	99,099	3,687	62,498	19,847	6,219	73,885	33,415	304,123
Object* and no vaccines recorded	0.9	1.0	0.5	1.6	1.3	1.1	1.0	1.2	1.2
Object* and at least 1 vaccine recorded	0.6	0.6	0.5	0.9	0.9	0.5	0.6	0.9	0.7
No objection and no vaccines recorded	2.2	2.0	1.9	1.8	1.7	1.2	1.9	2.6	2.0
No objection and partially immunised by 24 months of age	3.8	4.8	3.8	3.3	4.3	3.8	4.2	5.2	4.3

* Recorded on the Australian Childhood Immunisation Register as parent having lodged an 'Immunisation exemption conscientious objection' form to the Australian Government Department of Human Services.

Source: Australian Childhood Immunisation Register.

Figure 17: Proportion of vaccination objectors, Australia, 2013, by Australian Bureau of Statistics Statistical Area 3



Cohort born in 2011.
Source: Australian Childhood Immunisation Register

nised' children were most commonly missing by 24 months of age was the 1st dose of MMR, with only 38.1% up-to-date nationally, and 26.9% in Tasmania. Five per cent more partially immunised children were up-to-date for meningococcal C conjugate vaccine, which is due at the same age of 12 months, compared with MMR nationally.

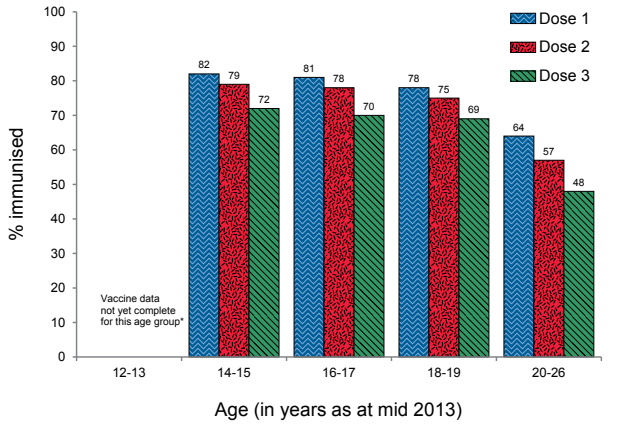
Human papillomavirus vaccine coverage

Vaccination coverage, as notified to the HPV Register, for dose 3 of the HPV vaccine for females aged 15 years in 2013 is shown in Table 12. For Australia, 71.0% of females completed a full course of the vaccine; the same figure as in 2012. Coverage varied by jurisdiction from a low of 62.8% in Tasmania to a high of 80.0% in the Northern Territory. Coverage in all age groups was higher for earlier doses; as high as 82.0% for the 1st dose in females aged 14–15 years (Figure 18). Coverage was higher in the younger age groups than the older age groups, with only 48.0% of females aged 20–26 years fully vaccinated according to data notified to the HPV Register. HPV coverage by Indigenous status was not available due to limitations in Indigenous status reporting on the HPV Register.

Provider type

GPs administer the large majority of immunisations in Australia (Figure 19); the proportion given by GPs has increased over the past 11 years by almost 5.0% (not shown). Regional differences are marked, with over 80.0% of immunisations administered by GPs in New South Wales, Queensland, and Tasmania, but the majority given by other providers in Victoria, the Australian Capital Territory, and the Northern Territory.

Figure 18: Human papillomavirus vaccination coverage for females vaccinated between April 2007 and June 2014, Australia by dose number



* In some states those aged 12–13 years in 2013 are not eligible for vaccination until 2014. Notification of 2014 doses to the Register is in progress.

Technical notes:

Includes valid doses and 'too close' doses for Clinically Complete Consumers.

Population is Estimated Resident Population provided by the Australian Bureau of Statistics – Cat 3101.0 Australia Demographic Statistics, Tables 51 to 58: Estimated Resident Population by single year of age by state and territory, published December 2013.

Age is age as at date of Estimated Resident Population estimate.

Coverage is calculated as doses administered and reported to the Human Papillomavirus (HPV) Register / estimated resident population, expressed as a percentage.

Excludes consumers who do not wish their details to be recorded on the HPV Register.

Published with permission from the Australian Government Department of Health.

Source: National HPV Vaccination Program Register, July 2014.

Table 11: Percentage of 'partially immunised' 2-year-old children* by whether up-to-date for vaccines due by 24 months of age, Australia, 2013, by state or territory

	State or territory								
	ACT	NSW	NT	Qld	SA	Tas.	Vic.	WA	Aust.
Total number of children	55	1,244	35	490	250	78	855	393	3,400
3 doses of DTPa	61.8	55.7	31.4	57.4	50.4	57.7	58.7	52.7	55.9
3 doses of IPV	61.8	55.0	31.4	56.3	50.0	57.7	57.4	52.7	55.1
4 doses of Hib	61.8	61.2	48.6	50.8	47.6	57.7	61.3	59.8	58.4
3 doses of Hep B	58.2	47.2	28.6	44.7	44.0	50.0	51.7	41.2	47.1
1 dose of MenCCV	38.2	40.8	48.6	49.2	44.4	34.6	42.8	41.7	42.8
1 dose of MMR	30.9	36.5	40.0	44.1	40.0	26.9	36.6	40.5	38.1

* Record of at least 1 vaccine recorded on the Australian Childhood Immunisation Register, the child's parent(s) are not vaccination objectors, and the child is not 'fully immunised' by 24 months of age. Cohort born 1 October to 31 December 2011 and assessed in 2013.

Source: Australian Childhood Immunisation Register.

A list of vaccine abbreviations is contained in the glossary.

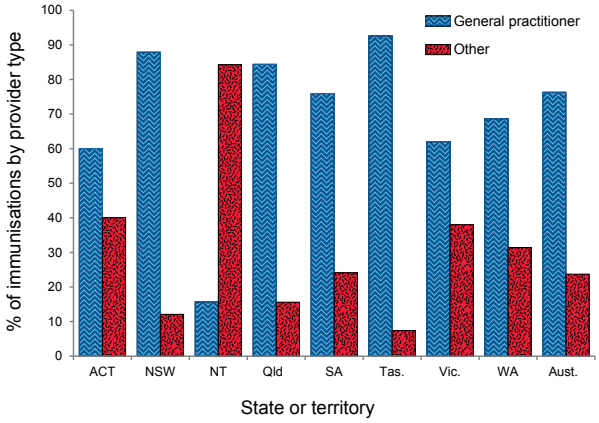
Table 12: Percentage of girls turning 15 years in 2011, 2012 and 2013 immunised for dose 3 of human papillomavirus vaccine, by state or territory

	State or territory								
	ACT	NSW	NT	Qld	SA	Tas.	Vic.	WA	Aust.
2011	73.2	72.7	79.5	70.2	66.0	64.0	74.5	64.8	71.2
2012	74.0	70.7	84.1	68.7	70.1	62.6	73.6	69.9	70.8
2013	73.2	68.4	80.0	70.5	71.7	62.8	74.8	70.8	71.0

Includes valid doses and 'too close' doses for Clinically Complete Consumers.

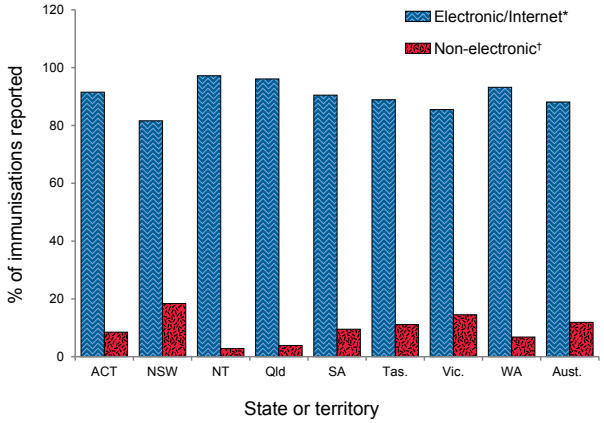
Source: National Human Papillomavirus Vaccination Program Register, July 2014.

Figure 19: Proportion of immunisations on the Australian Childhood Immunisation Register given by provider type, January to December 2013, by state or territory



Source: Australian Childhood Immunisation Register.

Figure 20: Proportion of immunisations on the Australian Childhood Immunisation Register lodged by type of reporting mechanism, January to December 2013, by state or territory



* Online claiming: Medicare Australia online claiming, a software application that allows the transmission of Australian Childhood Immunisation Register (ACIR) data via the immunisation provider's desktop software or Internet Data Interchange (IDI): approved immunisation providers can send immunisation details using the IDI upload facility through the ACIR secure area within Medicare Australia's website or record encounter: approved immunisation providers can send immunisation details using the record encounter facility through the ACIR secure area within Medicare Australia's website.

† Manual voucher: by completing an 'immunisation encounter form' and sending it to Medicare Australia or a history form: to record a child's vaccination details that may be missing from the ACIR. This form must be completed by a doctor or immunisation provider and sent to the ACIR.

Source: Australian Childhood Immunisation Register.

Mechanisms of reporting to the Australian Childhood Immunisation Register

The proportions of vaccinations on the ACIR lodged by electronic/Internet mechanisms versus non-electronic mechanisms by jurisdiction are shown in Figure 20. Most reporting in 2013 occurred through electronic/Internet mechanisms, for all jurisdictions, with the proportion reported through this method varying from 97.2% in the Northern Territory to 81.6% in New South Wales.

Discussion

This report shows that 'fully immunised' coverage targets (90%) have been reached nationally and in most jurisdictions for children 12, 24 and 60 months of age.

Coverage has been largely stable for the 12– and 24-month age groups since late 2003. However, during 2013, coverage for the 12-month age group declined by 1.8 percentage points. Half a percentage point of this decrease was due to the inclusion of the 13-valent pneumococcal conjugate vaccine in the coverage calculation algorithm for 'fully immunised' at 12 months of age, as shown by comparison with coverage calculated according to the old algorithm. The remainder of this decrease may be due to the cessation of an ACIR mail-out to parents in late 2012. Prior to December 2012, the ACIR conducted a mail-out every quarter to parents of children who were identified as not up-to-date at 9 months of age according to ACIR records.

The children targeted were those who would have been assessed against the 12-month age cohort for the following coverage quarter. The mail-out reminder letters were reinstated in December 2014.

'Fully immunised' coverage at 24 months of age continues to be stable. Coverage at this age milestone typically exceeds that at 12 months of age, which is likely related to the longer time available for late vaccinations to be assessed and due to varicella vaccine not being included in the calculation of 'fully immunised'.

Fully immunised' coverage at 60 months of age was stable in 2013 at over 91.0%, after having reached the 90.0% target for the first time in 2012.[6] The more than 10 percentage points increase in coverage in this age group since 2009 is likely due to a focus on improved timeliness of vaccination, facilitated by a change to the ACIR overdue rules in January 2009, where children became overdue for their pre-school boosters at 49 months of age instead of the previous 60 months. This change had an impact on eligibility for parent incentive payments and outcome payments for providers.

The ACIR has shown the rapid uptake of new vaccines unlike some other developed countries[23,24] However, a number of vaccines that are included in the NIP are not included when calculating 'fully immunised' status or in eligibility for incentive payments. Coverage estimates for meningococcal C conjugate vaccine in 2013 were comparable with estimates for vaccines that are included in 'fully immunised' calculations, but estimates for varicella and rotavirus remained substantially lower than for other vaccines. Varicella vaccine coverage is probably lower due to both the shorter time it has been on the NIP and the age of administration (18 months). The 18-month schedule point was historically associated with similar coverage levels prior to 2003, when there was an 18-month pertussis booster, and there was a gap of over 2 years from 2003 to 2005 when no vaccine was administered at 18 months. When we assessed varicella vaccine coverage at 36 months of age instead of 24 months we observed higher estimates across all jurisdictions, ranging from 2.6 to 5.3 percentage points higher. We also found that national varicella vaccine coverage increased by 3.5 percentage points after the introduction of MMRV vaccine in mid-2013, so further increases in coverage may occur as a result of this schedule change. For rotavirus vaccines, strict upper age limits for administration, which reduce the ability to receive late doses, may explain lower coverage when compared with other vaccines assessed at 12 months of age. The implications of lower coverage for rotavirus and varicella vaccines also differ. In the case of rotavirus vaccine, coverage of 80% or greater has resulted in substantial herd immunity and decreases in rotavirus hospitalisations in Australia and elsewhere.[25,26] In contrast, modelling studies suggest that low coverage (70.0% to 90.0%) with varicella vaccine may result in a shift of disease to older age groups with higher disease severity.[27]

Coverage for vaccines recommended for Indigenous children only (hepatitis A and a booster dose of pneumococcal polysaccharide vaccine) remained sub-optimal in 2013. The extent of under-reporting to the ACIR for these vaccines is unknown but may be more than for universal vaccines, given the lack of incentive payments for notification to the ACIR. However, lower coverage for vaccines targeted at Indigenous people has been a relatively consistent finding using a range of different methods for both children[28] and adults.[29] Both a lack of provider knowledge about the recommendations for high-risk groups, and poor identification of Indigenous children by immunisation providers are likely to be important contributing factors. Differences in schedules between jurisdictions may also contribute. While coverage for 2 doses of hepatitis A vaccine was only 60.1%, an additional 10.4% of Indigenous children received a single dose, which provides a protective antibody response in the majority of children.[30] From July 2013, the hepatitis A vaccination schedule for Indigenous children changed with dose 1 administered at 12 months of age and dose 2 at 18 months of age in all relevant jurisdictions (the Northern Territory, Queensland, South Australia and Western Australia). Hopefully this change to administration at standard schedule points will result in an increase in 2nd dose completion.

Although most children eventually complete the scheduled vaccination series by the 24-month milestone, many still do not do so in a timely manner. On-time vaccination in 2013 did not improve from 2012, for vaccines assessed at 12 and 24 months of age. Poorer timeliness in Indigenous children aged 2 years has been noted previously.[31] Timeliness has continued to improve for vaccines due at 48 months of age and assessed at 60 months of age, for both Indigenous and non-Indigenous children. However, as coverage and timeliness of vaccines assessed at 60 months of age has improved, the disparity in timeliness between Indigenous and non-Indigenous children remains. In 2013 more than 70.0% of children in the Australian Capital Territory, New South Wales, Queensland, Tasmania and Victoria received the 1st dose of DTPa vaccine prior to 8 weeks of age, in line with recommendations encouraging early protection of many young infants from pertussis infection. However, delayed vaccination for later doses is a concern, especially for diseases such as pertussis where the disease risk among young infants is significant.

Immunisation at the earliest appropriate age should be a public health goal for countries such as Australia where high levels of vaccine coverage at milestone ages have been achieved. This is especially so for the 2nd dose of the measles-mumps-rubella vaccine where vaccination delay has consistently been an issue. The change in scheduling of this dose to 18 months of age that occurred in mid-2013 may improve the timeliness of this dose.

There are 1.9% of children registered as having parental vaccination objection (a percentage that has slowly increased over time), and some others are likely unvaccinated due to unregistered objection. However incomplete immunisation is also often due to access and logistic issues. Further in-depth analysis and interpretation of the data on incompletely immunised children will be the subject of an upcoming National Centre for Immunisation Research and Surveillance of Vaccine Preventable Diseases report. Areas of low coverage have been identified in many remote areas and areas containing higher proportions of vaccination objectors. Further vaccination coverage estimates in small areas have been provided by the National Health Performance Authority for children in 2012 to 2013.[7]

Coverage data for HPV from the National HPV Vaccination Program Register reflect a successful school-based program with lower coverage for the catch-up program.[32,33] Under-notification to the HPV Register of doses administered in general practice is likely to contribute to the apparently lower coverage in women aged 20 years or over, with independent coverage estimates from population surveys in this age group suggesting under-notification of around 5.0% to 15.0%.[33,34] The 9.0% to 16.0% drop in coverage between dose 1 and 3 across all age groups may also reflect under-notification of doses missed in school and caught up in general practice but not notified to the HPV Register, as well as demonstrating the challenges in delivering a 3-dose vaccination course to adolescents.

Australia's HPV vaccination program is the most broadly targeted program in the world, with no other country having provided a free catch-up program up to the age of 26 years. The coverage achieved in the program has been sufficient to result in demonstrable decreases in HPV prevalence in young women,[35] and in genital warts[36] and cervical abnormalities.[37]

Unfortunately, coverage data for any vaccines are not available for Indigenous adolescents. For adults, data are only available from the Aboriginal and Torres Strait Islander Health Survey, last conducted in 2004–2005.[38]

Data provided in this report reflect continuing successful delivery of the NIP in Australia, while identifying some areas for improvement. Coverage for varicella and rotavirus vaccines is below that for other vaccines. Timeliness of vaccination could be improved, particularly for Indigenous infants, and coverage for vaccines recommended only for Indigenous infants was lower than for other vaccines. From July 2013, the pneumococcal conjugate vaccine has been included in coverage assessments for 'fully immunised' at 12 months of age, and thereby in eligibility for provider and parent incentives[39] and it will be important to evaluate the impact of this change in coming years.

Acknowledgements

This study was funded by the Australian Government Department of Health. Thanks to Donna Armstrong for editing the draft report.

Author details

Brynley P Hull[1]
Aditi Dey[1]
Frank H Beard[1]
Robert I Menzies[1]
Julia M Brotherton[2]
Peter B McIntyre[1]

1. National Centre for Immunisation Research and Surveillance of Vaccine Preventable Diseases, The Children's Hospital at Westmead and University of Sydney, New South Wales
2. National HPV Vaccination Program Register, Victorian Cytology Service, East Melbourne, Victoria

Corresponding author: Mr Brynley Hull, National Centre for Immunisation Research and Surveillance of Vaccine Preventable Diseases, The Children's Hospital at Westmead and University of Sydney, Locked Bag 4001, WESTMEAD NSW 2145. Telephone: +61 2 9845 1435. Facsimile: +61 2 9845 1418. Email: brynley.hull@health.nsw.gov.au

List of vaccine abbreviations

7vPCV	7-valent pneumococcal conjugate vaccine
10vPCV	10-valent pneumococcal conjugate vaccine
13vPCV	13-valent pneumococcal conjugate vaccine
23vPPV	23-valent pneumococcal polysaccharide vaccine
Comvax	*Haemophilus influenzae* type b conjugate (meningococcal protein conjugate) and hepatitis B (recombinant) vaccine
dTpa	diphtheria-tetanus-acellular pertussis (adults, adolescents and children aged ≥10 years formulation)
DTPa	diphtheria-tetanus-acellular pertussis (children aged <10 years formulation)
DTPa-hepB-IPV-Hib	combined diphtheria-tetanus-acellular pertussis-hepatitis B-inactivated poliovirus-*Haemophilus influenzae* type b
DTPa-IPV	diphtheria-tetanus-acellular pertussis-inactivated poliovirus
Engerix-B	recombinant DNA hepatitis B vaccine (paediatric formulation)
Flu	influenza
H-B-VAX II	hepatitis B (paediatric formulation)
Hep A	hepatitis A
Hep B	hepatitis B
Hib	*Haemophilus influenzae* type b
Hib-MenCCV	*Haemophilus influenzae* type b-meningococcal C conjugate vaccine
HPV	human papillomavirus
IPV	inactivated poliovirus
MenCCV	meningococcal C conjugate vaccine
MMR	measles-mumps-rubella
MMRV	measles-mumps-rubella-varicella
PedvaxHIB	*Haemophilus influenzae* type b conjugate vaccine (meningococcal protein conjugate)
PCV	pneumococcal conjugate vaccine
PRP-OMP	*Haemophilus influenzae* type b conjugate vaccine
PRP-T	*Haemophilus influenzae* type b conjugate vaccine
VZV	varicella-zoster virus

References

1. Hull B, Deeks S, Menzies R, McIntyre P. Immunisation coverage annual report, 2007. *Commun Dis Intell 2009;33(2):170–187.*
2. Hull BP, Mahajan D, Dey A, Menzies RI, McIntyre PB. Immunisation coverage annual report, 2008. *Commun Dis Intell* 2010;34(3):241–258.
3. Hull B, Dey A, Mahajan D, Menzies R, McIntyre PB. Immunisation coverage annual report, 2009. *Commun Dis Intell* 2011;35(2):132–148.
4. Hull B, Dey A, Menzies R, McIntyre P. Annual immunisation coverage report, 2010. Commun Dis Intell 2013;37(1):E21–39.
5. Hull BP, Dey A, Menzies RI, Brotherton JM, McIntyre PB. Immunisation coverage annual report, 2011. *Commun Dis Intell* 2013;37(4):E291–E312.
6. Hull BP, Dey A, Menzies RI, Brotherton JM, McIntyre PB. Immunisation coverage, 2012. *Commun Dis Intell* 2014;38(3):E208–231.
7. National Health Performance Authority. Healthy communities: Immunisation rates for children in 2012–13. 2014. Accessed on 2 December 2014. Available from: http://www.nhpa.gov.au/internet/nhpa/publishing.nsf/Content/Our-reports
8. Hull BP, Deeks SL, McIntyre PB. The Australian Childhood Immunisation Register–A model for universal immunisation registers? *Vaccine* 2009;27(37):5054–5060.
9. Australian Government Department of Health. Frequently asked questions: Changes to national immunisation schedule and related payments. 2013. Accessed on 8 May 2015. Available from: http://www.humanservices.gov.au/customer/subjects/immunising-your-children#a5
10. Hull BP, McIntyre PB, Heath TC, Sayer GP. Measuring immunisation coverage in Australia. A review of the Australian Childhood Immunisation Register. *Aust Fam Physician* 1999;28(1):55–60.
11. Australian Technical Advisory Group on Immunisation (ATAGI). The Australian immunisation handbook. 10th edn. Canberra: Australian Government Department of Health and Ageing; 2013.
12. O'Brien ED, Sam GA, Mead C. Methodology for measuring Australia's childhood immunisation coverage. *Commun Dis Intell* 1998;22(3):36–37.
13. Hull B, McIntyre P. Immunisation coverage reporting through the Australian Childhood Immunisation Register—an evaluation of the third-dose assumption. Aust N Z J Public Health 2000;24(1):17–21.
14. Hull BP, Lawrence GL, MacIntyre CR, McIntyre PB. Estimating immunisation coverage: is the 'third dose assumption' still valid? *Commun Dis Intell* 2003;27(3):357–361.
15. Hull B. Australian childhood immunisation coverage, 1 October to 31 December cohort, assessed as at 31 March 2014. *Commun Dis Intell* 2013;38(3):E260–E261.
16. Australian Population and Migration Research Centre. ARIA and accessibility. Accessibility/Remoteness Index of Australia – ARIA+ (2011). 2011. Accessed on 17 November 2014. Available from: http://www.adelaide.edu.au/apmrc/research/projects/category/aria.html
17. Rank C, Menzies RI. How reliable are Australian Childhood Immunisation Register coverage estimates for Indigenous children? An assessment of data quality and coverage. *Commun Dis Intell* 2007;31(3):283–287.
18. Australian Bureau of Statistics. Australian Statistical Geography Standard (ASGS). 2011. Accessed on 17 November 2014. Available from: http://www.abs.gov.au/websitedbs/d3310114.nsf/home/australian+statistical+geography+standard+%28asgs%29
19. MapInfo. MapInfo version 12.0. In. Stamford, Connecticut, USA: Pitney Bowes; 2013.
20. Australian Bureau of Statistics. Australian Statistical Geography Standard (ASGS): Correspondences, July 2011. 2012. Accessed on 17 November 2014. Available from: http://www.abs.gov.au/AUSSTATS/abs@.nsf/Lookup/1270.0.55.006Main+Features1July%20 2011?OpenDocument
21. Australian Institute of Health and Welfare. 2009 Adult Vaccination Survey: summary results. 2011. Accessed on 2 December 2014. Available from: http://www.aihw.gov.au/publication-detail/?id=10737418409
22. Lawrence G. Coverage of the birth dose of hepatitis B vaccine among infants. In: National Centre for Immunisation Research and Surveillance; 2006. p. 19.
23. National, state, and local area vaccination coverage among children aged 19–35 months – United States, 2012. *MMWR Morb Mortal Wkly Rep* 2013;62(36):733–740.
24. Health and Social Care Information Centre. NHS immunisation statistics, England, 2012–13. 2013. Accessed on 2 December 2014. Available from: http://www.hscic.gov.uk/catalogue/PUB11665
25. Buttery JP, Lambert SB, Grimwood K, Nissen MD, Field EJ, Macartney KK, et al. Reduction in rotavirus-associated acute gastroenteritis following introduction of rotavirus vaccine into Australia's national childhood vaccine schedule. *Pediatr Infect Dis J* 2011;30(1 Suppl):S25–S29.
26. Dey A, Wang H, Menzies R, Macartney K. Changes in hospitalisations for acute gastroenteritis in Australia after the national rotavirus vaccination program. *Med J Aust* 2012;197(8):453–457.
27. Brisson M, Edmunds WJ, Gay NJ, Law B, De Serres G. Modelling the impact of immunization on the epidemiology of varicella zoster virus. *Epidemiol Infect* 2000;125(3):651–669.
28. Hull BP, McIntyre PB. What do we know about 7vPCV coverage in Aboriginal and Torres Strait Islander children? *Commun Dis Intell* 2004;28(2):238–243.
29. Menzies R, Turnour C, Chiu C, McIntyre P. Vaccine preventable diseases and vaccination coverage in Aboriginal and Torres Strait Islander people, Australia, 2003 to 2006. *Commun Dis Intell* 2008;32 Suppl:S2–S67.
30. Plotkin S, Orenstein WA, Offit PA. Vaccines. 5th edn. Philadelphia, PA: Saunders Elsevier; 2008.
31. Hull BP, McIntyre P. Timeliness of childhood immunisation in Australia. *Vaccine* 2006;24:4403–4408.
32. Brotherton JM, Murray SL, Hall MA, Andrewartha LK, Banks CA, Meijer D, et al. Human papillomavirus vaccine coverage among female Australian adolescents: success of the school-based approach. *Med J Aust* 2013;199(9):614–617.
33. Brotherton J, Gertig D, Chappell G, Rowlands L, Saville M. Catching up with the catch-up: HPV vaccination coverage data for Australian women aged 18–26 years from the National HPV Vaccination Program Register. *Commun Dis Intell* 2011;35(2):197–201.

34. Brotherton JM, Liu B, Donovan B, Kaldor JM, Saville M. Human papillomavirus (HPV) vaccination coverage in young Australian women is higher than previously estimated: independent estimates from a nationally representative mobile phone survey. *Vaccine* 2014;32(5):592–597.

35. Tabrizi SN, Brotherton JM, Kaldor JM, Skinner SR, Liu B, Bateson D, et al. Assessment of herd immunity and cross-protection after a human papillomavirus vaccination programme in Australia: a repeat cross-sectional study. *Lancet Infect Dis* 2014;14(10):958–966.

36. Ali H, Donovan B, Wand H, Read TR, Regan DG, Grulich AE, et al. Genital warts in young Australians five years into national human papillomavirus vaccination programme: national surveillance data. *BMJ* 2013;346:f2032.

37. Gertig DM, Brotherton JM, Budd AC, Drennan K, Chappell G, Saville AM. Impact of a population-based HPV vaccination program on cervical abnormalities: a data linkage study. *BMC Med* 2013;11:227.

38. Australian Bureau of Statistics. 4715.0 – National Aboriginal and Torres Strait Islander Health Survey, 2004–05. 2006. Accessed on 2 December 2014. Available from: http://www.abs.gov.au/ausstats/abs@.nsf/mf/4715.0/

39. Australian Government Department of Health. Immunise Australia Program. 2014. Accessed on 2 December 2014. Available from: http://www.health.gov.au/internet/immunise/publishing.nsf/Content/home

Quarterly reports

National Notifiable Diseases Surveillance System, 1 October to 31 December 2015

A summary of diseases currently being reported by each jurisdiction is provided in Table 1. There were 59,081 notifications to the National Notifiable Diseases Surveillance System (NNDSS) between 1 October to 31 December 2015 (Table 2). The notification rate of diseases per 100,000 population for each state or territory is presented in Table 3.

Table 1: Reporting of notifiable diseases by jurisdiction

Disease	Data received from:
Bloodborne diseases	
Hepatitis (NEC)	All jurisdictions
Hepatitis B (newly acquired)	All jurisdictions
Hepatitis B (unspecified)	All jurisdictions
Hepatitis C (newly acquired)	All jurisdictions except Queensland
Hepatitis C (unspecified)	All jurisdictions
Hepatitis D	All jurisdictions
Gastrointestinal diseases	
Botulism	All jurisdictions
Campylobacteriosis	All jurisdictions except New South Wales
Cryptosporidiosis	All jurisdictions
Haemolytic uraemic syndrome	All jurisdictions
Hepatitis A	All jurisdictions
Hepatitis E	All jurisdictions
Listeriosis	All jurisdictions
Shiga toxin-producing *Escherichia coli*	All jurisdictions
Salmonellosis	All jurisdictions
Shigellosis	All jurisdictions
Typhoid fever	All jurisdictions
Quarantinable diseases	
Avian influenza in humans	All jurisdictions
Cholera	All jurisdictions
Middle East respiratory syndrome coronavirus	All jurisdictions
Plague	All jurisdictions
Rabies	All jurisdictions
Severe acute respiratory syndrome	All jurisdictions
Smallpox	All jurisdictions
Viral haemorrhagic fever	All jurisdictions
Yellow fever	All jurisdictions
Sexually transmissible infections	
Chlamydia	All jurisdictions
Donovanosis	All jurisdictions
Gonococcal infection	All jurisdictions
Syphilis - congenital	All jurisdictions
Syphilis <2 years duration	All jurisdictions
Syphilis >2 years or unspecified duration	All jurisdictions

Table 1 *continued*: Reporting of notifiable diseases by jurisdiction

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

NEC Not elsewhere classified.

Table 2: Notifications of diseases received by state and territory health authorities, 1 October to 31 December 2015, by date of diagnosis*

Disease	State or territory								Total 4th quarter 2015	Total 3rd quarter 2015	Total 4th quarter 2014	Last 5 years mean 4th quarter	Ratio	Year to date 2015	Last 5 years YTD mean
	ACT	NSW	NT	Qld	SA	Tas.	Vic.	WA							
Bloodborne diseases															
Hepatitis (NEC)	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0
Hepatitis B (newly acquired)†	0	4	1	8	2	0	5	4	24	38	33	44.6	0.5	141	192.4
Hepatitis B (unspecified)‡	25	554	17	283	72	12	136	148	1,247	1,814	1,567	1,599.8	0.8	6,187	6,601.2
Hepatitis C (newly acquired)†	3	4	2	0	8	5	28	66	116	119	95	99.0	1.2	449	421.8
Hepatitis C (unspecified)‡	50	934	54	669	126	66	219	271	2,389	2,389	2,533	2,516.0	0.9	9,811	10,224.0
Hepatitis D	0	0	0	1	1	0	1	0	3	12	15	13.2	0.2	38	49.2
Gastrointestinal diseases															
Botulism	0	0	0	0	0	0	0	1	1	0	0	0.0	0	3	1.4
Campylobacteriosis	198	NN	88	2,105	518	322	2,464	889	6,584	4,971	5,711	4,777.0	1.4	21,385	17,002.6
Cryptosporidiosis	11	350	54	222	83	5	312	65	1,102	480	466	440.8	2.5	4,051	2,538.2
Haemolytic uraemic syndrome	0	2	0	0	0	0	1	0	3	6	3	3.8	0.8	17	15.6
Hepatitis A	0	9	1	7	2	0	6	7	32	28	53	45.0	0.7	176	199.8
Hepatitis E	0	9	0	1	0	0	3	0	13	7	13	9.6	1.4	37	40.2
Listeriosis	1	10	0	1	1	0	8	1	22	14	20	20.4	1.1	70	78.0
STEC§	0	16	0	3	16	0	10	0	45	19	21	27.4	1.6	129	116.4
Salmonellosis	73	1,006	136	1,030	294	69	833	474	3,915	2,670	4,187	3,225.6	1.2	17,053	12,837.6
Shigellosis	6	38	36	26	22	1	85	15	229	274	286	169.2	1.4	1,174	636.0
Typhoid fever	1	9	0	6	3	0	4	0	23	19	29	29.6	0.8	114	125.0
Quarantinable diseases															
Avian influenza in humans	0	0	0	0	0	0	0	0	0	0	0	0.0	0	0	0.0
Cholera	0	1	0	0	0	0	0	0	1	0	0	0.4	2.5	2	3.8
Middle East respiratory syndrome coronavirus	0	0	0	0	0	0	0	0	0	0	0	0.0	0	0	0.0
Plague	0	0	0	0	0	0	0	0	0	0	0	0.0	0	0	0.0
Rabies	0	0	0	0	0	0	0	0	0	0	0	0.0	0	0	0.0
Severe acute respiratory syndrome	0	0	0	0	0	0	0	0	0	0	0	0.0	0	0	0.0
Smallpox	0	0	0	0	0	0	0	0	0	0	0	0.0	0	0	0.0
Viral haemorrhagic fever	0	0	0	0	0	0	0	0	0	0	0	0.0	0	0	0.0
Yellow fever	0	0	0	0	0	0	0	0	0	0	0	0.0	0	0	0.5

Table 2 *continued:* Notifications of diseases received by state and territory health authorities, 1 October to 31 December 2015, by date of diagnosis*

Disease	State or territory								Total 4th quarter 2015	Total 3rd quarter 2015	Total 4th quarter 2014	Last 5 years mean 4th quarter	Ratio	Year to date 2015	Last 5 years YTD mean
	ACT	NSW	NT	Qld	SA	Tas.	Vic.	WA							
Sexually transmissible infections															
Chlamydia[||,¶]	278	5,648	645	5,220	1,313	378	492	2,631	16,605	16,494	20,243	19,653.0	0.8	72,172	81,554.2
Donovanosis	0	0	0	0	0	0	0	0	0	0	0	0.0	0	0	0.6
Gonococcal infection[¶]	29	1,284	477	763	199	12	358	605	3,727	3,594	3,762	3,339.4	1.1	16,626	13,386.2
Syphilis <2 years duration[¶]	2	164	55	132	23	0	151	47	574	735	533	387.8	1.5	2,550	1,551.2
Syphilis >2 years or unspecified duration[‡,¶]	4	143	12	70	36	2	277	18	562	497	450	363.2	1.5	2,038	1,520.4
Syphilis – congenital	0	0	0	0	0	0	0	0	0	2	1	0.6	0	3	4.2
Vaccine preventable diseases															
Diphtheria	0	0	0	0	0	0	0	0	0	1	1	0.4	0	2	1.8
Haemophilus influenzae type b	0	0	1	0	0	0	0	1	2	8	6	4.6	0.4	16	18.8
Influenza (laboratory confirmed)	72	1,742	241	1,788	1,139	110	914	699	6,705	79,571	6,264	4,551.2	1.5	100,558	36,260.4
Measles	0	2	0	0	3	0	2	2	9	19	36	42.6	0.2	74	192.0
Mumps	1	20	9	12	5	1	1	234	283	197	39	38.2	7.4	642	170.8
Pertussis	133	5,358	22	913	525	6	992	639	8,588	5,637	4,274	7,687.2	1.1	22,495	24,385.4
Pneumococcal disease – invasive	2	95	12	48	31	13	80	33	314	587	352	364.8	0.9	1,496	1,691.6
Poliovirus infection	0	0	0	0	0	0	0	0	0	0	0	0.0	0	0	0.0
Rubella	0	3	0	2	0	1	0	0	6	3	5	5.8	1	18	36.2
Rubella – congenital	0	0	0	0	0	0	0	0	0	1	0	0.2	0	1	0.6
Tetanus	0	0	1	0	0	0	1	0	2	0	2	1.2	1.7	3	3.8
Varicella zoster (chickenpox)	7	NN	52	80	141	14	5	140	439	453	572	600.6	0.7	1,952	2,021.0
Varicella zoster (shingles)	55	NN	104	15	522	78	4	415	1,193	1,082	1,419	1,186.8	1	5,436	4,419.6
Varicella zoster (unspecified)	31	NN	1	1,725	83	43	2	345	2,230	2,140	3,160	2,660.6	0.8	10,660	9,839.2
Vectorborne diseases															
Barmah Forest virus infection	0	18	4	55	1	0	1	12	91	83	104	395.0	0.2	630	2,008.4
Chikungunya virus infection	0	4	0	0	1	0	5	1	11	15	50	22.4	0.5	110	72.6
Dengue virus infection	5	83	6	45	7	6	56	53	261	265	261	301.6	0.9	1,696	1,430.4
Flavivirus infection (unspecified)	0	0	0	1	0	0	1	0	2	1	1	2.4	0.8	9	13.2
Japanese encephalitis virus infection	0	0	0	1	0	0	0	0	1	0	0	0.0	0	3	1.2
Kunjin virus infection	0	1	0	0	0	0	0	0	1	0	0	0.6	1.7	1	1.4
Malaria	2	14	4	11	1	0	12	14	58	60	63	92.4	0.6	231	381.4
Murray Valley encephalitis virus infection	0	0	0	0	0	0	0	0	0	0	0	0.2	0	2	3.4
Ross River virus infection	3	184	57	616	24	0	35	135	1,054	809	1,517	996.8	1.1	9,541	4,916.4

Table 2 *continued*: Notifications of diseases received by state and territory health authorities, 1 October to 31 December 2015, by date of diagnosis*

Disease	State or territory								Total 4th quarter 2015	Total 3rd quarter 2015	Total 4th quarter 2014	Last 5 years mean 4th quarter	Ratio	Year to date 2015	Last 5 years YTD mean
	ACT	NSW	NT	Qld	SA	Tas.	Vic.	WA							
Zoonoses															
Anthrax	0	0	0	0	0	0	0	0	0	0	0	0.0	0	0	0.2
Australian bat lyssavirus infection	0	0	0	0	0	0	0	0	0	0	0	0.0	0	0	0.2
Brucellosis	0	1	0	1	0	0	0	0	2	5	3	6.0	0.3	18	24.0
Leptospirosis	0	3	3	9	0	0	1	0	16	17	10	19.8	0.8	73	126.6
Lyssavirus infection (NEC)	0	0	0	0	0	0	0	0	0	0	0	0.0	0	0	0.0
Ornithosis	0	2	0	1	0	0	1	1	5	2	14	23.8	0.2	11	62.2
Q fever	0	51	0	41	2	0	17	4	115	161	109	101.4	1.1	576	405.2
Tularaemia	0	0	0	0	0	0	0	0	0	0	0	0.3	0	0	0.5
Other bacterial infections															
Legionellosis	0	16	1	18	10	2	18	22	87	90	116	106.2	0.8	371	395.8
Leprosy	0	2	0	0	0	0	2	0	4	5	2	2.8	1.4	13	10.2
Meningococcal infection – invasive**	0	10	0	6	7	1	14	3	41	66	46	45.0	0.9	182	202.2
Tuberculosis	1	111	8	57	18	4	105	40	344	313	377	369.8	0.9	1,244	1,334.0
Total	993	17,905	2,104	15,992	5,239	1,151	7,662	8,035	59,081	125,773	58,824			312,290	

* The date of diagnosis is the onset date or where the date of onset was not known, the earliest of the specimen collection date, the notification date, or the notification receive date. For hepatitis B (unspecified), hepatitis C (unspecified), leprosy, syphilis (> 2 years or unspecified duration) and tuberculosis, the public health unit notification receive date was used.

† Newly acquired hepatitis includes cases where the infection was determined to be acquired within 24 months prior to diagnosis. Queensland reports hepatitis C newly acquired under hepatitis unspecified.

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

§ Infection with Shiga toxin-producing *Escherichia coli.*

|| Includes *Chlamydia trachomatis* identified from cervical, rectal, urine, urethral and throat samples, except for South Australia, which reports only cervical, urine and urethral specimens.

¶ The national case definitions for chlamydial, gonococcal and syphilis diagnoses include infections that may be acquired through a non-sexual mode (especially in children – e.g. perinatal infections, epidemic gonococcal conjunctivitis).

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

NN Not notifiable

NEC Not elsewhere classified

Totals comprise data from all states and territories. Cumulative figures are subject to retrospective revision so there may be discrepancies between the number of new notifications and the increment in the cumulative figure from the previous period.

Table 3: Notification rates of diseases, 1 October to 31 December 2015, by state or territory. (Annualised rate per 100,000 population)*,†

Disease	State or territory								
	ACT	NSW	NT	Qld	SA	Tas.	Vic.	WA	Aust.
Bloodborne diseases									
Hepatitis (NEC)	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
Hepatitis B (newly acquired)‡	0.0	0.2	1.6	0.7	0.5	0.0	0.3	0.6	0.4
Hepatitis B (unspecified)§	25.9	29.5	27.8	24.0	17.1	9.3	9.3	23.1	21.2
Hepatitis C (newly acquired)‡	3.1	0.2	3.3	0.0	1.9	3.9	1.9	10.3	2.0
Hepatitis C (unspecified)§	51.9	49.7	88.3	56.7	29.9	51.3	15.0	42.3	40.7
Hepatitis D	0.0	0.0	0.0	0.1	0.2	0.0	0.1	0.0	0.1
Gastrointestinal diseases									
Botulism	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.2	0.0
Campylobacteriosis	205.5	NN	143.9	178.3	122.9	250.2	168.8	138.6	165.0
Cryptosporidiosis	11.4	18.6	88.3	18.8	19.7	3.9	21.4	10.1	18.8
Haemolytic uraemic syndrome	0.0	0.1	0.0	0.0	0.0	0.0	0.1	0.0	0.1
Hepatitis A	0.0	0.5	1.6	0.6	0.5	0.0	0.4	1.1	0.5
Hepatitis E	0.0	0.5	0.0	0.1	0.0	0.0	0.2	0.0	0.2
Listeriosis	1.0	0.5	0.0	0.1	0.2	0.0	0.5	0.2	0.4
STEC‖	0.0	0.9	0.0	0.3	3.8	0.0	0.7	0.0	0.8
Salmonellosis	73.7	53.1	222.4	87.2	68.8	53.6	56.4	73.8	66.2
Shigellosis	6.2	2.0	58.9	2.2	5.2	0.8	5.8	2.3	3.9
Typhoid fever	1.0	0.5	0.0	0.5	0.7	0.0	0.3	0.0	0.4
Quarantinable diseases									
Avian influenza in humans	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
Cholera	0.0	0.1	0.0	0.0	0.0	0.0	0.0	0.0	0.0
Middle East respiratory syndrome coronavirus	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
Plague	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
Rabies	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
Severe acute respiratory syndrome	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
Smallpox	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
Viral haemorrhagic fever	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
Yellow fever	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
Sexually transmitted infections									
Chlamydia¶,**	288.5	300.6	1,054.7	442.2	311.6	293.8	33.7	410.2	282.9
Donovanosis	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
Gonococcal infection**	30.1	68.3	780.0	64.6	47.2	9.3	24.5	94.3	63.5
Syphilis < 2 years duration**	2.1	8.7	89.9	11.2	5.5	0.0	10.3	7.3	9.8
Syphilis > 2 years or unspecified duration§,**	4.2	7.6	19.6	5.9	8.5	1.6	19.0	2.8	9.6
Syphilis – congenital	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
Vaccine preventable diseases									
Diphtheria	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
Haemophilus influenzae type b	0.0	0.0	1.6	0.0	0.0	0.0	0.0	0.2	0.0
Influenza (laboratory confirmed)	74.7	92.7	394.1	151.5	270.3	85.5	62.6	109.0	114.2
Measles	0.0	0.1	0.0	0.0	0.7	0.0	0.1	0.3	0.2
Mumps	1.0	1.1	14.7	1.0	1.2	0.8	0.1	36.5	4.8
Pertussis	138.0	285.2	36.0	77.4	124.6	4.7	68.0	99.6	146.3
Pneumococcal disease – invasive	2.1	5.1	19.6	4.1	7.4	10.1	5.5	5.1	5.4
Poliovirus infection	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
Rubella	0.0	0.2	0.0	0.2	0.0	0.8	0.0	0.0	0.1
Rubella – congenital	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0

Table 3 *continued*: Notification rates of diseases, 1 October to 31 December 2015, by state or territory. (Annualised rate per 100,000 population)*,†

Disease	State or territory								Aust.
	ACT	NSW	NT	Qld	SA	Tas.	Vic.	WA	
Vaccine preventable diseases, *cont'd*									
Tetanus	0.0	0.0	1.6	0.0	0.0	0.0	0.1	0.0	0.0
Varicella zoster (chickenpox)	7.3	NN	85.0	6.8	33.5	10.9	0.3	21.8	11.0
Varicella zoster (shingles)	57.1	NN	170.1	1.3	123.9	60.6	0.3	64.7	29.9
Varicella zoster (unspecified)	32.2	NN	1.6	146.1	19.7	33.4	0.1	53.8	55.9
Vectorborne diseases									
Barmah Forest virus infection	0.0	1.0	6.5	4.7	0.2	0.0	0.1	1.9	1.6
Chikungunya virus infection	0.0	0.2	0.0	0.0	0.2	0.0	0.3	0.2	0.2
Dengue virus infection	5.2	4.4	9.8	3.8	1.7	4.7	3.8	8.3	4.4
Flavivirus infection (unspecified)	0.0	0.0	0.0	0.1	0.0	0.0	0.1	0.0	0.0
Japanese encephalitis virus infection	0.0	0.0	0.0	0.1	0.0	0.0	0.0	0.0	0.0
Kunjin virus infection	0.0	0.1	0.0	0.0	0.0	0.0	0.0	0.0	0.0
Malaria	2.1	0.7	6.5	0.9	0.2	0.0	0.8	2.2	1.0
Murray Valley encephalitis virus infection	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
Ross River virus infection	3.1	9.8	93.2	52.2	5.7	0.0	2.4	21.0	18.0
Zoonoses									
Anthrax	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
Australia bat lyssavirus infection	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
Brucellosis	0.0	0.1	0.0	0.1	0.0	0.0	0.0	0.0	0.0
Leptospirosis	0.0	0.2	4.9	0.8	0.0	0.0	0.1	0.0	0.3
Lyssavirus infection (NEC)	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
Ornithosis	0.0	0.1	0.0	0.1	0.0	0.0	0.1	0.2	0.1
Q fever	0.0	2.7	0.0	3.5	0.5	0.0	1.2	0.6	2.0
Tularaemia	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
Other bacterial diseases									
Legionellosis	0.0	0.9	1.6	1.5	2.4	1.6	1.2	3.4	1.5
Leprosy	0.0	0.1	0.0	0.0	0.0	0.0	0.1	0.0	0.1
Meningococcal infection – invasive††	0.0	0.5	0.0	0.5	1.7	0.8	1.0	0.5	0.7
Tuberculosis	1.0	5.9	13.1	4.8	4.3	3.1	7.2	6.2	5.9

* The date of diagnosis is the onset date or where the date of onset was not known, the earliest of the specimen collection date, the notification date, or the notification receive date. For hepatitis B (unspecified), hepatitis C (unspecified), leprosy, syphilis (> 2 years or unspecified duration) and tuberculosis, the public health unit notification receive date was used.

† Rate per 100,000 of population. Annualisation Factor was 4.0

‡ Newly acquired hepatitis includes cases where the infection was determined to be acquired within 24 months prior to diagnosis. Queensland reports hepatitis C newly acquired under hepatitis C unspecified.

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

|| Infection with Shiga toxin-producing *Escherichia coli*.

¶ Includes *Chlamydia trachomatis* identified from cervical, rectal, urine, urethral and throat samples, except for South Australia, which reports only cervical, urine and urethral specimens.

** The national case definitions for chlamydia, gonococcal and syphilis diagnoses include infections that may be acquired through a non-sexual mode (especially in children – e.g. perinatal infections, epidemic gonococcal conjunctivitis).

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

NEC Not elsewhere classified.

NN Not notifiable.

Australian childhood immunisation coverage, 1 July to 30 June cohort, assessed as at 30 September 2015

Alexandra Hendry for the National Centre for Immunisation Research and Surveillance of Vaccine Preventable Diseases

Introduction

The National Centre for Immunisation Research and Surveillance of Vaccine Preventable Diseases (NCIRS) provides commentary on the trends in ACIR data. For further information please contact NCIRS at: telephone +61 2 9845 1423, email: alexandra.hendry@health.nsw.gov.au

Tables 1, 2 and 3 provide the latest rolling annualised quarterly report on childhood immunisation coverage from the Australian Childhood Immunisation Register (ACIR) for all children.

The data show the percentage of all children 'fully immunised' at 12 months, 24 months and 60 months, for four 3-month birth cohorts of children assessed at the stated ages between 1 July 2014 and 30 June 2015 using ACIR data up to 30 September 2015. 'Fully immunised' refers to vaccines on the National Immunisation Program Schedule, but excludes rotavirus, and is outlined in more detail below.

'Fully immunised' at 12 months of age is defined as a child having a record on the ACIR of 3 doses of a diphtheria (D), tetanus (T) and pertussis-containing (P) vaccine, 3 doses of polio vaccine, 2 or 3 doses of *Haemophilus* B conjugate (PMP-OMP) containing *Haemophilus influenzae* type b (Hib) vaccine or 3 doses of any other Hib vaccine, 3 doses of hepatitis B vaccine, and 3 doses of 13-valent pneumococcal conjugate vaccine.

'Fully immunised' at 24 months of age is defined as a child having a record on the ACIR of 3 doses of a DTP-containing vaccine, 3 doses of polio vaccine, 3 or 4 doses of PRP-OMP Hib vaccine or 4 doses of any other Hib vaccine, 3 doses of hepatitis B vaccine, 2 doses of a measles-mumps-rubella-containing (MMR) vaccine, 1 dose of meningococcal C vaccine, and 1 dose of varicella vaccine.

'Fully immunised' at 60 months of age is defined as a child having a record on the ACIR of 4 doses of a DTP-containing vaccine, 4 doses of polio vaccine, and 2 doses of an MMR-containing vaccine.

A full description of the basic methodology used can be found in *Commun Dis Intell* 1998;22(3):36–37.

Results

The rolling annualised percentage of all children 'fully immunised' by 12 months of age for Australia increased marginally from the previous report by 0.4 of a percentage point to 91.7% (Table 1). All jurisdictions experienced small increases in the percentage of children 'fully immunised' by 12 months of age. For individual vaccines due by 12 months of age all jurisdictions achieved coverage greater than 91%.

The rolling annualised percentage of all children 'fully immunised' by 24 months of age for Australia decreased again for the 3rd consecutive report by 0.6 percentage points to 88.6% (Table 2). All jurisdictions experienced similar decreases

Table 1. Percentage of children immunised at 12 months of age for the birth cohort 1 July 2013 to 30 June 2014, preliminary results, by disease and state or territory; assessment date 30 September 2015

Vaccine	State or territory								Aust
	ACT	NSW	NT	Qld	SA	Tas	Vic	WA	
Total number of children	5,656	98,857	3,739	62,832	20,077	5,924	76,197	34,229	307,511
Diphtheria, tetanus, pertussis (%)	94.3	92.3	92.5	92.7	92.4	91.9	92.4	92.5	92.5
Poliomyelitis (%)	94.3	92.3	92.5	92.6	92.3	91.8	92.4	92.5	92.4
Haemophilus influenzae type b (%)	94.0	92.1	92.4	92.5	92.2	91.8	92.2	92.3	92.3
Hepatitis B (%)	93.9	92.0	92.6	92.5	92.1	91.7	92.1	92.2	92.2
Pneumococcal	94.0	91.9	92.5	92.4	92.0	91.7	92.1	92.1	92.1
Fully immunised (%)	93.3	91.5	92.0	92.1	91.6	91.3	91.5	91.6	91.7

in fully immunised coverage for this age group. Coverage for individual vaccines due by 24 months remained high in all jurisdictions, except for varicella and the measles, mumps and rubella vaccine. This is likely due to the fact that the cohort used in this report for coverage at 24 months of age is the first full 12-month birth cohort (4 quarters) to be assessed at 24 months of age for the dose of measles-mumps-rubella-varicella vaccine due at 18 months of age (the 2nd dose of MMR and the 1st dose of varicella).

The rolling annualised percentage of all children 'fully immunised' by 60 months of age for Australia was the same as the previous report (92.3%) (Table 3). There were also only marginal changes in fully immunised coverage at 60 months of age in all jurisdictions. Coverage for individual vaccines due by 60 months remained greater than 90% in all jurisdictions.

The Figure shows the trends in vaccination coverage from the first ACIR-derived published coverage estimates in 1997 to the current estimates. There is a clear trend of increasing vaccination coverage over time for children aged 12 months, 24 months and 60 months (from December 2007). From September 2014, coverage at 24 months is lower than coverage at 12 and 60 months of age. This was most likely due to the change in the 24 month coverage assessment algorithm as described above.

Figure: Trends in vaccination coverage, Australia, 1997 to 30 June 20153, by age cohorts

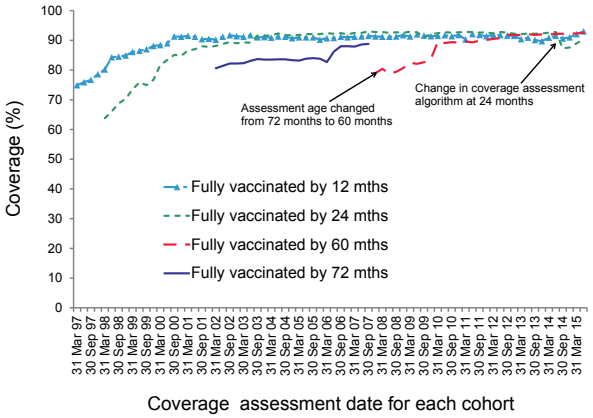


Table 2. Percentage of children immunised at 24 months of age for the birth cohort 1 July 2012 to 30 June 2013, preliminary results, by disease and state or territory; assessment date 30 September 2015*

Vaccine	State or territory								
	ACT	NSW	NT	Qld	SA	Tas	Vic	WA	Aust
Total number of children	5,631	100,593	3,580	63,074	20,194	5,926	77,077	34,222	310,297
Diphtheria, tetanus, pertussis (%)	96.3	95.2	94.8	95.1	95.0	95.3	95.8	95.0	95.3
Poliomyelitis (%)	96.2	95.2	94.8	95.1	94.9	95.2	95.8	95.0	95.3
Haemophilus influenzae type b (%)	95.2	93.9	94.2	94.3	93.7	93.7	94.6	93.8	94.2
Measles, mumps, rubella (%)	92.5	90.4	90.4	91.1	89.8	89.5	90.9	89.1	90.5
Hepatitis B (%)	95.9	94.9	95.0	94.8	94.6	95.0	95.5	94.5	95.0
Meningococcal C (%)	94.7	93.8	94.1	94.3	92.9	93.9	94.1	92.9	93.9
Varicella (%)	94.1	91.5	90.0	91.6	90.7	89.7	92.2	90.4	91.5
Fully immunised (%)	90.8	88.3	87.2	89.7	87.2	86.9	89.0	87.0	88.6

Table 3. Percentage of children immunised at 60 months of age for the birth cohort 1 July 2009 to 30 June 2010, preliminary results, by disease and state or territory; assessment date 30 September 2015

Vaccine	State or territory								
	ACT	NSW	NT	Qld	SA	Tas	Vic	WA	Aust
Total number of children	5,596	101,839	3,443	65,350	20,350	6,258	76,579	34,324	313,739
Diphtheria, tetanus, pertussis (%)	94.0	93.4	93.2	92.7	91.6	93.4	93.3	91.4	92.9
Poliomyelitis (%)	94.0	93.4	93.2	92.7	91.6	93.3	93.3	91.4	92.9
Measles, mumps, rubella (%)	93.8	93.3	93.6	92.7	91.6	93.2	93.3	91.3	92.9
Fully immunised (%)	93.5	92.9	92.5	92.2	91.0	92.7	92.7	90.7	92.3

Australian Gonococcal Surveillance Programme, 1 July to 30 September 2015

Monica M Lahra, Rodney P Enriquez, The Prince of Wales Hospital, Randwick, for The National Neisseria Network

Introduction

The Australian National Neisseria Network (NNN) comprises reference laboratories in each state and territory that report data on sensitivity to an agreed group of antimicrobial agents for the Australian Gonococcal Surveillance Programme (AGSP). The antibiotics are penicillin, ceftriaxone, azithromycin and ciprofloxacin, which are current or potential agents used for the treatment of gonorrhoea. Azithromycin testing has been recently introduced by all states and territories as it is part of a dual therapy regimen with ceftriaxone recommended for the treatment of gonorrhoea in the majority of Australia. Because of the substantial geographic differences in susceptibility patterns in Australia, regional as well as aggregated data are presented. In certain remote regions of the Northern Territory and Western Australia gonococcal antimicrobial resistance rates are low and an oral treatment regimen comprising amoxycillin, probenecid and azithromycin is recommended for the treatment of gonorrhoea. When in vitro resistance to a recommended agent is demonstrated in 5% or more of isolates from a general population, it is usual to remove that agent from the list of recommended treatments.[1] Additional data on other antibiotics are reported in the AGSP annual report. The AGSP has a program-specific quality assurance process. The AGSP data are presented quarterly in tabulated form, as well as in the AGSP annual report. For more information see *Commun Dis Intell* 2016;40(1):E178–E179.

Results

A summary of the proportion of isolates with decreased susceptibility to ceftriaxone, and the proportion resistant to azithromycin, penicillin and ciprofloxacin are shown in Table 1.

Penicillin

Penicillin resistant *Neisseria gonorrhoeae* are defined as those isolates with a minimum inhibitory concentration (MIC) to penicillin equal to or greater than 1.0 mg/L. Penicillin resistance includes penicillinase producing *N. gonorrhoeae* (PPNG), and *N. gonorrhoeae* that have chromosomally mediated resistance to penicillin (CMRP). In certain areas of the Northern Territory and Western Australia, which are classified as remote, a treatment regimen based on oral amoxycillin, probenecid and azithromycin is used. Due to the distance specimens must travel in these remote regions to a laboratory, low numbers of cultures are collected, and thus, by necessity, nucleic acid amplification testing (NAAT) is used. In remote Western Australia the introduction of a targeted NAAT, developed by the NNN to detect PPNG, is in use to enhance surveillance.[2,3]

Table 1: Gonococcal isolates showing decreased susceptibility to ceftriaxone and resistance to azithromycin, penicillin and ciprofloxacin, Australia, 1 July to 30 September 2015, by state or territory

State or territory	Number of isolates tested	Decreased susceptibility		Resistance					
		Ceftriaxone		Azithromycin		Penicillin		Ciprofloxacin	
		n	%	n	%	n	%	n	%
Australian Capital Territory	18	0	0.0	0	0.0	1	5.6	2	11.0
New South Wales	506	7	1.4	12	2.4	159	31.0	178	35.0
Queensland	176	3	1.7	5	2.8	40	23.0	38	22.0
South Australia	57	5	8.8	2	3.5	18	32.0	26	46.0
Tasmania	3	0	0.0	0	0.0	1	33.0	0	0.0
Victoria	399	7	1.8	3	0.8	55	14.0	83	21.0
Northern Territory/Urban and Rural	16	0	0.0	0	0.0	0	0.0	0	0.0
Northern Territory/Remote	32	0	0.0	0	0.0	0	0.0	0	0.0
Western Australia/Urban and Rural	101	1	1.0	0	0.0	14	14.0	19	19.0
Western Australia/Remote	25	0	0.0	0	0.0	1	4.0	1	4.0
Australia	1,333	23	1.7	22	1.7	289	22.0	347	26.0

Ciprofloxacin

Ciprofloxacin resistance includes isolates with an MIC to ciprofloxacin equal to or greater than 1.0 mg/L.

Azithromycin

Azithromycin resistance is defined as a MIC to azithromycin equal to or greater than 1.0 mg/L.

Ceftriaxone

Ceftriaxone MIC values in the range 0.06–0.125 mg/L have been reported in the category decreased susceptibility since 2005.

In the 3rd quarter of 2015 the states that reported isolates with decreased susceptibility to ceftriaxone were New South Wales, Victoria, Queensland, South Australia and urban Western Australia. All states, except for South Australia, reported a decrease in the proportion of NG isolates with DS to ceftriaxone when compared with the same quarter in 2014; and the annual data for 2014.[4]

From New South Wales, there were 7/506 strains with decreased susceptibility to ceftriaxone. Of those, 5 (72%) were multi-drug resistant (MDR); all (100%) were from males; and 2 (29%) were isolated from extragenital sites (rectal and pharyngeal). From Victoria, there were 7/399 strains with decreased susceptibility to ceftriaxone and, of those, 3 (43%) were MDR; all (100%) were from males; and 6 (86%) were isolated from extragenital sites. From Queensland, there were 3/176 strains with decreased susceptibility to ceftriaxone. Of those, none were MDR; 2 (67%) were from males; and 2 (67%) were from extragenital sites. From urban Western Australia, there was 1/101 strains with decreased susceptibility to ceftriaxone. The isolate was from a male; it was not MDR; nor isolated from extragenital sites. From South Australia in this quarter, there were 5/57 strains with decreased susceptibility to ceftriaxone. All (100%) were MDR; 4 (80%) were from males and 35 (60%) were isolated from extragenital sites.

The proportion of strains with decreased susceptibility to ceftriaxone is of increasing concern in Australia and overseas, as this is phenotypic of the genotype with the key mutations that are the precursor to ceftriaxone resistance.[5] There are recent reports of ceftriaxone 500 mg treatment failures in patients from Victoria and New South Wales in patients with pharyngeal gonococcal infections. In these patients the infecting gonococcal strains had ceftriaxone MIC values in the range 0.03–0.06 mg/L.[6,7] Until 2013 there had not been an isolate reported in Australia with a ceftriaxone MIC value >0.125 mg/L.[4] In late December 2013 there was a new multi-drug resistant gonococcal strain (A8806) with a ceftriaxone MIC of 0.5 mg/L, the highest ever reported in Australia, that was isolated from a female traveller from Central Europe. This infection was acquired in Sydney from another traveller, also from Europe. The patient was tested in the Northern Territory, but had travelled to north eastern Queensland before the results were available, and was treated there. To date there has been no evidence of spread of this strain.[8]

The category of ceftriaxone decreased susceptibility as reported by the AGSP includes the MIC values 0.06 and 0.125 mg/L (Table 2).

Dual therapy of ceftriaxone plus azithromycin is the recommended treatment for gonorrhoea as a strategy to temper development of more widespread resistance.[8] Patients with infections in extra genital sites, where the isolate has decreased susceptibility to ceftriaxone, are recommended to have test of cure cultures collected. Continued surveillance to monitor N. gonorrhoeae with elevated MIC values, coupled with sentinel site surveillance in high risk populations remains important to inform therapeutic strategies, to identify incursion of resistant strains, and to detect instances of treatment failure.

Table 2: Percentage of gonococcal isolates with decreased susceptibility to ceftriaxone MIC 0.06–0.125 mg/L, Australia, 2010 to 2014, 1 January to 31 March 2015, 1 April to 30 June 2015, and 1 July to 30 September, by state or territory

Ceftriaxone MIC mg/L	2010	2011	2012	2013	2014	2015 Q1	2015 Q2	2015 Q3
0.06	4.6	3.2	4.1	8.2	4.8	1.6	1.1	1.7
0.125	0.1	0.1	0.3	0.6	0.6	0.1	0.0	0.0

References

1. Surveillance of antibiotic susceptibility of *Neisseria gonorrhoeae* in the WHO western Pacific region 1992–4. WHO Western Pacific Region Gonococcal Antimicrobial Surveillance Programme. *Genitourin Med* 1997;73(5):355–361.
2. Speers DJ, Fisk RE, Goire N, Mak DB. Non-culture *Neisseria gonorrhoeae* molecular penicillinase production surveillance demonstrates the long-term success of empirical dual therapy and informs gonorrhoea management guidelines in a highly endemic setting. *J Antimicrob Chemother* 2014;69(5):1243–1247.
3. Goire N, Freeman K, Tapsall JW, Lambert SB, Nissen MD, Sloots TP, et al. Enhancing gonococcal antimicrobial resistance surveillance: a real-time PCR assay for detection of penicillinase-producing *Neisseria gonorrhoeae* by use of noncultured clinical samples. *J Clin Microbiol* 2011;49(2):513–518.
4. Lahra MM. Australian Gonococcal Surveillance Programme, 2013. *Commun Dis Intell* 2015;39(1):E137–E145.
5. Goire N, Lahra MM, Chen M, Donovan B, Fairley CK, Guy R, et al. Molecular approaches to enhance surveillance of gonococcal antimicrobial resistance. *Nat Rev Microbiol* 2014;12(3):223–229.
6. Chen YM, Stevens K, Tideman R, Zaia A, Tomita T, Fairley CK, et al. Failure of 500 mg of ceftriaxone to eradicate pharyngeal gonorrhoea, Australia. *J Antimicrob Chemother* 2013;68(6):1445–1447.
7. Read PJ, Limnios EA, McNulty A, Whiley D, Lahra MM. One confirmed and one suspected case of pharyngeal gonorrhoea treatment failure following 500 mg ceftriaxone in Sydney, Australia. *Sex Health* 2013;10(5):460–462.
8. Australasian Sexual Health Association. *The Australian Sexually Transmitted Infection Management Guidelines 2014*. [Online]. Available from: www.sti.guidelines.org.au

Australian Sentinel Practices Research Network, 1 October to 31 December 2015

Monique B-N Chilver, Daniel Blakeley, Nigel P Stocks for the Australian Sentinel Practices Research Network

Introduction

The Australian Sentinel Practices Research Network (ASPREN) is a national surveillance system that is funded by the Australian Government Department of Health, owned and operated by the Royal Australian College of General Practitioners and directed through the Discipline of General Practice at the University of Adelaide.

The network consists of general practitioners who report presentations on a number of defined medical conditions each week. ASPREN was established in 1991 to provide a rapid monitoring scheme for infectious diseases that can alert public health officials of epidemics in their early stages as well as play a role in the evaluation of public health campaigns and research of conditions commonly seen in general practice. Electronic, web-based data collection was established in 2006.

Since 2010, ASPREN GPs have been collecting nasal swab samples for laboratory testing, allowing for viral testing of 20% of influenza-like illness (ILI) patients for a range of respiratory viruses including influenza A, influenza B and A(H1N1) pdm09.

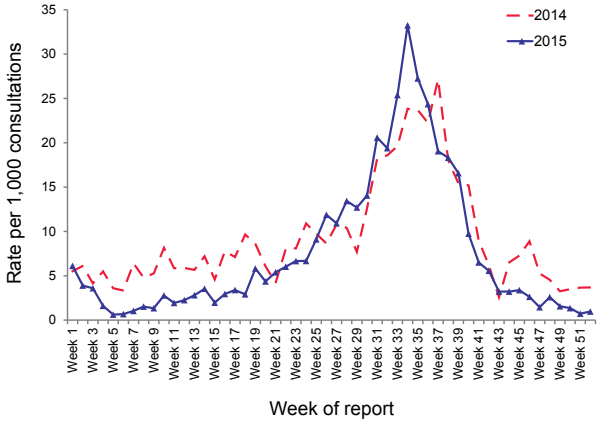
The list of conditions reported is reviewed annually by the ASPREN management committee. In 2015, 4 conditions are being monitored. They include ILI, gastroenteritis and varicella infections (chickenpox and shingles). Definitions of these conditions are described in Surveillance systems reported in CDI, published in *Commun Dis Intell* 2015;39(1):E180.

Results

Sentinel practices contributing to ASPREN were located in all 8 states and territories in Australia. A total of 240 general practitioners regularly contributed data to ASPREN in the 4th quarter of 2015. Each week an average of 224 general practitioners provided information to ASPREN at an average of 17,017 (range 10,834 to 18,362) consultations per week and an average of 152 (range 63 to 235) notifications per week.

ILI rates reported from 1 October to 31 December 2015 averaged 3.3 cases per 1,000 consultations (range 0.7 to 9.8 cases per 1,000 consultations). This was lower compared with rates in the same reporting period in 2014, which averaged 6.1 cases per 1,000 consultations (range 2.6 to 15.2 cases per 1,000 consultations, Figure 1). ILI rates peaked in week 34 at a rate of 33.2 ILI cases per 1,000 consultations.

Figure 1: Weighted* consultation rates for influenza-like illness, ASPREN, 2014 and 1 January to 31 December 2015, by week of report



* Results are weighted to account for population size differences between jurisdictions, using population estimates from the Australian Bureau of Statistics 2011 Census.

The ASPREN ILI swab testing program continued in 2015 with 241 tests being undertaken from 1 October to 31 December. The most commonly reported virus during this reporting period was influenza A (12.0% of all swabs performed, Figure 2), with the second most common virus being influenza B (7.1% of all swabs performed).

From the beginning of 2015 to the end of week 52, 831 cases of influenza were detected with 522 of these typed as influenza B (18.7% of all swabs performed) and the remaining 309 being influenza A (11.1% of all swabs performed) (Figure 2).

During this reporting period, consultation rates for gastroenteritis averaged 5.7 cases per 1,000 consultations (range 3.2 to 8.1 cases per 1,000, Figure 3). This was similar to the rates in the same reporting period in 2014 where the average was 5.9 cases per 1,000 consultations (range 3.3 to 9.8 cases per 1,000).

Figure 2: Influenza-like illness swab testing results, ASPREN, 1 January to 31 December 2015, by week of report

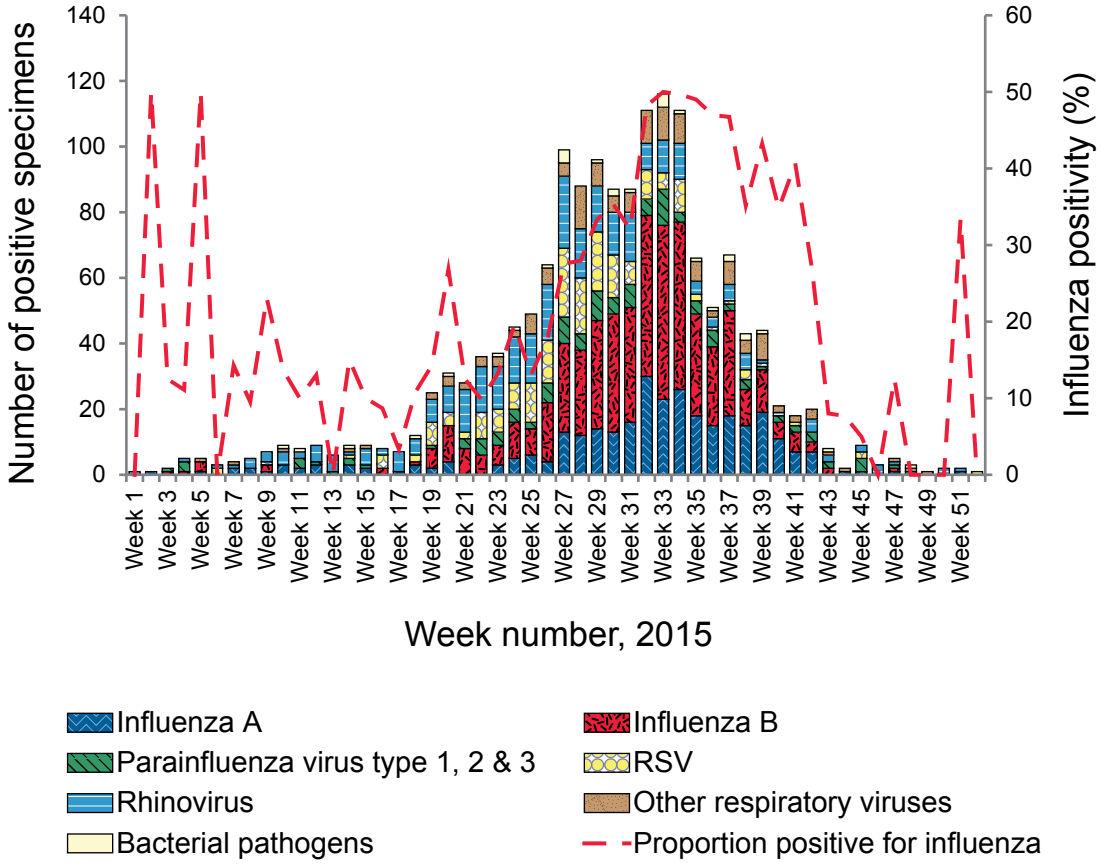


Figure 3: Consultation rates for gastroenteritis, ASPREN, 2014 and 1 January to 31 December 2015, by week of report

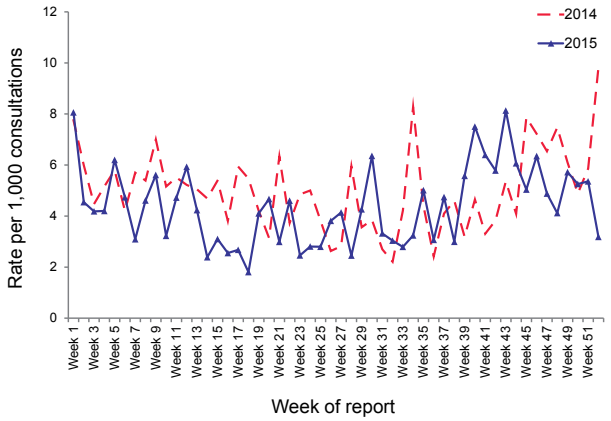
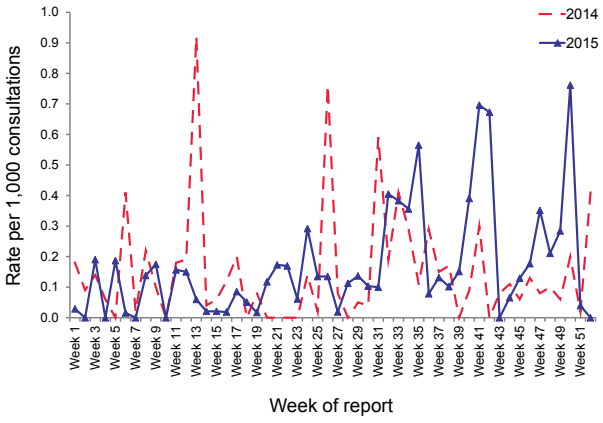


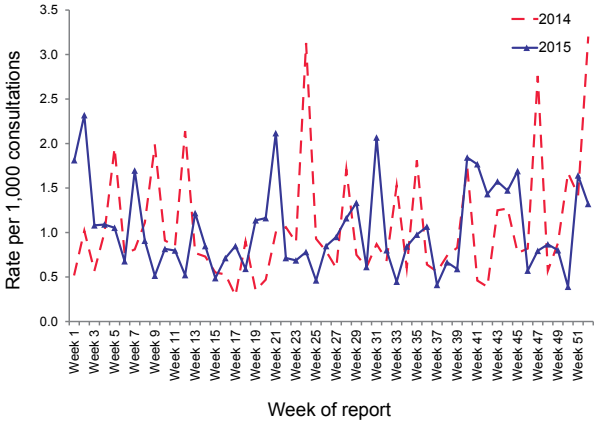
Figure 4: Consultation rates for chickenpox, ASPREN, 2014 and 1 January to 31 December 2015, by week of report



Varicella infections were reported at a higher rate for the 4th quarter of 2015 compared with the same period in 2014. From 1 October to 31 December 2015, recorded rates for chickenpox averaged 0.3 cases per 1,000 consultations (range 0.0 to 0.8 cases per 1,000 consultations, Figure 4).

In the 4th quarter of 2015, reported rates for shingles averaged 1.2 cases per 1,000 consultations (range 0.4 to 1.8 cases per 1,000 consultations, Figure 5) This was similar to the rates in the same reporting period in 2014 where the average shingles rate was 1.3 cases per 1,000 consultations (range 0.4 to 3.2 cases per 1,000 consultations).

Figure 5: Consultation rates for shingles, ASPREN, 2014 and 1 January to 31 December 2015, by week of report



Invasive pneumococcal disease surveillance Australia, 1 October to 31 December 2015

Rachel de Kluyver, Cindy Toms and the Enhanced Invasive Pneumococcal Disease Surveillance Working Group, for the Communicable Diseases Network Australia

Summary

The number of notified cases of invasive pneumococcal disease (IPD) in the 4th quarter of 2015 was fewer than the previous quarter and less than the number of notified cases in the 4th quarter of 2014. Overall, the decline in disease due to the serotypes targeted by the 13-valent pneumococcal conjugate vaccine (13vPCV) has been maintained across all age groups, since the 13vPCV replaced the 7-valent pneumococcal conjugate vaccine (7vPCV) in the childhood immunisation program from July 2011.

Key points

In the 4th quarter of 2015, there were 316 cases of IPD reported to the NNDSS. This was a 10% reduction on the number of cases reported for the same period in 2014 (n=352) (Table 1). For the calendar year, the total number of cases was similar to 2014 (n=1,543). For the reporting quarter and the 2015 calendar year, serotypes 3, 19A and 22F were the most common serotypes, which together accounted for 24% of annual cases (Table 2).

In non-Indigenous Australians, the number of notified cases was highest in the 60–64 years age group followed by the under 5 years age group. In Indigenous Australians, notified cases were highest in the under 5 years age group followed by the 50–54 years age group (Table 3). The proportion of cases reported as Indigenous increased to 16% compared with 11% in the 4th quarter of 2014 (http://www.health.gov.au/internet/main/publishing.nsf/Content/cda-cdi3902p.htm).

There were 41 cases of IPD reported in children aged under 5 years. The number of cases in this age group for this reporting period was 28% less than the 4th quarter of 2014 (n=57). Of those cases with known serotype, 24% (n=8) were due to a serotype included in either the 7vPCV or the 13vPCV compared with 40% (n=21) of cases in the 4th quarter of 2014 (Figure 1). For the 2015 calendar year, there was a small reduction in the total number of cases aged less than 5 years (n=198) compared with 2014 (n=215) and a small decline in the annual rate from 14 per 100,000 in 2014 to 13 per 100,000 in 2015. Serotypes 23B and 19A continued to be the most common serotypes affecting this age group, noting that 19A is included in the 13vPCV (Table 2).

In the 4th quarter of 2015, there were 7 cases reported in fully vaccinated children aged less than 5 years who were considered to be 13vPCV vaccine failures. For the 2015 calendar year, there were 44 13vPCV vaccine failures. Serotype 19A was reported as the cause of disease in 57% (n=4) of cases reported this period (Table 4) and 57% of vaccine failures in children aged less than 5 years this year.

Table 1: Notified cases of invasive pneumococcal disease, Australia, 1 October to 31 December 2015, by Indigenous status, serotype completeness and state or territory

Indigenous status	ACT	NSW	NT	Qld	SA	Tas.	Vic.	WA	Total qtr 4 2015	Total qtr 3 2015	Total qtr 4 2014	Year to date 2015
Indigenous	0	9	11	9	8	1	1	11	50	63	40	208
Non-Indigenous	2	78	1	39	23	12	55	22	232	453	271	1,120
Not stated/ unknown	0	10	0	0	0	0	24	0	34	73	41	171
Total	2	97	12	48	31	13	80	33	316	589	352	1,501
Indigenous status completeness* (%)	100	90	100	100	100	100	70	100	89	–	–	–
Serotype completeness† (%)	100	89	100	98	81	100	93	91	91	–	–	–

* Indigenous status completeness is defined as the reporting of a known Indigenous status, excluding the reporting of not stated or unknown Indigenous status.

† Serotype completeness is the proportion of all cases of invasive pneumococcal disease that were reported with a serotype or reported as non-typable. Serotype incompleteness may include when no isolate was available as diagnosis was by polymerase chain reaction and no molecular typing was attempted or was not possible due to insufficient genetic material; the isolate was not referred to the reference laboratory or was not viable; typing was pending at the time of reporting, or no serotype was reported by the notifying jurisdiction to the National Notifiable Diseases Surveillance System.

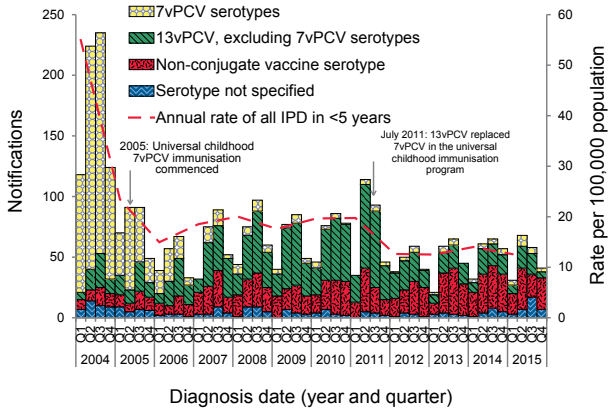
Table 2: Frequently notified serotypes of invasive pneumococcal disease, Australia, 1 October to 31 December 2015, by age group

Serotype	Age group			Serotype total*
	Under 5 years	5 to 64 years	Over 65 years	
19A	4	13	13	30
22F	2	14	12	28
3	1	17	10	28
19F	3	8	9	20
23A	3	12	5	20
7F	0	12	4	16
23B	4	5	5	14
11A	0	4	8	12
8	1	7	4	12
9N	0	7	5	12
15A	1	5	5	11
16F	2	2	6	10
35B	0	3	6	9
6C	2	3	3	8
33F	2	2	2	6
12F	1	4	0	5
15C	2	1	2	5
Other	6	22	15	43
Serotype unknown	7	14	6	27
Total	41	155	120	316

* Serotypes that only occur in less than 5 cases per quarter are grouped as Other and include non-typable samples this quarter.

† Serotype unknown includes those serotypes reported as no isolate, not referred, not viable, typing pending and untyped.

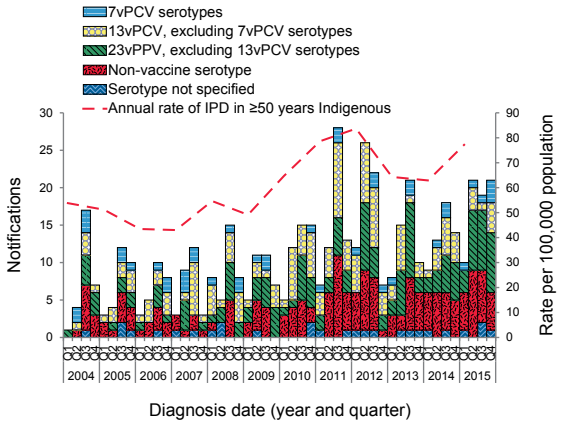
Figure 1: Notifications and annual rates* of invasive pneumococcal disease in children aged less than 5 years, Australia, 2004 to 2015, by vaccine serotype group



* Annual rates are shown on Q4.

There were 21 cases of IPD reported in Indigenous Australians aged 50 years or over. Of those cases with a reported serotype, 75% (n=15) were due to a serotype included in the 23-valent polysaccharide pneumococcal vaccine (23vPPV) (Figure 2). The

Figure 2: Notifications and annual rates* of all invasive pneumococcal disease in Indigenous Australians aged 50 years or over, Australia, 2004 to 2015, by vaccine serotype group



* Annual rates are shown on Q4.

Table 3: Notified cases of invasive pneumococcal disease, Australia, 1 October to 31 December 2015, by Indigenous status and age group

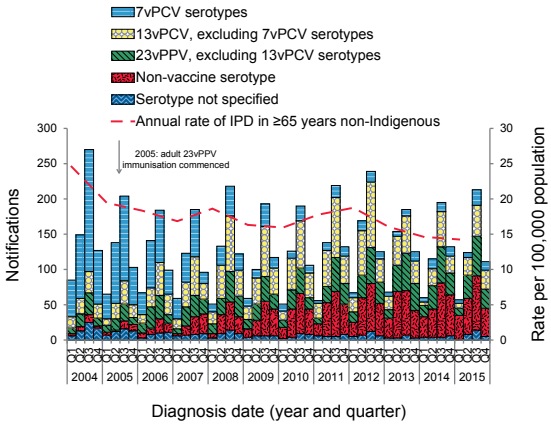
Age group	Indigenous status*			Total
	Indigenous	Non-Indigenous	Not reported	
0–4	12	28	1	41
5–9	4	4	2	10
10–14	0	1	1	2
15–19	0	2	1	3
20–24	1	1	4	6
25–29	1	4	1	6
30–34	2	3	4	9
35–39	1	4	9	14
40–44	3	9	4	16
45–49	5	6	5	16
50–54	6	13	0	19
55–59	5	14	0	19
60–64	2	32	1	35
65–69	5	26	1	32
70–74	1	26	0	27
75–79	2	23	0	25
80–84	0	17	0	17
85+	0	19	0	19
Total	50 (16%)	232 (73%)	34 (11%)	316

* Indigenous status completeness is defined as the reporting of a known Indigenous status, excluding the reporting of not stated or unknown Indigenous status.

number of notified cases of IPD in this age group was 10% higher than the previous quarter (n=19) and 43% more than the same quarter of 2014 (n=14). Compared with the previous quarter, the proportion of cases due to serotypes included in the 23vPPV increased markedly from 59% to 75% of cases with a reported serotype. During 2015, the annual rate increased to 77 per 100,000, a 22% increase from the 2014 rate of 63 per 100,000.

There were 111 cases of IPD reported in non-Indigenous Australians aged 65 years or over. Of those cases with a reported serotype, 62% (n=66) were due to a serotype included in the 23vPPV (Figure 3). The number of notified cases of IPD in this age group was 16% less than in the 4th quarter of 2014 (n=132) and 48% lower than the previous quarter (n=213). Compared with the previous quarter, the proportion of IPD due to 23vPPV serotypes increased slightly from 61% to 62% of cases with a reported serotype. In the 2015 calendar year, the annual rate was 14 per 100,000, a 44% reduction from the peak rate of 2004 (25 per 100,000 population) and a small reduction on 2014 (16 per 100,000).

Figure 3: Notifications and annual rates* of all invasive pneumococcal disease in non-Indigenous Australians aged 65 years or over, Australia, 2004 to 2015, by vaccine serotype group



* Annual rates are shown on Q4.

Table 4: Characteristics of 13vPCV failures in children aged less than 5 years, Australia, 1 October to 31 December 2015

Age	Indigenous status	Serotype	Clinical category	Risk factor/s
1 year	Non-Indigenous	19F	Pneumonia	No risk factor identified
1 year	Non-Indigenous	3	Pneumonia	No risk factor identified
2 years	Non-Indigenous	19A	Pneumonia	No risk factor identified
2 years	Non-Indigenous	19A	Pneumonia	Premature and other
3 years	Non-Indigenous	19A	Meningitis	No risk factor identified
2 years	Non-Indigenous	19A	Pneumonia	Childcare attendee and other
4 years	Non-Indigenous	19F	Meningitis	Unknown

In this quarter there were 19 deaths attributed to 14 different IPD serotypes. There were 2 deaths reported in children aged under 5 years that were associated with serotype 19A and 6C respectively.

Notes

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

In Australia, pneumococcal vaccination is recommended as part of routine immunisation for children, the medically at risk, and older Australians. More information on the scheduling of the pneumococcal vaccination can be found on the Immunise Australia Program web site (www.immunise.health.gov.au).

In this report, fully vaccinated describes cases that have completed the primary course of the relevant vaccine(s) required for their age according to the most recent edition of *The Australian Immunisation Handbook,* at least 2 weeks prior to disease onset with at least 28 days between doses of vaccine. NB: A young child who has had all the required doses for their age but is not old enough to have completed the primary course would not be classified as fully vaccinated.

There are 4 pneumococcal vaccines available in Australia, each targeting multiple serotypes (Table 5). Note that in this report serotype analysis is generally grouped according to vaccine composition.

Follow-up of all notified cases of IPD is undertaken in all states and territories except New South Wales and Victoria who conduct targeted follow-up of notified cases aged under 5 years, and 50 years or over for enhanced data.

Acknowledgements

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

Enhanced Invasive Pneumococcal Disease Surveillance Working Group contributors to this report include (in alphabetical order): David Coleman (Tas.), Heather Cook (NT and secretariat), Rachel de Kluyver (Health), Carolien Giele (WA), Robin Gilmour (NSW), Vicki Krause (Chair), Rob Menzies (NCIRS), Shahin Oftadeh (Centre for Infectious Diseases and Microbiology–Public Health, Westmead Hospital), Sue Reid (ACT), Stacey Rowe (Vic.), Vitali Sintchenko (Centre for Infectious Diseases and Microbiology–Public Health, Westmead Hospital), Helen Smith (Queensland Health Forensic and Scientific Services), Janet Strachan (Microbiological Diagnostic Unit, University of Melbourne), Cindy Toms (Health), Hannah Vogt (SA), Angela Wakefield (Qld).

Author details

Corresponding author: Dr Rachel de Kluyver, Vaccine Preventable Diseases Surveillance Section, Office of Health Protection, Australian Government Department of Health, GPO Box 9484, MDP 14, Canberra, ACT 2601. Telephone: +61 2 6289 1463. Facsimile: +61 2 6289 1070. Email: Rachel.de.kluyver@health.gov.au

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

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

Administration

Communicable Diseases Intelligence instructions for authors

Aims and objectives

Communicable Diseases Intelligence (CDI) is a peer-reviewed scientific journal published quarterly by the Office of Health Protection, Department of Health. The journal aims to disseminate information on the epidemiology, surveillance, prevention and control of communicable diseases of relevance to Australia. The objectives of CDI are to:

- report on surveillance of communicable diseases of relevance to Australia
- publish high quality original articles relevant to communicable disease epidemiology in Australia, and
- provide information on activities relevant to the surveillance, prevention and control of communicable disease in Australia.

Finding and accessing articles

CDI is listed on MEDLINE and indexed by PubMed, an online searchable index of published articles and authors. CDI is open access. All articles are published and made available free of charge. Subscribe to receive an email alert when new content is published. Subscription is open to anyone with an interest in public health.

CDI is published electronically only and is available in 2 formats from this website; Adobe Acrobat - Portable Document Format (.PDF) and HTML format.

CDI has been published by the Department of Health since 1976, initially as a fortnightly typewritten bulletin. CDI became a professionally printed publication in 1991. In mid-1992 parts of CDI became available electronically. The journal has been published quarterly since 2001. Hard copy printing ceased in 2011. CDI does not mail out hard copies of articles.

Submissions and article types

CDI encourages submissions consistent with the objectives from practitioners in all disciplines across the public health field. Advanced trainees and post graduate students are also encouraged to submit manuscripts. CDI publishes original articles, short reports, annual reports and quarterly reports, letters to the editor and editorials. Original articles and short reports are peer-reviewed. If you are interested in becoming a reviewer for CDI please email the CDI Editorial Team with your details and areas of expertise.

Manuscripts for submission

Manuscripts submitted to CDI must be offered exclusively to the journal. All manuscripts should be accompanied by a covering letter that should include:

- confirmation that the manuscript content (in part or in full) has not been submitted or published elsewhere; and
- whether the manuscript is being submitted as an article, short report, surveillance summary, outbreak report or case report.

In addition, manuscripts should include a title page that should contain the following information:

- title (e.g. Prof, Dr, Ms, Miss, Mrs, Mr), full name including middle initial, position held, and institution at the time the article was produced, of each author;
- name of corresponding author, including current postal address, telephone, and email; and
- word count of the main text and of the abstract.

On receipt of a manuscript, authors will be sent a brief acknowledgment. Accepted manuscripts are edited for style and clarity and final proofs are returned to the corresponding author for checking prior to publication.

Authorship

Authorship should be based on substantial contribution to the article. Each author should have participated sufficiently to take public responsibility for the article. Others contributing to the work should be recognised in the acknowledgments.

Types of manuscript

Original articles

The text of articles must be structured to contain an abstract, introduction, methods, results,

discussion, acknowledgments and references. Manuscripts submitted as articles must be 3,000 words or less and will be peer-reviewed.

Original articles may be submitted at any time and will be included in an issue once their review and revision has been completed. Articles may be published ahead of the scheduled issue, in the 'early release' format.

Systematic reviews submitted to CDI will be expected to conform to the Preferred Reporting Items for Systematic Reviews and Meta-Analyses (PRISMA) guidelines (http://www.prisma-statement.org/).

Letters to the Editor

The editorial team welcome comments on articles published in CDI in the form of letters to the Editor. Letters should normally be less than 500 words, include no more than a single chart and less than 6 references.

Short reports

Short reports may be submitted for peer review or for publication without peer review, depending on the content. Articles of particular relevance for rapid dissemination (such as timely outbreak reports) may be fast-tracked for early release prior to the next issue of CDI. Please discuss your requirements with the editorial team. Short reports may include an abstract. Types of short reports include:

Surveillance summaries

A report of 1,000 words or less that briefly reports on changes in the local epidemiology of a communicable disease, changes in surveillance systems, or new interventions, such as introducing vaccination in an at-risk group. Surveillance summaries should provide a brief description of the setting and a discussion of the significance of the events, changes or interventions.

Case reports

Brief reports of 500 to 1,000 words on cases of communicable disease will be considered based on their public health significance. Authors must note the instructions on the protection of patient's right to privacy (refer to the Ethics committee approvals and patients' right to privacy below). Some discussion of the significance of the case for communicable disease control should be included.

Outbreak reports

Reports of communicable disease outbreaks of 500 to 1,000 words will be considered for publication based on their public health significance. Reports should include details of the investigation, including results of interventions and the significance of the outbreak for public health practice. More comprehensive reports on outbreaks should be submitted as articles.

An outbreak report may be structured as below (the subheadings can be adjusted to suit), or may be unstructured if very brief.

Most outbreak reports will present only the descriptive epidemiology of the outbreak, with suspected risk factors for infection. The findings of any analytic study would usually be presented in an article at a later date, though authors may choose to present preliminary analyses from analytic studies.

Suggested structure

Abstract

A very brief unstructured abstract should be included

Background and methods

Including initial detection of the outbreak, case finding and interview techniques, study design and any statistical methods

Description of outbreak

Case definition, number of cases, number laboratory confirmed, symptoms. Time, place and person, epidemic curve

A maximum of 2 tables and/or figures is suggested.

Laboratory, trace back and environmental investigations

Details of the proportion of laboratory confirmation of cases.

Public health response

A very brief description of any actions taken to prevent further cases may be included.

Discussion

Including the significance of the outbreak for public health practice

References

A maximum of 20 references is suggested

Peer review process

Articles provisionally accepted for publication will undergo a peer review process and articles may be rejected without peer review. Short reports may be submitted for peer review, or may be reviewed at the discretion of the Editor. Articles will be subject to review by 2 experts in the field and short reports by 1 or 2 reviewers (if any).

When submitting your manuscript, you may specify reviewers who are qualified to referee the work, who are not close colleagues and who would not have a conflict of interest. Suggestions regarding reviewers will be considered, however, the Editor has the final decision as to who to invite to review a particular article.

Authors may be asked to revise articles as a result of the review process before the final decision about publication is made by the Editor. Revised articles are to be returned with a covering letter addressing each comment made by each reviewer.

Annual reports and quarterly reports are not subject to peer review.

Document preparation

Articles and reports must be written in clear, comprehensible English. Authors should pay particular attention to the style guides, web accessibility requirements and table and figure formatting requirements provided on these pages.

Articles are only accepted in electronic form, in Microsoft Word and Microsoft Excel. Graphics may be provided in a range of other formats (see section below on illustrations). In addition:

- Arial font is preferred but if not available use Times New Roman.
- Abstracts should not exceed 250 words. Do not cite references in abstracts.
- Structured abstracts are acceptable.
- Include up to 10 keywords.
- Avoid too many abbreviations.
- Use sentence case for all headings.

Manuscripts should be submitted with a 1 or 2 sentence summary of the article.

Tables

Tables and table headings should be located within the body of the manuscript and all tables should be referred to within the results section.

Information in tables should not be duplicated in the text.

Headings should be brief.

Simplify the information as much as possible, keeping the number of columns to a minimum and avoid merged cells as much as possible.

Separate rows or columns are to be used for each information type (e.g. percentage and number should be in separate columns rather than having one in parentheses in the same column).

If abbreviations are used these should be explained in a footnote.

Footnotes should use the following symbols in sequence:

* † ‡ § ¶ ** †† ‡‡, etc

Do not use blank rows or blank columns for spacing.

A short summary of each table should be included to satisfy government accessibility requirements (refer to Web accessibility requirements).

Figures and illustrations

Figures and illustrations, including headings, should be provided in the body of the manuscript and should be referred to within the results section. They should also be provided as a separate file.

Examples of each of the following can be found in the on-line version of Instructions to authors (http://www.health.gov.au/internet/wcms/publishing.nsf/Content/cda-pubs-cdi-auth_inst.htm)

A long text description should be included to satisfy government accessibility requirements (refer to Web accessibility requirements).

Figures

Use Microsoft Excel.

Each figure should be created as a separate worksheet rather than as an object in the datasheet (use the 'as new sheet' option for chart location).

The numerical data used to create each figure must be included on a separate worksheet (see example on the Department of Health web site).

Worksheets should be appropriately titled to distinguish each graph (e.g. Figure 1, Figure 2; Figure 1 data, Figure 2 data).

Do not include the graph heading on the Excel worksheet.

Graphs should be formatted to CDI requirements as much as possible. These requirements are available on the Health web site (http://www.health.gov.au/internet/main/publishing.nsf/Content/cda-pubs-cdi-auth_excel_fig.htm).

Illustrations

Illustrations or flow charts can be included if required.

Images should preferably be at least 300 dpi.

Electronic copies of computer-generated illustrations should preferably be saved in a vector image program such as Adobe Illustrator or other similar graphic but charts created in either Word or PowerPoint are acceptable. Use a sans serif font for figures (e.g. Arial). Symbols, lettering and numbering should be clear and large enough to be legible when reduced in size.

Photographs

Photographs may be submitted if required.

Photos need to be at least 300 dpi.

Electronic copies should be saved in Adobe Photoshop, or similar graphic software in one of the following graphic formats (in preferential order):

- PSD
- TIFF
- EPS
- JPEG (JPG).

Maps

Maps created by mapping programs such as MapInfo or ArcGIS should be saved at 300 dpi and in one of the following graphic formats (in preferential order) to allow editing of font size and colours:

- AI
- EMF

If this is not possible the following graphic formats should be used (in preferential order):

- TIFF
- EPS
- GIF.

Other images

Other images may be submitted in one of the following graphic formats (in preferential order):

- PSD
- TIFF
- EPS, or
- GIF.

Authors should aim for maximum levels of contrast between shaded areas. Use a sans serif font for text. Symbols, lettering and numbering should be clear and large enough to be legible when reduced in size.

Web accessibility requirements

The Australian Government is required to meet level AA of the Web Content Accessibility Guidelines version 2.0 (WCAG 2.0). These guidelines include the need for alternate methods of presenting the information depicted in images—including figures and maps—for readers with vision impairment and other disabilities using text readers. Complex tables also present challenges for text readers.

Articles and reports should be submitted with:

- a short summary of any tables
- a long text description of any figures;
- a long text description of any maps, flowcharts, or other images. For thermal maps showing disease rates by statistical location, a data table may be a preferred alternative.

Keep in mind that the description should be sufficient for a sight impaired person to understand what the information image is trying to convey,

Samples of descriptors for tables and figures can be found here on the Health web site (http://www.health.gov.au/internet/main/publishing.nsf/Content/cda-pubs-cdi-auth_web.htm).

Further information about WCAG 2.0 is available from the Australian Government Information Management Office (http://agimo.gov.au/)

References

References should be identified consecutively in the text using the Vancouver reference style. Any punctuation should precede the reference indicators.

Abbreviate journal names as in the PubMed journal database (http://www.ncbi.nlm.nih.gov/entrez/query.fcgi?db=journals) (e.g. Commun Dis Intell). Include the surnames and initials of all authors (or only the first 6 authors, et al, if there are more than 6). Cite the first and last page numbers in full, and specify the type of reference (e.g. letter, editorial).

Examples of the Vancouver reference style are available on the Health web site. See also the International Committee of Medical Journal Editors Uniform requirements for manuscripts submitted to biomedical journals Ann Intern Med 1997;1126:36–47 (http://www.nlm.nih.gov/bsd/uniform_requirements.html).

Cite personal communications and unpublished papers in the text, not in the reference list, with the exception of material that has been accepted for publication (in press). Obtain written permission from people cited, and include their title, position and affiliation.

The accuracy of references is the responsibility of authors.

Ethics committee approvals and patients' rights to privacy

All investigations on human subjects must include a statement that the subjects gave their written informed consent, unless data collection was covered by public health legislation or similar studies have been considered by a relevant ethics committee and a decision made that its approval was not required. The name of the ethics committee that gave approval for the study should be included in the text. Alternatively, if approval is not required a statement to this effect should appear in the manuscript.

Ethical approval and patient consent may also be required for case reports. Identifying details about patients should be omitted if they are not essential, but data should never be altered or falsified in an attempt to attain anonymity.

Copyright

All authors are asked to transfer copyright to the Commonwealth before publication. A copyright form will be sent to the corresponding author. All authors are required to sign the copyright release. The Commonwealth copyright will be rescinded if the article is not accepted for publication.

Submission of manuscripts

Manuscripts should be provided electronically by email to: cdi.editor@health.gov.au

Please contact the editorial team at cdi.editor@health.gov.au if you require any further information.

March 2016.