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Original article

Was infectious syphilis being misclassified in remote Australian outbreaks? Evidence that informed modification of the national case definition

Annie Preston-Thomas, Nathan Ryder, Sonia Harmen, Patricia Fagan

Abstract

Objective: To assess the ability of the national case definition to identify infectious syphilis during an outbreak affecting predominantly Aboriginal and Torres Strait Islander people in a remote Australian region.

Methods: A retrospective case series study of all non-congenital syphilis cases in the region notified between 1 January 2009 and 31 December 2012 was performed. The national infectious syphilis case definition was compared with an expanded case definition derived from experienced clinician assessment and the definition proposed in the *Interim Guidelines for the Public Health Management of Syphilis Outbreaks in Remote Populations in Australia* from the Communicable Diseases Network Australia (CDNA).

Results: Two hundred and forty syphilis cases were notified, of which 44 (18.3%) were symptomatic. The national case definition classified 106 (44.2%) cases as infectious, compared with 182 (75.8%) using the clinician-derived expanded case definition and 165 (68.8%) by the interim guidelines case definition. Seven confirmed and 6 probable cases were diagnosed as a result of contact tracing of probable infectious cases identified using the expanded case definition.

Conclusions and implications: The national case definition for infectious syphilis applied in this remote Australian outbreak underestimated infectious cases when compared with experienced clinicians' evaluation by up to 76 cases (42%) and was inadequate to monitor the magnitude of a syphilis outbreak in such a setting. This may compromise surveillance and resource allocation decisions, and could reduce the capacity to interrupt transmission and contain an outbreak. A revised national case definition, informed by this analysis, was released by CDNA in July 2015. *Commun Dis Intell* 2015;39(4):E571–E577.

Keywords: infectious syphilis; case definition; Aboriginal health

Introduction

Syphilis is a sexually transmissible infection (STI) of public health importance due to the significant perinatal morbidity and mortality caused when it is acquired in pregnancy,[1] the increased potential for HIV transmission,[2] the immediate impacts on the individual—symptoms, shame, stigma—and the risk of transmission to others. The natural history of syphilis is complex. Primary, secondary and tertiary stages of the infection have clinical manifestations and patients with the first 2 stages are infectious. There are also asymptomatic stages (early latent and late latent syphilis) detectable only by serology. The classification of an asymptomatic person with positive serology as early (infectious) or late (non-infectious) latent disease depends on the interpretation of previous serology, clinical and treatment history and, arguably, epidemiological context. A person with late latent syphilis remains at risk of progression to tertiary disease, but is not considered infectious.

The classification of the stage of infection has clinical, public health and surveillance implications.[3] Treatment decisions are based on the stage of infection. To prevent tertiary syphilis, an infectious case requires 1 dose of 1.8 gm intromuscular (IM) benzathine penicillin, whereas late latent disease requires 3 weekly doses.[4] From a public health perspective, the priority is to identify all potentially infectious cases so they and their sexual contacts can be treated and to stop transmission. National surveillance monitors syphilis epidemiology to help inform policy and prevention strategies and to facilitate disease control. For surveillance, data collected must be reliable and consistent.

Syphilis is a legally notifiable condition in every state and territory in Australia and nationally. The Communicable Diseases Network Australia (CDNA) periodically reviews the national case definition for infectious syphilis. Prior to July 2015, this definition included symptomatic primary and secondary cases and asymptomatic cases for

which there was definitive evidence of serological conversion within the previous 2 years (early latent syphilis) (Table 1).[5] Only cases with unequivocal proof of recent infection were counted as infectious (i.e. the definition was highly specific). Asymptomatic cases for which there was no syphilis serology documented within the preceding 2 years were classified as 'syphilis of more than 2 years duration or unspecified duration' (late latent/unknown duration) even in the presence of compelling clinical or epidemiological evidence suggesting recent infection. The reliance on dated serological testing techniques in combination with relatively non-specific clinical information to confirm cases results in imperfect public health case definitions compared with most other notifiable diseases. Furthermore, while it is known that intrinsic assay variability requires rapid plasma reagin (RPR) titres to be tested in parallel with previous specimens from the same laboratory, in most Australian settings laboratories are unable to perform this, as they no longer retain specimens.

There is no gold standard for the diagnosis of asymptomatic infectious syphilis. Case definitions from America, New Zealand and Europe include consideration of clinical history and/or an epidemiological link with an infectious case.[6–8]

Despite a general decline in syphilis notifications in the Australian Aboriginal and Torres Strait Islander population over recent decades,[9] sporadic outbreaks have continued to occur in remote settings.[10] From late 2010, the number of cases of syphilis notified from a single health district in remote North Queensland increased dramatically, predominantly affecting young Aboriginal and Torres Strait Islander people (Queensland Health, Statewide Syphilis Surveillance System, unpublished data). During the outbreak investigation a considerable number of cases had evidence suggesting recent syphilis acquisition but could not be classified as 'infectious' using the national case definition, being asymptomatic and without recent prior syphilis serology. If the national case definition alone were relied on to determine the magnitude of the outbreak and need for contact tracing, understanding of the extent of public health response required and interruption of disease transmission would have been jeopardised. In recognition of this, an additional classification category 'probable infectious syphilis' was proposed in the CDNA *Interim Guidelines for the Public Health Management of Syphilis Outbreaks in Remote Populations in Australia* (Interim Guidelines), released in February 2014 (Table 1). This new category includes consideration of additional epidemiological and case history information.[11]

This study assessed the extent to which potentially infectious syphilis cases may have been misclassified as non-infectious during the first years of this outbreak by comparing the number of cases classified as infectious using the national case definition with 1. an expert clinician-derived expanded case definition that takes into account clinical and epidemiological factors, and 2. the Interim Guidelines case definition.[11]

Methods

Demographic, clinical and laboratory characteristics of all non-congenital cases of syphilis diagnosed in a north Queensland district from 1 January 2009 to 31 December 2012 were extracted from the Queensland Health state-wide syphilis surveillance system database. Data assessed were age, gender, Indigenous status, clinical history, epidemiological link with a confirmed case, reported gender of sexual partner(s), previous serology and RPR titre. Data had been collected in the course of routine enhanced surveillance. All cases met criteria for notification with either positive syphilis serology and/or a positive nucleic acid amplification test.

The cases were independently assessed by 2 clinicians (PF and NR) experienced in syphilis diagnosis. The clinicians classified each case as either infectious, probable infectious or non-infectious taking all factors into account. Where a discrepancy existed between the clinicians, available data were clarified and they reviewed their original classification. The extent to which the clinicians' classifications were concordant was analysed using Cohen's kappa coefficient. The first author then reviewed the cases classified as 'probable infectious' by the clinicians, and thus derived the expanded case definition outlined in Table 1.

A review was conducted of the case classifications (infectious, probable infectious or non-infectious syphilis) resulting from the application of the national case definition for infectious syphilis; the clinician-derived expanded case definition, and the Interim Guidelines case definition.

The absence of a gold standard for the diagnosis of infectious cases prevents identification of 'true positives', precluding statistical analysis of the positive or negative predictive value of the case definitions. A comparative analysis of the proportion of cases identified as infectious by the 3 case definitions was conducted. The number of additional contacts and positive contacts identified using the expanded case definition was assessed.

The research project, performed on de-identified notifiable condition data for clinical audit and quality improvement, was exempt from ethical review.

Table 1: Case definitions for infectious syphilis comparing the national case definition; expanded case definition derived from experienced clinician assessment and Interim Guidelines case definition

Case definition	National- less than 2 years duration	Expanded case definition	Interim guidelines
Confirmed infectious cases (primary, secondary or early latent syphilis)	1. Laboratory definitive evidence* **OR** 2. Laboratory suggestive evidence AND clinical evidence†		
Probable infectious cases	Not Applicable	Does not meet the criteria for infectious syphilis (primary, secondary or of less than 2 years duration) **AND** A. RPR 1:128 or higher **OR** B. RPR 1:8 or higher **AND** the case satisfies one of the following: 1. Aboriginal and/or Torres Strait Islander and age less than 30 years at the time of testing **OR** 2. A contact of a case of infectious syphilis‡ **OR** C. Aboriginal and/or Torres Strait Islander and age less than 20 years, in a community with an infectious syphilis outbreak and suggestive clinical history or epidemiological link with outbreak	Does not meet the criteria for infectious syphilis (primary, secondary or of less than 2 years duration) **AND** RPR 1:32 or higher **AND** the case satisfies one of the following: 1. Aboriginal and/or Torres Strait Islander and age less than 30 years at the time of testing **OR** 2. A man of any age who has sex with men **OR** 3. A contact of a case of infectious syphilis

* Laboratory definitive evidence is defined by either seroconversion in the past two years (reactive specific test confirmed by either reactive non-specific or a different specific treponemal test) or a 4-fold or greater rise in rapid plasma reagin (RPR), with confirmation of positive results by a reactive specific treponemal test.

† Laboratory suggestive evidence includes *Treponema pallidum* identification by microscopy (darkfield, fluorescent antibody or equivalent) or nucleic acid testing, or a combination of reactive specific and non-specific serological tests. Clinical evidence is defined as 'presence of a primary chancre (or ulcer) or clinical signs of secondary syphilis'.

‡ Defined as: contact with a case of confirmed primary, secondary or early latent syphilis in the 12 months before, or 3 months after diagnosis.

Results

Demographics

Two hundred and forty cases of non-congenital syphilis were notified in the district during 2009–2012. Case demographics are shown in Table 2. None were co-infected with HIV. The median age of the 44 symptomatic cases was 21 years, range 12–46 years. Thirty-six (81.8%) of these were aged less than 30 years.

Infectious syphilis

Table 3 displays the classification of syphilis cases by year, comparing the number of cases classified as infectious by the 3 case definitions.

With regard to infectious status, there was 97.8% agreement, kappa coefficient k=0.953 (CI 0.91–0.99) between the clinician assessors. For four of the 240 cases (2.2% of the infectious cases as classified by the expanded case definition) the clinicians were unable to reach consensus. All 4 cases were Indigenous, aged 18–21 years and with RPR titres of 1:2, 1:4, 1:8 and non-reactive, respectively. None were known contacts of infectious syphilis cases. These were classified as non-infectious.

The national case definition classified 106 (44.2%) of the 240 cases as infectious, compared with 182 (75.8%) by the expanded case definition (Table 3), i.e. 76 additional probable infectious cases were identified. These cases named 141 contacts, of whom 111 were successfully followed

Table 2: Demographic characteristics of all non-congenital syphilis cases compared with infectious syphilis cases defined by national case definition in a north Queensland district, 2009 to 2012

	All cases of syphilis	Per cent of all cases	Cases of infectious syphilis	Per cent of infectious cases
Number of cases	240		106	
Females	135	56.3	61	57.5
Males	105	43.7	45	42.5
Indigenous status:				
Aboriginal and/or Torres Strait Islander	232	96.7	104	98.1
Non-Indigenous	8	3.3	2	1.9
Median age	21		20	
Aged less than 30 years	174	72.5	92	86.8
Age range (years)	12–84		12–59	
Transmission (self-report):				
Heterosexual	210	87.5	103	97.2
Homosexual	1	0.4	1	0.9
Not stated	29	12.1	2	1.9
Primary or secondary (symptomatic) infection	44	18.3	44	41.5

Table 3: Non-congenital syphilis notifications in a district of north Queensland, 2009 to 2012, and number of cases of infectious and non-infectious syphilis classified by the national case definition, expanded case definition and Interim Guidelines case definition

Year	Total notifications of syphilis	Classification by national case definition		Classification by expanded case definition		Classification by Interim Guidelines case definition	
		Infectious	Non-infectious*	Infectious and probable infectious	Non-infectious	Infectious and probable infectious	Non-infectious
2009	22	1	21	2	20	2	20
2010	21	15	6	16	5	16	5
2011	99	44	55	85	14	74	25
2012	98	46	52	79	19	73	25
Total	240	106	134	182	58	165	75

* Case definition includes cases of unknown duration.

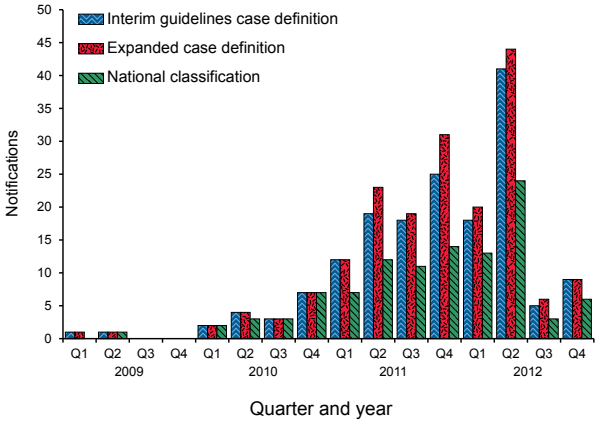
up. Seven of the confirmed and six of the probable cases of infectious syphilis included in our analysis had been identified as a consequence of contact tracing of the probable infectious cases. Demographics of the additional 76 probable infectious cases were similar to, though slightly younger than the infectious cases identified by national criteria. All 76 cases were Indigenous, 49 (64.5%) female, median age 18 years, range 12–37 years; 73 (96.1%) were aged less than 30 years. Twenty had an epidemiological link with a confirmed case of infectious syphilis.

Applying the Interim Guidelines criteria classified 59 cases as probable infectious syphilis, in addition to those confirmed as infectious cases using the national definition. All of the cases identified using this case definition had also been classified as probable infectious syphilis by the expanded case definition. The Figure compares the epidemic curve of infectious syphilis over time in the district utilising the three classification methods.

Discussion

This large outbreak has provided a valuable opportunity to examine the implications of infectious syphilis case definitions for public health surveillance and response in a real world situation. This study demonstrated that the national case

Figure: Epidemic curve of infectious syphilis in a north Queensland district, 2009 to 2012, by national definition, expanded case definition and Interim Guidelines case definition



definition for infectious syphilis underestimated infectious cases by up to 42% when compared with an expert clinician-derived expanded case definition. Application of the expanded case definition and the resultant increased number of contacts traced in this setting resulted in the identification of a further 13 probable or confirmed infectious cases. This highlights that adherence to the narrow national case definition could have implications for outbreak control if these additional cases were not assessed and treated. Applying the Interim Guidelines case definition identified 59 of the 76 cases of probable infectious syphilis that were considered probably infectious by the clinicians and did not result in any clinician classified non-infectious cases being identified as probably infectious.

Use of the probable infectious syphilis category would not have resulted in earlier identification of this outbreak; the divergence in the number of cases identified as infectious by use of the expanded case definition became apparent in the 1st quarter of 2011, after the outbreak was established. There may still be implications for the timeliness of an outbreak response. Missed identification opportunities and delays in STI outbreak response in this remote setting have significant consequences given the sexual and reproductive health vulnerability of this population, evidenced by the excess burden of chlamydia and gonorrhoea,[9] and the on-going risk of HIV/AIDS in remote Australia.

The national case definition for infectious syphilis requires clinical symptoms and/or baseline laboratory evidence to confirm a recent infection. If a person is asymptomatic and has not had syphilis serology performed in the previous 2 years, as is the case for many young Aboriginal and Torres Strait Islander people, they cannot be classified as infectious, even in the context of an established outbreak, a high RPR titre, or a clear epidemiological link to an infectious case. In our study, 55 probable infectious cases had never been tested previously for syphilis, and 21 probable infectious cases had previous serology performed more than 2 years ago. Under the national case definition, a change in the amount of syphilis testing performed could affect the number of cases classified as infectious, purely because of a change in the number of people with a baseline serology result.

There are anecdotal reports that the specificity of the national case definition for infectious syphilis in Australia has led to dissonance among public health professionals who classify cases, resulting in an inconsistent approach within and across jurisdictions. This could have an impact on the consistency of national data, as in the American context.[3] One American study found that approximately half of notified early latent cases did not meet the national case definition, and different jurisdictions had developed their own case definitions. They were applying these case definitions to the nationally reported notifications, resulting in inconsistent data that could not be aggregated.[3] Additionally, loosely defined criteria resulted in inconsistent misclassification within the jurisdiction. To our knowledge, no such study has been performed in Australia.

Establishing a 'probable infectious syphilis' category is one mechanism to clarify management of affected individuals, optimise outbreak control and at the same time preserve national data consistency. Other surveillance programs have definitions that include consideration of clinical and/or epidemiological information. The American case definition for 'probable early latent syphilis' includes asymptomatic cases with evidence of recent serological conversion, but also allows for cases with laboratory evidence of syphilis and 1. a clinical history of primary or secondary syphilis symptoms in the previous 12 months, 2. an epidemiological link to a partner with infectious syphilis within the previous 12 months, or 3. a history of sexual debut within the last 12 months.[6] However, the reliance on recall of symptoms and clinician interpretation of such findings has been criticised as difficult to standardise.[12] The European Centre for Disease Prevention and Control classification of 'probable early latent syphilis' is defined as a person with clinical criteria and an epidemiological link.[8] The New Zealand case definition for early latent syphilis includes a clear history of primary or secondary syphilis symptoms within the previous 2 years or sexual contact with a confirmed case of infectious syphilis within the previous 2 years.[7]

The Interim Guidelines 'probable infectious syphilis' case definition relies on a RPR of ≥ 1:32 and allows consideration of an epidemiological link. The inclusion of cases due to ethnicity, age or sexual orientation may warrant further refinement, as these criteria bias case classification towards groups currently experiencing higher rates of syphilis and potentially amplify this rate differential because the ability to detect infectious cases in other populations would not be similarly enhanced.

RPR titre has been proposed for use in early latent syphilis case definitions, based on pre-treatment titre or rapid decrease in response to treatment, suggestive of early disease.[12] Although high RPR titres are strongly associated with recent infection,[13] there is no specific level of RPR titre that can reliably distinguish between early latent and late latent syphilis in asymptomatic people. One study found considerable overlap in RPR titre distributions between stages of disease,[12] making the choice of a threshold titre for disease staging problematic. However, the authors concluded that in asymptomatic patients, RPR titre could provide a more objective and reliable record of syphilis trends compared with a system based on inconsistently applied definitions. We assessed the number of probable infectious cases that would be identified using the Interim Guidelines case definition, but with an RPR titre cut-off of 1:8, instead of 1:32. This resulted in an additional 12 probable infectious cases (in total, 92% of the probable infectious cases by the expanded case definition), however it also captured 2 cases as 'probable infectious' that were assessed to be non-infectious by the clinicians.

The strength of the agreement between the clinicians in this study was very good. The clinician assessors made their classifications independently of each other based on data provided by the statewide surveillance system.

Limitations

There is no gold standard for the diagnosis of asymptomatic infectious syphilis, which precludes accurate assessment of the validity of the proposed probable infectious syphilis case definition. In lieu of this we employed an expert clinician derived expanded case definition that took account of the clinical, laboratory and epidemiological information of all of the cases as a 'proxy gold standard'.

This study is based on a retrospective analysis of cases, however enhanced surveillance at the time of notification resulted in comprehensive clinical information being obtained. The 2 clinician assessors independently agreed on all but 4 cases. One of the clinicians (PF) was the author of the Interim Guidelines and the other (NR) was a member of the reference group for the project. It is possible that this could bias the concordance of the clinician consensus with the Interim Guidelines definition. However, the number of clinicians with experience in this area is limited, and most of those with relevant expertise would most likely have been consulted during the guideline development.

In Australia, there are 2 groups among whom the majority of syphilis cases are diagnosed; men who have sex with men (MSM) and predominantly heterosexual transmission in young, regional and remote-living Indigenous people.[9] The criterion in the Interim Guidelines case definition relating to MSM is largely redundant in the remote Australian setting. It is likely that similar issues as identified here relating to the adequacy of case definitions operate in the context of MSM, however it is unclear to what extent these findings would be applicable in consideration of case definitions for MSM. This may increase the challenge in developing appropriate case definitions. As observed in Peterman's study,[3] criteria that cases met to qualify as early latent syphilis varied by site, and these variations may be more extreme when considering the vastly different life circumstances of the 2 population groups mainly affected by syphilis in Australia.

Conclusion

The national case definition for infectious syphilis identified only cases with definite evidence of recent infection, and did not capture many cases with strong clinical or epidemiological evidence suggesting recent infection. This study does not claim to resolve the conundrum of a perfect infectious syphilis case definition, but demonstrates that in the context of an outbreak in a remote Indigenous setting, a considerable number of likely infectious cases are not captured. This can result in an inability to adequately monitor and contain outbreaks and an underestimate of their size, with implications for resourcing an adequate public health response. There are also implications for the reliability of national surveillance data. This study assessed the utility of an additional 'probable infectious syphilis' classification, which performed well in this setting. The national case definition for infectious syphilis should be revised to improve monitoring of this serious public health issue.

At the time this article was undergoing peer review, CDNA released a revised infectious syphilis case definition, in part informed by this study, incorporating a 'probable infectious' category.[14] Applying the new CDNA case definition to this dataset identified 67 of the 76 probable infectious cases as classified by the expanded case definition.

The authors believe the revised definition will better reflect the true extent of an outbreak in remote Indigenous settings, and commend this revision.

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References

1. De Santis M, De Luca C, Mappa I, Spagnuolo T, Licameli A, Straface G, et al. Syphilis infection during pregnancy: fetal risks and clinical management. *Infect Dis Obstet Gynecol*. 2012; doi: 10.1155/2012/430585. PMCID: PMC3398589.
2. French P. Syphilis. *BMJ* 2007:334:143–147.
3. Peterman TA, Kahn RH, Ciesielski CA, Ortiz-Rios E, Furness BW, Blank S, et al. Misclassification of the stages of syphilis: implications for surveillance. *Sex Transm Dis* 2005; 32 (3):144–149.
4. Antibiotic Expert Groups. Therapeutic Guidelines: antibiotic. Version 15. Melbourne: Therapeutic Guidelines Limited; 2014.
5. Communicable Diseases Network Australia. Surveillance Case Definitions for the Australian National Notifiable Diseases Surveillance System. 1 January 2004 to 1 January 2014. Updated July 2014; accessed on 20 October 2014. Available from: http://www.health.gov.au/internet/main/publishing.nsf/Content/cdna-casedefinitions.htm
6. Centers for Disease Control and Prevention. STD Surveillance Case Definitions. Updated 10 December 2013; accessed on 19 August 2014. Available from: http://www.cdc.gov/std/stats/CaseDefinitions-2014.pdf
7. The Institute of Environmental Science and Research Ltd. Enhanced surveillance of infections syphilis in New Zealand sexual health clinics – 2013. Porirua, New Zealand. Accessed on 13 February 2015. Available from: https://surv.esr.cri.nz/PDF_surveillance/STIEnhancedsyphilis/EnhancedSyphilis2013.pdf
8. European Centre for Disease Prevention and Control. Case definition: syphilis. Accessed on 29 January 2015. Available from: http://www.ecdc.europa.eu/en/activities/surveillance/sti/Pages/Case%20definition.aspx
9. The Kirby Institute. *Bloodborne Viral and Sexually Transmitted Infections in Aboriginal and Torres Strait Islander People: Surveillance and Evaluation Report 2014*. Sydney: The Kirby Institute, University of New South Wales; 2014. Accessed on 17 March 2015. Available from: http://kirby.unsw.edu.au/surveillance/2014-aboriginal-surveillance-report-hiv-viral-hepatitis-stis
10. Ward JS, Guy RJ, Akre SP, Middleton MG, Giele CM, Su JY, 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.
11. Communicable Diseases Network Australia. *Interim Guidelines for the Public Health Management of Syphilis Outbreaks in Remote Populations in Australia*. Accessed on 20 October 2014. Available from: http://webarchive.nla.gov.au/gov/20150622155130/http://www.health.gov.au/internet/main/publishing.nsf/Content/cdna-song-syphilis.htm
12. Samoff E, Koumans EH, Gibson JJ, Ross M, Markowitz LE. Pre-treatment syphilis titers: distribution and evaluation of their use to distinguish early from late latent syphilis and to prioritize contact investigations. *Sex Transm Dis* 2009;36(12):789–793.
13. Hart G. Syphilis tests in diagnostic and therapeutic decision making. *Ann Intern Med* 1986;104:368–376.
14. Communicable Disease Network of Australia. Syphilis – infectious (primary, secondary and early latent), less than 2 years duration case definition. Accessed on 16 July 2015. Available from: http://www.health.gov.au/internet/main/publishing.nsf/Content/cda-surveil-nndss-casedefs-cd_syphl2.htm

Treatment of latent tuberculosis in migrants to Victoria

Michael G Flynn, Lynne K Brown

Abstract

The proportion of eligible persons identified who are tested for latent tuberculosis (TB), offered treatment, and complete treatment are performance indicators in tuberculosis control. We report a retrospective database review of the Migrant Screening Clinic, Department of Respiratory and Sleep Disorders Medicine at Western Health Footscray Hospital during the years 1996–2006. Of 7,225 migrants aged less than 35 years, tuberculin skin testing (TST) was performed for 3,589 (49.7%), including 2,641 (65.6%) of 4,024 migrants under 35 years with an abnormal chest radiograph, and 2,297 (59.0%) of 3,893 migrants born in a high-burden country. Of 3,589 persons with both chest radiograph and TST results, 1,487 (41.4%) were referred for follow-up, including 81.3% of those with TST ≥10 mm. Outcome data were available for 1,047 persons considered for treatment of latent TB, of whom 12.5% did not attend an initial appointment, 21.6% attended and were not offered treatment, 65.9% attended and were offered treatment, and 41.7% completed treatment for latent TB. The Victorian program for treatment of latent TB in migrants has testing, treatment offer and treatment completions rates similar to other published studies. The impact on TB control is limited by the small proportion of migrants referred to this program. *Commun Dis Intell* 2015;39(4):E578–E583.

Keywords: screening; tuberculin; latent tuberculosis, treatment completion rate

Introduction

The incidence of notified tuberculosis (TB) in Australia was 5.5 per 100,000 persons per year in 2013.[1] In Victoria, the incidence of notified TB was 6.7 per 100,000; and 88% of notified cases occurred in persons born overseas.[1] Notification of active TB in Victoria is mandatory under the *Public Health and Wellbeing Act 2008*.[2]

In 2009, Australia issued 186,421 permanent visas. Of 4.0 million temporary entry visas, 3.3 million were visitors and 320,368 were overseas students.[3] In the same year Victoria received 24.5% of all permanent visa applicants, 18.3% of all visitors and 33.1% of students.[3] The top 3 countries of birth for permanent additions to the Victorian population were India, Peoples' Republic of China, and the United Kingdom. Overseas students in Victoria increased from 13,500 in 1993 to 46,401 in 2004 and 117,711 in 2009.[3,4]

People who want to migrate permanently to Australia, or stay temporarily, must satisfy the health requirement specified in the Migration Regulations of the Australian Government.[5–8] Applicants for permanent entry are asked to undergo a medical examination, and chest radiograph if 11 or more years of age. Where x-rays show possible evidence of TB the applicant is asked to provide sputum for smear microscopy and culture, and may be asked to provide serial chest radiographs over 3–6 months. If active TB is found, Australian migration law does not allow a visa to be granted until the person has undergone treatment and been declared free of active TB. This is documented with repeat chest radiograph and sputum examination. Any required medical examinations and x-rays must be conducted by qualified doctors and radiologists nominated by the immigration department.[8] If a chest radiograph shows evidence of previous but now inactive TB the applicant may be asked to sign a Tuberculosis Health Undertaking (TBU) at the time of visa grant.[6] By signing the TBU, the applicant agrees to contact the Health Undertaking Service on a free call number on arrival in Australia. The applicant also agrees to report to a state or territory health authority for follow-up as directed by the Health Undertaking Service.[5,7] Applicants for temporary entry may be asked to undergo a chest x-ray, medical examination, and possibly further tests including sputum examination and repeat chest x-ray, depending on the length of intended stay in Australia, intended activity, and the TB risk rating of the applicant's country.[8] TBUs originate from visa applications lodged outside Australia (off-shore TBU), or inside Australia (on-shore TBU). Both off-shore and on-shore TBUs include permanent and temporary residency immigrants, refugees and other humanitarian entrants.[9]

Under arrangements with the Department of Health Victoria, the Department of Respiratory and Sleep Disorders Medicine at Western Health Footscray Hospital has provided post-migration screening for TB in Victoria since 1996. Migrants who have been issued an off-shore TBU or have an abnormal chest radiograph at the time of an on-shore visa application are referred to the Migrant

Screening Clinic (MSC). In this clinic the prevalence of active TB at entry is 505 per 100,000.[10] Migrants are offered a tuberculin skin test (TST) if under 35 years of age and the chest radiograph is abnormal, or if under 35 years of age and a refugee,[12] or if under 35 years of age and born in one of the high burden countries whose migrants have the highest TB incidence in Victoria.[13,14] The age of 35 years was chosen based on Australian guidelines current during the period under study.[11,15] The frequency and severity of isoniazid-associated hepatitis increase with age.[16]

The MSC is a screening service, seeing each person once, and referring persons who need further assessment to other health services. Persons with symptoms suggestive of TB, or with one or more chest radiographs suggestive of active TB, are defined as suspected active TB and referred to other specialist clinics. The Victorian Government TB program is advised of non-attendees with a chest radiograph suggestive of active TB; TB program nurses are asked to contact the person, and may make a home visit. Those without such features, but with a history of TB diagnosis, a positive TST, or one or more chest x-rays thought likely to represent previous TB, are defined as inactive TB. Migrants with a positive TST[17] are referred to specialist respiratory or infectious disease clinics for treatment.

The objective of this study was to determine from a retrospective review of the Migrant Screening database the proportion of eligible persons identified in the MSC during the years 1996–2006 who were tested for latent TB, offered treatment, and completed treatment.[18]

Methods

Study population

All persons under 35 years of age with TST and chest radiograph performed in the MSC during the period November 1996 to June 2006 were identified from a search of the Migrant Screening database. The information extracted included whether the chest radiograph was normal or abnormal, the result of the TST, and the outcome of referral to specialist clinics for treatment of latent TB. Persons notified to the Department of Health with active TB were excluded from the study population, as they were not eligible for treatment of latent infection. Descriptive summary statistics were used to report findings.

Ethics approval

The study was approved by the Human Research Ethics Committee of the Department of Health Victoria, and by the Human Research Ethics Committee of the Royal Melbourne Hospital Research Foundation.

Results

Between November 1996 and June 2006, 18,359 persons were given an appointment at the MSC, of whom 15,352 (83.6%) attended. Of the 7,225 attendees aged less than 35 years, 6,949 (96%) had a chest radiograph result recorded. TST results were available for 3,589 (49.7%) attendees including 2,641 (65.6%) of 4,024 migrants under 35 years with an abnormal chest radiograph, and 2,297 (59.0%) of 3,893 migrants under 35 years born in a high-burden country. Of 3,589 persons with both chest radiograph and TST results, 1,487 (41%) were referred for follow-up, including 81.3% of those with TST of 10 mm or more, and 93.9% of those with TST of 15 mm or more (Table 1).

Table 1: Percentage of persons tested who are referred for follow-up, by tuberculin skin test diameter

TST	Tested	Referred	
mm	n	n	%
0–4	1,490	70	4.7
5–9	415	48	11.6
10–14	431	192	44.5
≥15	1,253	1,177	93.9
Total	3,589	1,487	41.4

TST Tuberculin skin test.

Of those referred to specialist clinics, 36 were found to have active TB. Of the remaining 1,451 persons referred, 1,334 had TST ≥10 mm, of whom 1,047 (78.5%) had an outcome recorded for the referral. These 1,047 persons eligible for the treatment of latent TB comprised 576 males (55.0%), mean age of 26.4 years, a mean TST of 20.8 mm in diameter, and 77.6% had an abnormal chest x-ray. Most of these people (84.8%) were born in a country with a high TB incidence.[14] The top 5 countries of birth were China 19.4%, Vietnam 15.7%, India 11.8%, Indonesia 5.2% and the Philippines 3.7%. The outcome of referral is shown in Table 2. Of 1,047 persons referred, 916 attended an initial appointment, 690 were offered, 546 commenced, and 437 completed treatment.

Table 2: Outcomes of referral for treatment of latent tuberculosis

Outcome	n	%
Did not attend initial appointment	131	12.5
Attended initial appointment but treatment for latent TB not offered	226	21.6
Treatment of latent TB offered but patient refused	144	13.8
Started but failed to complete treatment for latent TB	109	10.4
Completed 6 months isoniazid	224	21.4
Completed 9 months isoniazid	205	19.6
Completed 2 months rifampicin + pyrazinamide or other treatment of latent TB	8	0.8
Total	1,047	100.0

Discussion

The National Tuberculosis Advisory Committee includes the screening of high risk groups for latent TB as a key activity in TB control.[19] In the United States of America the proportion of eligible persons identified who are tested for latent TB, offered treatment, and complete treatment are recommended performance indicators in tuberculosis control.[18]

This study found that the proportion of the target groups tested for latent TB was 65.6% in those with an abnormal chest radiograph, and 59.0% in those born in a high-burden country. At the MSC, migrants are advised that tuberculin skin testing is not required for immigration assessment and many decline the offer of TST.

Although 93.9% of persons with a TST ≥ 15 mm were referred for follow-up, only 44.5% of those with TST in the 10–14 mm range were referred. This likely relates to former Australian guidelines that in the presence of a history of previous bacille Calmette-Guérin (BCG) vaccination, tuberculin reactions of 15 mm or more may be regarded as an indication of possible super-infection with tuberculosis.[11] However, Marks and others found that the risk of TB in tuberculin positive refugees increases linearly with TST reaction size above 10 mm; the risk, and the relation of risk to TST reaction size, were unrelated to BCG scar status.[20] More recent Victorian guidelines[15] state that a single BCG vaccination in early childhood (before 5 years of age) is likely to be followed by a negative TST response when testing is performed after 10 years or more. Any reaction of 10 mm or greater in this setting is significant, and should not necessarily be attributed to BCG.[15]

A limitation of this study is that we did not perform interferon-gamma response assays (IGRA). One study of contact tracing has shown that an IGRA had a higher positive predictive value than TST.[21] Another study which included recently exposed immigrant close contacts found that the positive predictive value of IGRA for subsequent development of TB disease was comparable to that of the TST, irrespective of the TST cutoff (10 or 15 mm).[22]

Among persons referred for follow-up, 12.5% did not attend an initial appointment, 21.6% attended and were not offered treatment, 65.9% attended and were offered treatment, and 41.7% completed treatment for latent TB. Of the 916 persons attending a specialist clinic, 690 (75%) were offered treatment. This is consistent with large scale studies in which physicians did not recommend treatment to 20%–30% of patients who appeared eligible.[23] Many factors may have influenced these outcomes. Hart found that physicians' reasons for not offering treatment of latent infection to Victorian migrants with TST > 15 mm included previous adequate treatment, reactivation thought unlikely, and TST likely due to prior BCG.[24] Anibarro et al.[25] found that recent immigration (<5 years residence) and social risk factors including unemployment were independently associated with a lower rate of treatment completion. They considered that preventive measures may not constitute a major priority for newly arrived immigrants; furthermore, communication with health care providers is not always easy due to language barriers and socio-cultural reasons that make it difficult to convince otherwise healthy people of the importance of treatment; and that precarious employment situations also complicate regular attendance at follow-up visits.[25] Overseas visitors who hold temporary visas are

generally not eligible for Medicare to cover their medical and hospital expenses, and may not hold overseas visitor health cover.[26]

The completion rate in our study, expressed as the number completing treatment over those initiating treatment, was 437/546 (80%), which is in the upper range for treatment completion in foreign born persons and similar to other data from Australia.[27,28] Completion rates for the treatment of latent TB in Europe are considered unsatisfactory.[29] Rennie et al. found that offering patients a choice of treatment (6 months isoniazid or 3 months isoniazid and rifampicin) improved completion rates, with most patients preferring the shorter regimen.[30] Shorter regimens may have higher completion rates.[31] Supervised, clinic-based administration of treatment for latent TB may significantly reduce adherence.[32] Monthly house calls may increase completions rates.[33] Younger patients are less likely to complete treatment of latent infection.[34] A shorter treatment regimen may result in better adherence.[35]

Data from this and other studies can be used to estimate the contribution of treatment of latent infection in migrants on a TBU to tuberculosis control. The current study is of a highly selected group of recently arrived migrants: those who have been issued a TBU for an abnormal chest x-ray at the time of visa application. The incidence of TB in this group is 160 per 100,000,[10] which is much higher than the incidence in all overseas-born Victorians of 21.9 per 100,000.[36] During the 10 years 1996 to 2006, 18,359 migrants on a TBU were referred to the MSC. This was only a small fraction of the total number of migrants entering Victoria – approximately 46,000 permanent visa applicants in 2009.[3] It is not surprising that MacIntyre found only 25 of 218 (11.5%) notified cases in 1991 had been on a previous TBU.[37]

Active TB among contacts contributes relatively little to the burden of TB in Victoria. In the years 2005–2010, 4.1% of cases were linked to another notified case.[36] Among 783 contacts of 231 cases of active TB in 1991 there were 8 incident cases of active TB in the following 2 years.[38] Isoniazid prophylaxis did not significantly predict risk of disease; however, only a low number of individuals received prophylaxis.[38] Thus the potential benefit of treatment of latent infection in Victoria is mainly to the individual being treated, with little effect on future transmission.

Denholm et al.[39] argued that since the considerable majority of new diagnoses of active tuberculosis in Australia arise from reactivation of previously latent infection in those born in high-prevalence regions, the potential exists to reduce tuberculosis incidence through new public health policy aimed at the detection and treatment of latent infection in immigrants, thereby preventing reactivation. However, modelling by Denholm and McBryde[40] suggests that while broad immigration-related strategies targeting latent infection would be effective for reducing TB incidence, they are likely to be too inefficient for introduction as an across the board public health measure. They suggested strategies targeting immigrants from high-prevalence regions may be an effective and efficient public health intervention, which could be considered for TB incidence reduction in Australia. The MSC has conducted a program of targeted testing and referral for latent TB since 1996. The current study shows it has had a modest rate of treatment completion.

Any plan to extend testing of immigrants for latent TB would need to consider cost-effectiveness, resources and a number of ethical issues.[39,41,42] Denholm and McBryde reported significant variation in clinical practice relating to the diagnosis, treatment and management of latent TB.[43] This includes the likelihood of recommending and commencing therapy and the practices relating to clinical management during treatment. A chest radiograph with features suggestive of previous TB is a risk factor for subsequent active TB, and an indication not only to exclude currently active TB, but also to consider the place of treatment of latent infection in that individual.[17]

Denholm and McBryde argue that in order for treatment of latent TB infection to be an effective public health strategy, the development of, and regular updating of guidelines that are relevant, consistent and most importantly acceptable, across a broad range of stakeholders is an important step.[43] It is encouraging that 75% of the targeted group who were referred by the MSC and who attended specialist clinics for consideration of treatment of latent TB were offered treatment, but consensus on who should be offered treatment is essential before testing for latent TB is expanded in this population.

The Victorian program for preventive treatment of latent TB in migrants has testing, treatment offer and treatment completions rates similar to other published studies. Even if all migrants on a TBU were offered testing and treatment for latent infection, the reduction in active TB cases would be small, principally because only a small fraction of migrants entering Victoria have an abnormal chest radiograph and are placed on a TBU.

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References

1. Toms C, Stapledon R, Waring J, Douglas P, National Tuberculosis Advisory Committee. Tuberculosis notifications in Australia, 2012 and 2013. Accessed 23 June 2015. Available from: http://www.health.gov.au/internet/main/publishing.nsf/Content/EE895A5F2FBD208FCA257E17007FDF29/$File/tb-notifications-2012-13.pdf
2. State Government of Victoria. *Public Health and Wellbeing Act 2008*. Accessed on 23 June 2015. Available from: http://www.legislation.vic.gov.au/Domino/Web_Notes/LDMS/LTObject_Store/LTObjSt9.nsf/DDE300B846EED9C7CA257616000A3571/A471FD6B321E3001CA257D72001BB396/$FILE/08-46aa024%20authorised.pdf
3. Department of Immigration and Citizenship. Population flows: immigration aspects 2008–2009. Canberra, Australia: Commonwealth of Australia, 2010.
4. Australian Bureau of Statistics. 4102.0 – Australian Social Trends, 1995. Participation in Education: Overseas students in higher education. Accessed 23 June 2015. Available from: http://www.abs.gov.au/AUSSTATS/abs@.nsf/Previousproducts/DE4FD4D0AF435AD4CA2570EC007521F0?opendocument
5. King K, Dorner RI, Hackett BJ, Berry G. Are health undertakings effective in the follow-up of migrants for tuberculosis? *Med J Aust* 1995;163(8):407–411.
6. Australian Government. Australian Immigration Fact Sheet 22. The Health Requirement. Accessed 23 June 2015. Available from: https://www.immi.gov.au/media/fact-sheets/22health.htm
7. Australian Government. Health requirement for permanent entry to Australia, Form 1071i. Canberra, Australia: Commonwealth of Australia, 2009
8. Australian Government. Health requirement for temporary entry to Australia, Form 1163i. Canberra, Australia: Commonwealth of Australia, 2009.
9. Correa-Velez I, Gifford SM, Bice SJ. Australian health policy on access to medical care for refugees and asylum seekers. *Aust New Zealand Health Policy* 2005;2:23.
10. Flynn M, Brown L, Tesfai A, Lauer T. Post-migration screening for active tuberculosis in Victoria, Australia. *Inter J Tuberc Lung Dis* 2012;16(1):50–54.
11. National Health and Medical Research Council. Prevention of tuberculosis. In: *Tuberculosis in Australia and New Zealand into the 1990s*. Canberra, Australia: Australian Government Publishing Service, 1990: pp 67–69.
12. Australasian Society for Infectious Diseases. TB. In: *Diagnosis, management and prevention of infections in recently arrived refugees*. Sydney, Australia: Dreamweaver Publishing, 2009: pp 12–14.
13. MacIntyre CR, Dwyer B, Streeton JA. The epidemiology of tuberculosis in Victoria. *Med J Aust* 1993;159:672–677.
14. Dye C, Scheele S, Dolin P, Pathania V, Raviglione MC. Global burden of tuberculosis: estimated incidence, prevalence and mortality by country. *JAMA* 1999;282:677–686.
15. Department of Human Services. Management. *Control and Prevention of Tuberculosis. Guidelines for Health Care Providers (2002–2005)*. Melbourne, Australia: Victorian Government, 2002.
16. Saukkonen JJ, Cohn DL, Jasmer RM, Schenker S, Jereb JA, Nolan CM, et al. An official ATS statement: Hepatotoxicity of antituberculosis therapy. *Am J Respir Crit Care Med* 2006;174(8):935–952.
17. American Thoracic Society. Targeted tuberculin testing and treatment of latent tuberculosis infection. *Amer J Respir Crit Care Med* 2000;161(4 Pt 2):S221–S247.
18. American Thoracic Society, Centers for Disease Control and Prevention, Infectious Diseases Society of America. Controlling tuberculosis in the United States. *Am J Respir Crit Care Med* 2005;172(9):1169–1227.
19. National Tuberculosis Advisory Committee. Essential components of a tuberculosis control program within Australia. *Commun Dis Intell* 2014;38(4):E397–E400.
20. Marks GB, Bai J, Simpson SE, Sullivan EA, Stewart GJ. Incidence of tuberculosis among a cohort of tuberculin-positive refugees in Australia: reappraising the estimates of risk. *Am J Respir Crit Care Med* 2000;162(5):1851–1854.
21. Diel R, Loddenkemper R, Niemann S, Meywald-Walter K, Nienhaus A. Negative and positive predictive value of a whole-blood interferon-γ release assay for developing active tuberculosis: an update. *Am J Respir Crit Care Med* 2011;18(1):88–95.
22. Kik SV, Franken WPJ, Mensen M, Cobelens FGJ, Kamphorst M, Arend SM, et al. Predictive value for progression to tuberculosis by IGRA and TST in immigrant contacts. *Eur Respir J* 2010;35(6):1346–1353.
23. Lobue P, Menzies D. Treatment of latent tuberculosis infection: an update. *Respirology* 2010;15(4):603–622.
24. Hart DHL. Factors influencing prescription of isoniazid chemo-preventive therapy [abstract]. *Respirology* 2000;5 Suppl:A72
25. Anibarro L, Casas S, Paz-Esquete J, Gonzalez L, Pena A, Guerra MR, et al. Treatment completion in latent tuberculosis infection at specialist tuberculosis units in Spain. *Int J Tuberc Lung Dis* 2010;14(6):701–707.
26. Australian Government. Overseas Visitors Health Cover. Accessed 19 October 2013. Available from: http://www.privatehealth.gov.au/healthinsurance/overseas/ovhc.htm
27. Hirsch-Moverman Y, Daftary A, Franks J, Colson PW. Adherence to treatment for latent tuberculosis infection: Systematic review of studies in the US and Canada. *Int J Tuberc Lung Dis* 2008;12(11):1235–1254.

28. Dobler CC, Marks GB. Completion of treatment for latent tuberculosis infection. *Eur Respir J* 2011;38,Suppl 55:2573.
29. Bothamley GH, Ditiu L, Migliori GB, Lange C. Active case finding of tuberculosis in Europe: a Tuberculosis Network European Trials Group (TBNET) survey. *Eur Respir J* 2008;32(4):1023–1030.
30. Rennie TW, Bothamley GH, Engova D, Bates IP. Patient choice promotes adherence in preventive treatment for latent tuberculosis. *Eur Respir J* 2007;30(4):728–735.
31. Jiménez-Fuentes MA, de Souza-Galvao ML, Mila Augé C, Solsona Peiró J, Altet-Gómez MN. Rifampicin plus isoniazid for the prevention of tuberculosis in an immigrant population. *Int J Tuberc Lung Dis* 2013;17(3):326–332.
32. Matteelli A, Casalini C, Raviglione MC, El-Hamad I, Scolari C, Bombana E, et al. Supervised preventive therapy for latent tuberculosis infection in illegal immigrants in Italy. *Am J Respir Crit Care Med* 2000;162(5):1653–1655.
33. Chang AH, Polesky A, Bhatia G. House calls by community health workers and public health nurses to improve adherence to isoniazid monotherapy for latent tuberculosis infection: a retrospective study. *BMC Public Health* 2013;13:894.
34. Kan B, Kalin M, Bruchfeld J. Completing treatment for latent tuberculosis: patient background matters. *Int J Tuberc Lung Dis* 2013;17(5):597–602.
35. Trajman A, Long R, Zylberberg D, Dion MJ, Al-Otaibi B, Menzies D. Factors associated with treatment adherence in a randomised trial of latent tuberculosis infection treatment. *Int J Tuberc Lung Dis* 2010;14(5):551–559.
36. Lavender CJ, Globan M, Kelly H, Brown LK, Sievers A, Fyfe JAM, et al. Epidemiology and control of tuberculosis in Victoria, a low-burden state in south-eastern Australia, 2005–2010. *Int J Tuberc Lung Dis* 2013;17(6):752–758.
37. MacIntyre CR, Plant AJ, Yung A, Streeton JA. Missed opportunities for prevention of tuberculosis in Victoria, Australia. *Int J Tuberc Lung Dis* 1997;1(12):135–141.
38. MacIntyre CR, Plant AJ. Preventability of incident cases of tuberculosis in recently exposed contacts. *Int J Tuberc Lung Dis* 1998;2(1):56–61.
39. Denholm JT, McBryde ES, Brown GV. Ethical evaluation of immigration screening policy for latent tuberculosis infection. *Aust N Z J Public Health* 2012;36:325–328.
40. Denholm JT, McBryde ES. Can Australia eliminate TB? Modelling immigration strategies for reaching MDG targets in a low-transmission setting. *Aust N Z J Public Health* 2014;38(1):78–82.
41. Denholm JT. Immigration screening for latent tuberculosis infection. *Med J Aust* 2013;198(10):524.
42. Campbell J, Marra F, Cook V, Johnston J. Screening immigrants for latent tuberculosis: do we have the resources. *CMAJ* 2014;186(4):246–247.
43. Denholm JT, McBryde ES. Management of latent tuberculosis infections in Australia and New Zealand: A review of current practice. *Tuberc Res Treat* 2010; 2010:284028. doi: 10.1155/2010/284028.

Surveillance summaries

National Notifiable Diseases Surveillance System surveillance report: Sexually transmissible infections in Aboriginal and Torres Strait Islander people

Amy Bright

Introduction

Aboriginal and Torres Strait Islander people represent 3% of the Australian population, of which more than two-thirds are less than 34 years of age.[1] The Indigenous population is considerably diverse, socially, culturally and geographically, providing a challenging environment to deliver culturally appropriate and accessible healthcare services.[2]

Despite improvements in health outcomes, disparities between the Indigenous and non-Indigenous populations are evident and occur for a range of health issues, including sexual health.[3] Indigenous people continue to be disproportionately represented in the sexually transmissible infections (STI) notification data, particularly in younger age groups residing in remote locations.

Methods

Notification data, for selected STIs, extracted from the Nationally Notifiable Diseases Surveillance System (NNDSS) as at 17 September 2015 were used for the analyses. HIV notification data, collected through the National HIV Registry, were sourced from the 2015 annual surveillance reports from the Kirby Institute.[4]

Case identification

For the purposes of this report, notifications with an Indigenous status field reported as not Indigenous or blank/unknown were considered to be non-Indigenous. In interpreting these data it is important to note that changes in notifications over time may not solely reflect changes in disease prevalence. Changes in screening programs,[5,6] the use of less invasive and more sensitive diagnostic tests[7] and periodic public awareness campaigns[8] may influence the number of notifications that occur over time. Rates for STIs are particularly susceptible to overall rates of testing.[9] As a priority and 'at risk' population, Indigenous people are commonly targeted for STI screening often resulting in a higher number of reported cases.

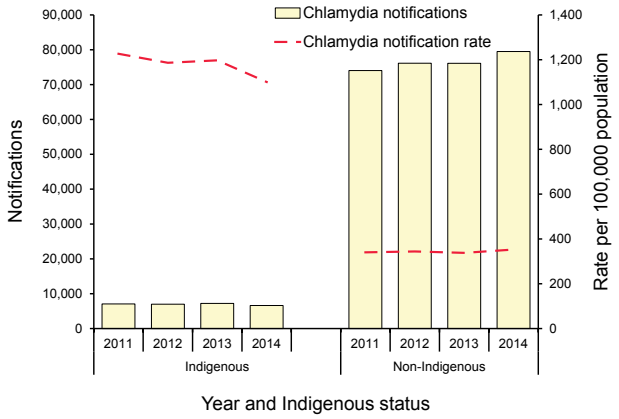
Results

In 2014, the notification rates for chlamydia, infectious syphilis and gonococcal infections in the Indigenous population were 3, 4 and 18 times higher respectively than the non-Indigenous population (Table, Figures 1–3).

Table: Notification rates per 100,000 for sexually transmissible infections and HIV and 5 year trend, Australia, 2014, by Indigenous status

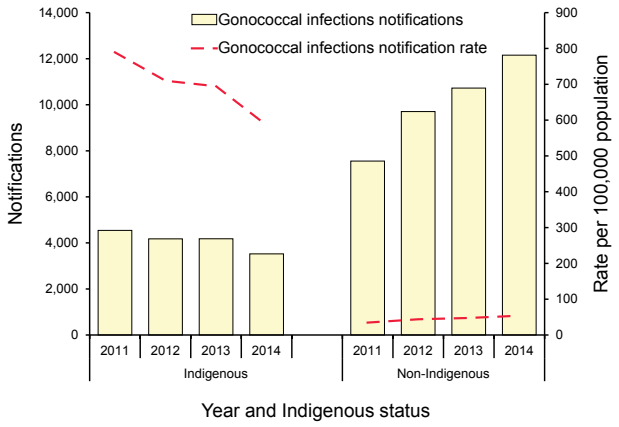
	Non-Indigenous		Indigenous	
Disease	2014	5-year trend	2014	5-year trend
HIV (newly diagnosed)	3.7	Stable from 2012 (men who have sex with men; urban areas)	5.9	Fluctuating (heterosexual contact and injecting drug use)
Chlamydia	389.0	Stable from 2011	1,341.0	Stable from 2011
Gonorrhoea	49.0	81% increase (men who have sex with men; urban areas)	859.0	Fluctuating – declines in younger age groups (heterosexual contact; remote areas)
Infectious syphilis	8.0	73% increase (men who have sex with men; urban areas)	32.0	46% increase (heterosexual contact; remote areas)
Donovanosis	–	1 case in 2012	–	1 case in 2014

Figure 1: Notifications and notification rate (unadjusted) for chlamydia, 2011 to 2014, by year and Indigenous status



* Data as at 17 September 2015.

Figure 2: Notifications and notification rate (unadjusted) gonococcal infection, 2011 to 2014, by year and Indigenous status

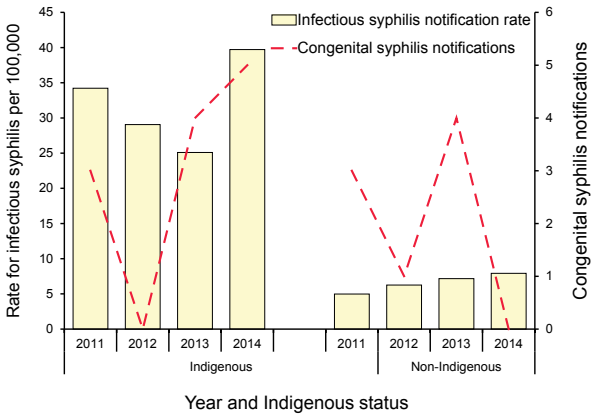


* Data as at 17 September 2015.

In 2014, in the Indigenous population:

- chlamydia notifications were predominately reported in females, representing 66% of notifications (non-Indigenous 56% of notifications were female) and 80% of all chlamydia notifications for Indigenous persons were in the 15–29 years age range (78% in non-Indigenous);
- gonococcal notifications were reported relatively evenly across the sexes indicating transmission was predominately heterosexual , with females representing 55% and males 45% of notifications (non-Indigenous 81% of notifications were male), and 71% of all gonorrhoea notifications for Indigenous persons were in the 15–29 years age range (56% in non-Indigenous);

Figure 3: Notification rate (unadjusted) for infectious syphilis and number of congenital syphilis cases, by year and Indigenous status, 2011 to 2014



* Data as at 17 September 2015.

- infectious syphilis notifications were reported evenly across the sexes indicating transmission was predominately heterosexual, with males representing 52% and females 48% of notifications (non-Indigenous 97% of notifications were male), and 67% of all infectious syphilis notifications for Indigenous persons were in the 15–29 years age range (44% in non-Indigenous);
- 1 case of donovanosis was reported in a 38-year-old male (no cases in the non-Indigenous population).
- An on-going outbreak of infectious syphilis affecting young Indigenous people in remote areas of northern Australia (Western Australia, the Northern Territory and Queensland) has resulted in a marked increase in the notification rate in Indigenous children aged 15–19 years between 2010 and 2014 (34 to 99 per 100,000).

The notification rate for infectious syphilis in the Indigenous population between 2013 and 2015 (17 September) almost doubled (25–49 per 100,000). Increases in infectious syphilis may coincide with increased cases of congenital syphilis (Figure 3).

Notification rates of HIV diagnosis in 2014 among the Indigenous population were higher than in the non-Indigenous population (5.9 and 3.7 per 100,000 respectively).

A summary of STI transmission, treatment, trends and risk groups in Australia is shown in the Appendix.

Data presented in this report may differ to those presented in the 2015 Annual Kirby surveillance

reports and the NNDSS annual report (pending publishing); however the overall trends are comparable.

Discussion

In Australia, notifications of non-congenital syphilis and gonorrhoea have continued to rise, with the majority of notifications occurring in non-Indigenous males residing in urban settings and young Indigenous people residing remote areas. In the last few years notifications of chlamydial infection have stabilised; however chlamydia continues to be the most commonly reported infection across all nationally notifiable diseases, largely affecting young Indigenous and non-Indigenous women.

Greater representation of women in STI notifications is linked to health seeking behaviour; in general women are more likely to access healthcare services as compared to men who often have biological, sociological and psychological factors determining their 'help-seeking' practices.[10] This is particularly apparent in Indigenous men, who face not only gender related issues but cultural and in some instances geographical barriers when accessing care.[10] Men who remain undiagnosed and therefore untreated have the potential to perpetuate transmission within communities. Repeat and persistent STI infection in Indigenous communities is common, and can have considerable impact on rates of notifications and the prevalence of serious complications of infection, including pelvic inflammatory disease.[11,12] Physical access to condoms, due to geographical location, and inconsistent condom use, are also contributing factors to high rates of STI infection in the Indigenous population.[13,14]

The Australian Government, in collaboration with states, territories and the community, continues to work towards effective public health interventions to prevent STI within the population, particularly among young people and other 'at risk' populations. The fourth *National Aboriginal and Torres Strait Islander BBV and STI Strategy 2014–2017*, part of a suite of National Strategies for BBV and STI, includes specific targets for the elimination of congenital syphilis, reducing the incidence of chlamydia, gonorrhoea and infectious syphilis in people under 30 years of age, and increasing the number of people with HIV receiving treatment.

The Strategy supports the goals of *National Aboriginal and Torres Strait Islander Health Plan 2013–2023*, a long-term, evidence-based policy framework as part of the overarching Council of Australian Governments' approach to Closing the Gap in Indigenous disadvantage.

To achieve the targets of the Strategy and address the disproportionate representation of Indigenous people with STI, the Australian Government continues to support innovative approaches, including Point-of-Care Testing to achieve same day STI testing and treatment for Indigenous people residing in remote communities; engagement of organisations to provide healthcare services specifically to Indigenous people, including routine screening for STI; and comprehensive community education and health promotion.

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Appendix: Sexually transmissible infections transmission, treatment, trends and risk groups, Australia

Disease	Transmission[15] (causative agent)	Recommended treatment[16]	Trends	Risk groups
Gonorrhoea	Sexual exposure – contact with exudate from mucus membranes of infected people. Sites of infection include – urogenital, rectum and pharynx/throat. Can be transmitted perinatallly (during childbirth). (*Neisseria gonorrhoeae*)	Ceftriaxone 500 mg single dose intramuscularly (decreased susceptibility reported)	Highest number of reported notifications in 2014 (more than 15,000). Notifications have increased substantially over the previous 7 years – with the highest increase between 2009 and 2010 (23%).	Men who have sex with men (MSM) Heterosexual contact within Indigenous populations
Chlamydia	Sexual exposure – genital contact and/or intercourse with an infected people (cases largely asymptomatic) Perinatal transmission occurs through exposure to mother's infected cervix during childbirth. (*Chlamydia trachomatis* [immunotypes D – K])	Azithromycin 1 g single oral dose	Most frequently reported infection in Australia. Notifications stabilised from 2011 after steady increase from 1999 to 2010.	Females 15–24 years Indigenous people (men and women)
Syphilis*	Sexual exposure – direct contact with infectious exudates from moist early lesions of skin and mucous membranes. Transplacental infection of the fetus occurs during pregnancy in an infected woman. (*Treponema pallidum*)	Penicillin 1.8 g single dose intramuscularly	Rates increasing for non-congenital cases –with the majority of cases reported in MSM. Congenital syphilis rare disease – with approximately 5 cases reported each year.	Men who have sex with men (MSM) Heterosexual contact within Indigenous populations
Donovanosis	Sexual exposure – contact with ulcers. (*Klebsiella granulomatis*)	Azithromycin 1 g oral dose weekly for 4–6 weeks	Targeted for elimination. Predominantly in Indigenous females in rural/remote communities in central/ northern Australia – relatively uncommon. Fewer than 6 cases notified each year since 2006.	
HIV (notified to the Kirby Institute)	Sexual exposure – exchange of bodily fluids during sex. Can be transmitted perinatallly and parenterally (including intravenous injection).	Antiretroviral therapy – to suppress HIV replication (no cure)	Newly diagnosed infection has stabilized over the last 3 years (2012–14). Estimated 27,150 people living with HIV – 12% unaware of their HIV positive status.	MSM Heterosexual contact within Indigenous populations.

Appendix (cont'd): Sexually transmissible infections transmission, treatment, trends and risk groups, Australia

Disease	Transmission[15] (causative agent)	Recommended treatment[16]	Trends	Risk groups
HPV (not nationally notifiable)	Sexual exposure – genital, rectal, mouth and oropharyngeal, skin to skin and mucosal to mucosal membrane contact and oral faecal contamination during sex. (Human papillomavirus – largely non-cancerous genotypes 6 and 11 (causing genital warts) and cancerous genotypes 16 and 18.	No treatment for virus but vaccine preventable – cancerous outcomes managed	Early markers of HPV infection (genital warts) have decreased substantially in young females post vaccination program (2007) – with marked decreases in largely heterosexual males (2013). Low and high grade cervical lesions (pre-cancer) have also declined in females.	All sexually active people.

* Includes unspecified syphilis (more than 2 years or unspecified duration), infectious syphilis (less than 2 years duration) and congenital syphilis.

References

1. Australian Bureau of Statistics. *Estimates and Projections, Aboriginal and Torres Strait Islander Australians, 2001 to 2026*. Canberra: Australian Bureau of Statistics; cat. no. 3238.0; 2014.
2. Australian Institute of Health and Welfare. *The Health and Welfare of Australia's Aboriginal and Torres Strait Islander Peoples*. Canberra: Australian Institute of Health and Welfare; cat. no. IHW 147; 2015.
3. Australian Institute of Health and Welfare. *Australia's Health 2014*. Australia's health series no. 14. Canberra: Australian Institute of Health and Welfare; cat. no. AUS 178; 2014.
4. The Kirby Institute. HIV, Viral Hepatitis and Sexually Transmissible Infections in Australia Annual Surveillance Report. Sydney NSW: The University of New South Wales; 2015.
5. 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.
6. 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.
7. 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.
8. 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.
9. 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 Trans Infect* 2012;88(7):552–557.
10. Smith J, Braunack-Mayer A, Wittert G. What do we know about men's help-seeking and health service use? *Med J Aust* 2006;184(2):81–83.
11. Guy R, Natoli L, Ward J, Causer L, Hengel B, Whiley D, et al. A randomised trial of point-of-care tests for chlamydia and gonorrhoea infections in remote Aboriginal communities: Test, Treat ANd GO- the "TTANGO" trial protocol. *BMC Infect Dis* 2013;13(485).
12. Strobel N, Ward J. *Education programs for Indigenous Australians about sexually transmitted infections and bloodborne viruses*. Resource sheet no. 14 for the Closing the Gap Clearinghouse. Canberra, ACT: Australian Institute of Health and Welfare; 2012.
13. Stark A, Hope A. Aboriginal women's stories of sexually transmissible infection transmission and condom use in remote central Australia. *Sex Health* 2007;4(4):2237–2242.
14. Larkins S, Page R, Panaretto K, Scott R, Mitchell M, Alberts V, et al. Attitudes and behaviours of young Indigenous people in Townsville concerning relationships, sex and contraception: the "U Mob Yarn Up" project. *Med J Aust* 2007;186(10):513–518.
15. Heymann D. *Control of Communicable Diseases Manual*. American Public Health Association; 2008.
16. Australasian Society for HIV Medicine. HIV, Viral Hepatitis and STIs: A Guide for Primary Care. Sydney: 2014. Available from: http://www.ashm.org.au/resources/Pages/1976963411.aspx

Policy and guidelines

An Australian guideline on the diagnosis of overseas-acquired Lyme disease/borreliosis

Gary D Lum, Jennie R Hood, Phil Wright

Executive summary

While classical Lyme disease cannot be acquired in Australia, patients may present who have travelled through endemic areas. Lyme disease is prevalent in north east United States of America, parts of Europe including Germany, Austria, Slovenia and Sweden as well as parts of the United Kingdom. Lyme disease can also be found in Russia, Japan and China. For patients who present with no history of overseas travel but with a tick bite and systemic symptoms, e.g. fever, contact with your specialist microbiologist to discuss test referral and with your infectious diseases physician to discuss antimicrobial treatment of tick-borne infections in the Australian context is advised.

Lyme disease should be considered in patients presenting with a history of tick bite from one of these areas along with a fever and mild influenza-like symptoms. An annular rash, erythema emigrans may be present in 70%–80% of patients presenting with Lyme disease. Other manifestations of Lyme disease can occur. For example, Lyme neuroborreliosis can manifest as meningoradiculoneuritis, meningitis, cranial neuritis (predominately involving the facial nerve), brachial plexus neuritis, and mononeuritis; Lyme carditis can manifest with palpitations, chest discomfort, shortness of breath, dizziness on exercise or syncope; and rheumatological Lyme disease can present with arthralgia and myalgia. Rheumatological presentations are more common with north American acquisition and neurological presentations with European acquisition.

If Lyme disease is being considered, patients should be referred for Lyme disease serology to your regular approved pathology practitioner.

The testing follows a two-tiered approach involving a screening immunoassay and a confirmatory immunoblot. If you have concerns or questions about the testing please contact your approved pathology laboratory's specialist microbiologist.

While this guidance document is focussed on the diagnosis of overseas-acquired Lyme disease in Australia, treatment advice can be found on the Infectious Diseases Society of America web site (http://www.idsociety.org/ViewAllLyme/). Should you require further advice please make contact with an infectious diseases physician.

Figure 1 shows the recommended steps for diagnosing overseas-acquired Lyme disease in Australia.

Purpose

This document is a diagnostic clinical aid* for medical practitioners in Australia who are unfamiliar with Lyme disease acquired overseas.†

An Australian Lyme disease-like syndrome (ALDLS) case description is not part of this document. The use of the term Australian Lyme disease-like syndrome is not a formal acknowledgment or designation of a new disease in Australia. If ALDLS can be better characterised as a chronic debilitating multi-organ illness affecting some Australians a separate ALDLS description can be written.

This diagnostic guide for overseas-acquired Lyme disease is not a national disease surveillance document. Lyme disease is not a notifiable disease in Australia. This document does not address chronic Lyme disease, relapsing fever or Lyme disease co-infections.‡ This guide is not a comprehensive 'text book' description of the natural history of an infection. Details on the epidemiology and *in vitro* diagnostic device specifications can be obtained elsewhere. Specific advice on the specifications of *in vitro* diagnostic assays used in Australia should be sought from the responsible specialist microbiologist or medical laboratory scientist offering Lyme disease testing.

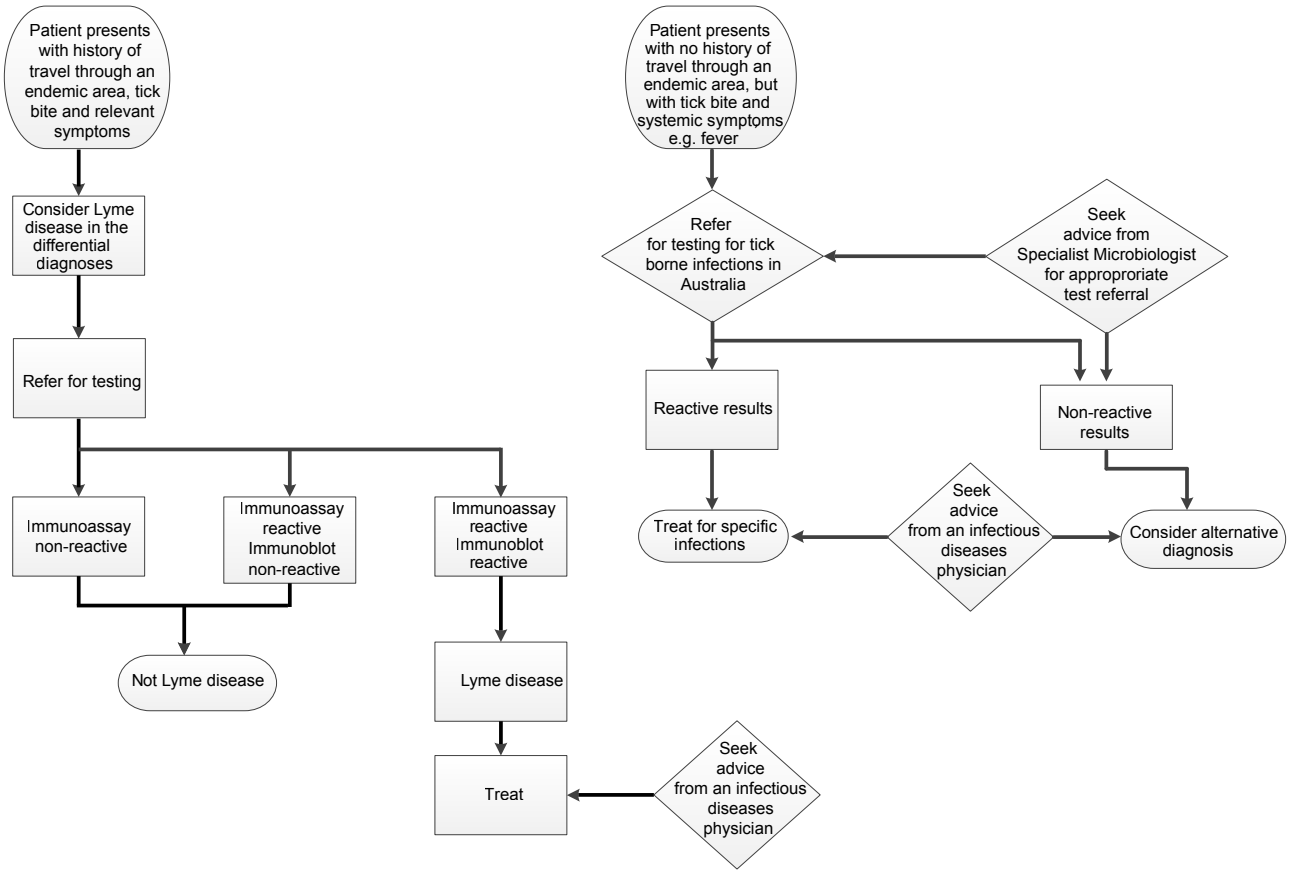
Input has been sought from members of the Diagnostic Pathway Working Group of the Chief Medical Officer's Clinical Advisory Committee

* The case definition is for guidance rather than being a prescriptive standard.

† Patients and their advocates have reported that some Australian registered medical practitioners are not considering a diagnosis of Lyme disease in patients who have travelled to overseas endemic areas.

‡ For example infections caused by *Babesia*, *Anaplasma*, *Bartonella* and *Ehrlichia*.

Figure 1: Flow chart for an Australian diagnostic guideline for overseas acquired Lyme disease



on Lyme disease and was discussed at the Lyme Disease Treatment Round Table held on Tuesday 27 May 2014. This guide has been considered by the Public Health Laboratory Network and the Communicable Diseases Network Australia. It has been endorsed by the Australian Health Protection Principal Committee on 13 August 2015.

As a guidance document, laboratory diagnostic testing is required for 2 reasons: 1. For overseas acquired infection by the genogroup *Borrelia burgdorferi* sensu lato, unless the clinician is familiar with the pathognomonic erythema migrans (EM) rash, it is clinically safer to obtain supportive evidence of infection through diagnostic testing, and 2. diagnostic laboratory support is preferred for patients presenting with non-specific signs and symptoms of a disease or syndrome, notwithstanding the limitations of diagnostic tests.

Lyme disease / borreliosis

An indigenous causative microorganism is yet to be found in Australia. This case definition can be applied to patients with Lyme disease acquired from endemic areas overseas.

Confirmed case

The diagnosis of overseas-acquired Lyme disease in Australia should rest on a careful medical history and objective clinical findings with the support of appropriate *in vitro* diagnostic tests. Laboratory support is essential because of the non-specific nature of many clinical manifestations, especially when the diagnosis is made in a non-endemic area.

A confirmed case requires laboratory definitive evidence AND clinical evidence AND epidemiological evidence.

Laboratory definitive evidence

Culture of *Borrelia* bacteria§ from clinical specimens offers the best confirmation of active infection. However, due to the low numbers of viable spirochaetes usually present in patient biopsies and the fastidious nature of *Borrelia* species, the sensitivity of culture is poor.[1,2,3] The inability to isolate an organism does not exclude active infection.[1] Relevant *Borrelia* species can be recovered from various tissues and body fluids including: biopsy and lavage specimens of EM skin lesions, biopsy specimens of acrodermatitis chronica atrophicans

§ Known to cause Lyme borreliosis in endemic areas

(ACA) skin lesions, biopsy specimens of borrelial lymphocytoma skin lesions, cerebrospinal fluid (CSF) specimens, synovial fluid and blood specimens.[3,4] There have also been reports of recovery of relevant *Borrelia* species from other tissues such as cardiac tissue.[3,5]

OR

Detection of relevant *Borrelia* species by nucleic acid amplification tests (NAT) (e.g. polymerase chain reaction (PCR)) taken from skin biopsies of EM or ACA skin lesions or appropriate body fluid samples of blood, CSF or synovial fluid (depending on clinical manifestations).[3,4] The use of urine-PCR has been investigated by several groups, but results are contradictory, therefore PCR on urine is not recommended for routine diagnosis.[1,4]

OR

Serological evidence by two tiered testing. An immunoblot (IB) should not be performed without a reactive immunoassay screening result. A positive diagnosis can only be achieved with a reactive screening immunoassay and sufficient number of reactive bands in an IB.

Serological evidence requires both:

- the presence of IgM (acute phase¶) or IgG (convalescent phase) detected by an immunoassay** using recombinant antigen. (In the convalescent period, a reactive IgM and a nonreactive IgG should be regarded as a falsely positive IgM result.)

AND

- the presence of IgG (convalescent phase) detected by an IB using recombinant antigen or whole cell preparations and fulfilling criteria for a positive result. Although IgM IBs are available, repeat testing for IgG by IB on a convalescent sample is advised.

Testing should be performed in a laboratory that has Lyme disease testing in its scope of accreditation and which is compliant with *AS ISO 15189 Medical laboratories — Particular requirements for quality and competence* or in nationally accredited laboratories in the location where the patient was infected. Commercial serological assays used in Australian laboratories with AS ISO 15189 medical testing accreditation are suitable for testing for Lyme disease acquired overseas in endemic regions. Consideration should be given to storing positive serum specimens for research and quality assurance purposes.

Clinical specimens that produce repeatedly equivocal results, indeterminate results and results from laboratories without AS ISO 15189 medical testing accreditation should be considered cautiously and expert advice from a specialist microbiologist should be obtained. It may be necessary to refer patient specimens to a suitably certified laboratory such as the US Centers for Disease Control and Prevention.

Clinical evidence

For the purposes of this case definition, a history of overseas travel to areas where Lyme disease/borreliosis is endemic is required when assessing a patient's clinical presentation. For a diagnosis of Lyme disease to be considered the patient must have been exposed to ticks, however, a history of documented tick bite is not essential because many tick bites go unnoticed.[1]

The clinical presentation of borreliosis depends on the different stages of the disease. It has also been demonstrated that different genospecies exert different organotropic and pathogenic potential.[6,7] For the purpose of this case definition three broad disease stages are recognised: early Lyme disease, early disseminated Lyme disease and late Lyme disease. Not all stages need to appear, stages may also overlap and the infection may also be asymptomatic.[8] Examples of the different clinical manifestations of the three most common genospecies (*B. burgdorferi* s.s., *B. afzelii* and *B. garinii*) are provided in the Appendix.

Early Lyme disease

Early Lyme disease generally occurs within several days (but may be up to 4 months) after a tick bite and may include symptoms such as the presence of an isolated EM rash (Figure 2), undifferentiated febrile illness and/or influenza-like symptoms (including fatigue and myalgia) or patients may be asymptomatic.[9]

EM is a characteristic expanding rash (usually larger than 5 cm) that may develop 3 days to 16 weeks after a tick bite and resolves spontaneously in a few weeks or months.[8] The rash is present in 70%–80% of infected persons. The rash begins at the site of the tick bite as a red macule or papule, rapidly enlarges and sometimes develops central clearing but is often homogenous.[1,7] The advancing edge is typically distinct and is often intensely

¶ Specific IgG is usually detectable 4 to 6 weeks after infection, although to determine a definite change in IgG levels a minimum of 3 months may be necessary to determine a change when tested in parallel.

** A non-reactive immunoassay should be regarded as a negative result.

coloured but not markedly elevated.[10] The duration of the rash is usually dependent on the infecting genospecies but will last approximately 4 to 14 days[9,11] Haemorrhagic or non-migrating forms have occasionally been observed.[12] Erythematous lesions occurring within a few hours after a tick bite represent hypersensitivity reactions and do not qualify as EM.[1] In endemic areas, EM is pathognomonic for infection.

Figure 2: Erythema migrans rash

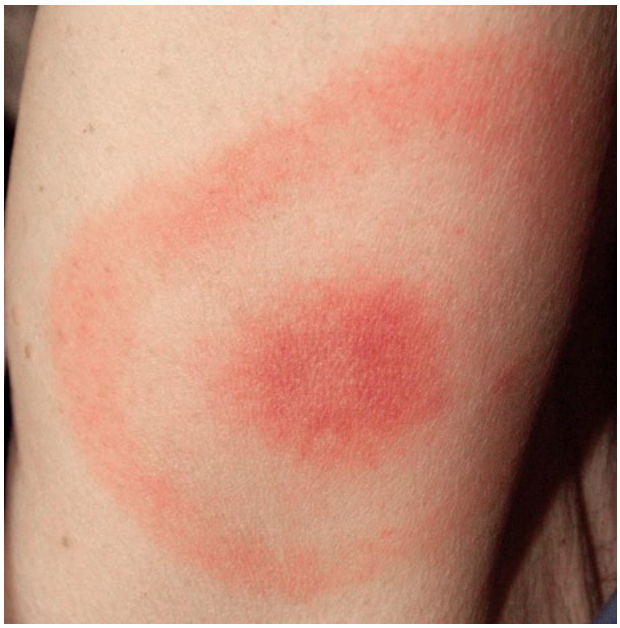


Image source : US Centers for Disease Control and Prevention Public Health Image Library; Photo by James Gathany.

Early disseminated Lyme disease

A few weeks to months after the initial infection, several organs may become affected, usually due to haematogenous spread.[7,8]

Lyme neuroborreliosis (LNB) is usually an acute disease (but can have chronic presentation), which develops within 1–12 weeks (mostly 4–6 weeks) after a tick bite.[1,13] Neurological features include meningoradiculoneuritis, meningitis, cranial neuritis (predominately involving the facial nerve), brachial plexus neuritis, mononeuritis, and rarely encephalitis, myelitis and cerebral vasculitis. These manifestations may occur separately or in association.[1,8,13,14] For a reliable diagnosis of LNB, indicative clinical neurological manifestations must be associated with inflammatory CSF pleocytosis and proof of intrathecal production of *Borrelia*-specific antibodies (IgG and/or IgM). Some commercially available serological assays are validated to test CSF specimens. CSF PCR and CSF culture may be corroborative if symptom duration is less than 6 weeks when *B. burgdorferi* antibodies may be absent.[13]

Lyme carditis usually manifests as varying degrees of transient atrioventricular defects which may result in the following symptoms: palpitations, chest discomfort, shortness of breath, dizziness on exercise, or syncope. Other features can include arrhythmias, (myo)pericarditis, and heart failure.[8,14]

Multiple EM-like lesions are common with some genospecies but uncommon with others. Borrelial lymphocytoma can appear in all stages but is most often observed during this stage.[8]

Arthralgia and myalgia signify early musculoskeletal involvement. Frank arthritis and myositis are occasionally observed. Regional lymphadenopathy and generalised lymphadenopathy may develop.[8] Joint involvement is common in North America and less common in Europe.

Other features of this stage can include eye disorders (such as uveitis, papillitis, keratitis and episcleritis), hepatomegaly, hepatitis and rarely, a dry cough and testicular swelling.[1,8]

Late Lyme disease

Late organ involvement may occur many months to years after infection and manifestations are primarily rheumatological and neurological.[7] Chronic fatigue is also commonly reported.

With some genospecies, recurrent brief episodes of monoarticular or oligoarticular arthritis are common especially in the large joints.[8,14] ACA is associated with certain genospecies, is almost exclusively seen in adults (predominantly women) and mainly affects the extensor surfaces of the extremities.[1,14]

Long term progressive Lyme encephalitis and encephalomyelitis are extremely rare even in areas of high endemicity in the United States of America (USA) and in Europe. Manifestations can be highly variable depending on the localisation of inflammatory foci in the brain. The course is typically chronic progressive and spasticity and cerebellar symptoms are often prominent clinical features. Also on rare occasions cerebral vasculitis, myositis, a dermatomyositis-like syndrome, reactive hyperplasia of the bone marrow, keratitis, and dilated cardiomyopathy may be present independently or associated with ACA.[8]

Impaired cognitive function following treated Lyme borreliosis may also be observed (such as memory problems, poor concentration, difficulties in formulating ideas and difficulties in word find-

ing), but it is difficult to attribute these symptoms directly to *Borrelia* infection or indirectly through effects of systemic infection or other toxic metabolic factors.[15–17] Patients with Late Lyme disease invariably have measurable serum specific antibodies. Post treatment late Lyme disease is not the same as chronic Lyme disease. Chronic Lyme disease is a controversial diagnosis and refers to ongoing active infection. Evidence for active long term infection remains controversial and is not widely accepted.

Epidemiological evidence

Epidemiological context is important. Determining a travel history and tick exposure prone activities are essential. The likelihood of Lyme disease increases as the probability of a tick bite increases in a geographically endemic area (particularly wooded, brushy, or grassy habitats). Endemic areas can be defined as those with established populations of vector ticks and evidence of enzootic transmission of relevant *Borrelia* species between the tick and the resident animal population.

In the USA, 13 states in the north east, where *Ixodes scapularis* is prevalent, account for 95% of reported cases; these states include Connecticut, Delaware, Maine, Maryland, Massachusetts, Minnesota, New Hampshire, New Jersey, New York, Pennsylvania, Vermont, Virginia, and Wisconsin.

In Europe, the regions with the highest prevalence include Germany, Austria, Slovenia, and Sweden (Figure 3). Lyme disease has been also diagnosed in the United Kingdom.

Lyme disease can also be acquired in Russia, Japan, and China.

Epidemiological evidence will be reviewed for an Australian context should an indigenous organism and its vector be identified.

Suspected case††

The presence of supporting laboratory evidence‡‡ AND epidemiological evidence without signs and symptoms consistent with early Lyme disease§§ (and no alternative explanation/diagnosis)

OR

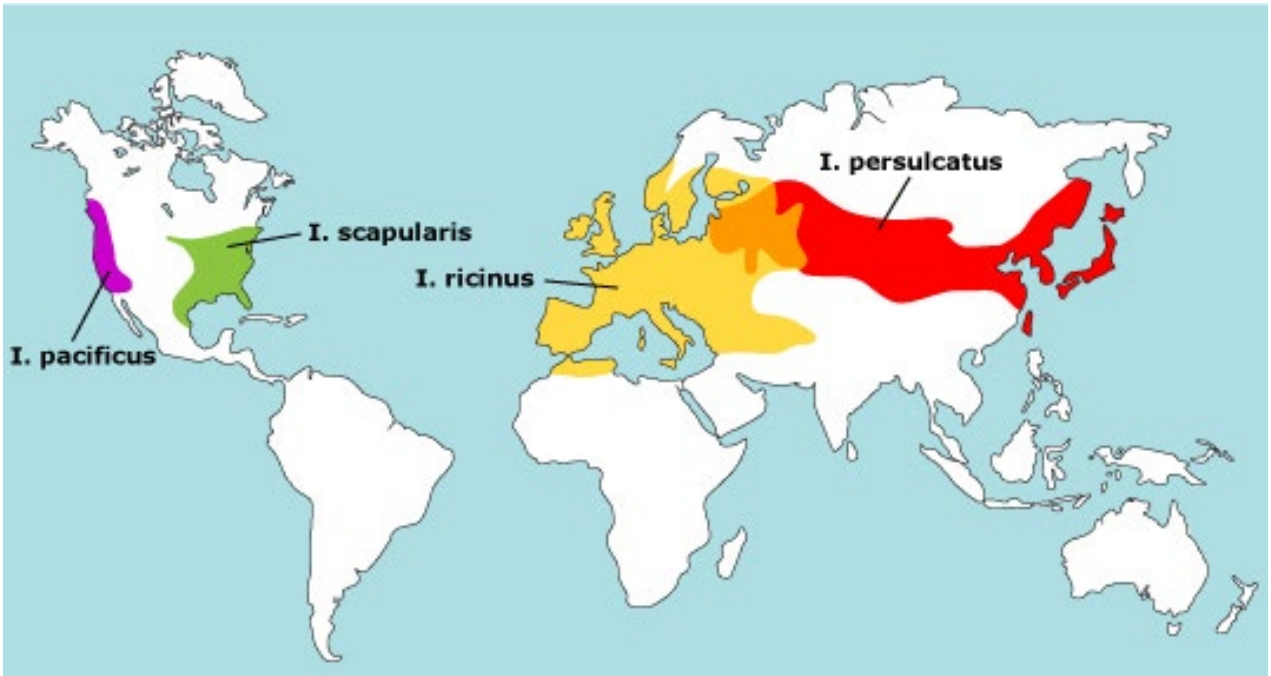
An EM rash AND epidemiological evidence (as defined above) (and no alternative explanation/diagnosis). Whilst this clinical manifestation may be pathognomonic in endemic countries unfamili-

†† Requires *in vitro* diagnostic testing to be performed in an AS ISO 15189 compliant laboratory.

‡‡ Positive nucleic acid amplification test results or reactive serological test results.

§§ For example, when there is history of a tick bite in an endemic region and Lyme disease testing has been requested and agreed.

Figure 3: Geographical distribution of the Ixodes tick vectors of the spirochetes that cause Lyme disease



Source: Eisen L, Lane RS. Vectors of *Borrelia burgdorferi* sensu lato. In: Lyme Borreliosis Biology, Epidemiology and Control, Gray JS, Kahl O, Lane RS, et al (Eds), CABI Publishing, Wallingford, Oxon, UK 2002. p. 91. Copyright ©2002 CAB International, Wallingford, UK.

arity with the presentation in Australia warrants laboratory confirmation (culture or PCR of the tissue) or more usually, antibody testing on a convalescent sample.

OR

Manifestations of LNB without CSF pleocytosis (including CSF not collected) and indeterminate laboratory results[¶¶] AND epidemiological evidence (and no alternative explanation/diagnosis)

OR

Manifestations of rheumatological Lyme disease and indeterminate laboratory results AND epidemiological evidence (and no alternative explanation/diagnosis).

Treatment guidance

While this guidance document is focussed on the diagnosis of overseas-acquired Lyme disease in Australia, treatment advice can be found on the Infectious Diseases Society of America web site (http://www.idsociety.org/viewalllyme). Should you require further advice please make contact with an infectious diseases physician. Australian infectious diseases physicians receive training that includes the management of classical Lyme disease.

Acknowledgement

Members of the Lyme Disease Diagnostic Pathway Working Group: Nikki Coleman, David Dickeson, Gull Herzberg, Bernie Hudson, Damon Langguth, Mualla McManus, Ann Mitrovic, Armin Schwarzbach.

¶¶ Non-reactive serological test results in CSF and equivocal or indeterminate serology results from serum specimens.

Appendix

Examples of different clinical manifestations of *Borrelia burgdorferi* s.s., *B. afzelii* and *B. garinii*

Certain clinical manifestations can occur more often with certain genospecies. The Appendix provides an indication of what symptoms are more likely to be associated with which genospecies.

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References

1. Stanek G, Fingerle V, Hunfeld KP, Jaulhac B, Kaiser R, Krause A, et al. Lyme borreliosis: Clinical case definitions for diagnosis and management in Europe. *Clin Microbiol Infect* 2011;17(1):69–79.
2. Ciceroni L, Ciarrochi S, Ciervo A, Mondarini V, Guzzo F, Caruso G, et al. Isolation and characterization of *Borrelia burgdorferi* sensu lato strains in an area of Italy where Lyme borreliosis is endemic. *J Clin Microbiol* 2001;39(6):2254–2260.
3. Aguero-Rosenfeld ME. Lyme disease: laboratory issues. *Infect Dis Clin North Am* 2008;22(2):301–313.
4. Wilske B, Fingerle V, Schulte-Spechtel U. Microbiological and serological diagnosis of Lyme borreliosis. *FEMS Immunol Med Microbiol* 2007;49(1):13–21.
5. Stanek G, Klein J, Bittner R, Glogar D. Isolation of *Borrelia burgdorferi* from the myocardium of a patient with longstanding cardiomyopathy. *N Engl J Med* 1990;322(4):249–252.

Appendix: Symptoms associated with *Borrelia* disease, by genospecies

Symptoms	*Borrelia burgdorferi* s.s.	*Borrelia afzelii*	*Borrelia garinii*
Erythema migrans	✓	✓	✓
Multiple erythema migrans	✓		
Lyme neuroborreliosis	Can occur		✓
Lyme carditis	✓	Rare but can occur	
Arthritis	✓		Can occur
Acrodermatitis chronica atrophicans		✓	

Please note that this is an indication of what symptoms are more likely associated with the genospecies, there will always be exceptions.

6. van Dam AP, Kuiper H, Vos K, Widjojokusumo A, de Jongh BM, Spanjaard L, et al. Different genospecies of *Borrelia burgdorferi* are associated with distinct clinical manifestations of Lyme borreliosis. *Clin Infect Dis* 1993;17(4):708–717.
7. Aguero-Rosenfeld ME, Wang G, Schwartz I, Wormser GP. Diagnosis of Lyme borreliosis. *Clin Microbiol Rev* 2005;18(3):484–509.
8. Pfister HW, Wilske B, Weber K. Lyme borreliosis: basic science and clinical aspects. *Lancet* 1994;343(8904):1013–1016.
9. Smith RP, Schoen RT, Rahn DW, Sikand VK, Nowakowski J, Parenti DL, et al. Clinical characteristics and treatment outcome of early Lyme disease in patients with microbiologically confirmed erythema migrans. *Ann Intern Med* 2002;136(6):421–428.
10. Stanek G, O'Connell S, Cimmino M, Aberer E, Kristoferitsch W, Granström M, et al. European Union concerted action on risk assessment in Lyme borreliosis: clinical case definitions for Lyme borreliosis. *Wien Klin Wochenschr* 1996;108(23):741–747.
11. Strle F, Nadelman RB, Cimperman J, Nowakowski J, Picken RN, Schwartz I, et al. Comparison of culture-confirmed erythema migrans caused by *Borrelia burgdorferi* sensu stricto in New York State and by *Borrelia afzelii* in Slovenia. *Ann Intern Med* 1999;130(1):32–36.
12. Wetter DA, Ruff CA. Erythema migrans in Lyme disease. *Can Med Assoc J* 2011;183(11):1281–1281.
13. Mygland Å, Ljøstad U, Fingerle V, Rupprecht T, Schmutzhard E, Steiner I. EFNS guidelines on the diagnosis and management of European Lyme neuroborreliosis. *Eur J Neurol* 2010;17(1):8–16, e1–e4.
14. Nadelman RB, Wormser GP. Lyme borreliosis. *Lancet* 1998;352(9127):557–565.
15. Krupp LB, Masur D, Schwartz J, Coyle PK, Langenbach LJ, Fernquist SK, et al. Cognitive functioning in late Lyme borreliosis. *Arch Neurol* 1991;48(11):1125–1129.
16. Cairns V, Godwin J. Post-Lyme borreliosis syndrome: a meta-analysis of reported symptoms. *Inter J Epidemiol* 2005;34(6):1340–1345.
17. Kaplan RF, Trevino RP, Johnson GM, Levy L, Dornbush R, Hu LT, et al. Cognitive function in post-treatment Lyme disease: do additional antibiotics help? *Neurol* 2003;60(12):1916–1922.

Policy recommendation: latent tuberculosis infection screening and treatment in children in immigration detention

Vicki Krause and the National Tuberculosis Advisory Committee

Background

Latent tuberculosis infection (LTBI) is a subclinical infection with *Mycobacteria tuberculosis* complex without any clinical, bacteriological or radiological evidence of manifest TB disease.[1] LTBI results from contact with an infectious case of TB. It is characterised by the presence of mycobacterial T-cell responses, assessed by either the tuberculin skin test (TST) or more recently, interferon gamma release assays (IGRAs).[2]

The World Health Organization estimates that one-third of the world's population is latently infected.[3] The diagnosis and treatment of LTBI is an important strategy for TB control and elimination, especially in low incidence countries where most adult cases result from reactivation of latent infection.[4]

Children with LTBI have an increased risk of developing active TB disease without treatment.[5] Children under 4 years of age are at highest risk.[2] In this age group the incubation or latency period is briefer and the disease more lethal, with invasive forms of the disease such as meningeal or miliary disease being more common.[6] Progression to active TB has been reported in up to 40% of infected infants.[7] Other children at particular risk for TB include those who are immunocompromised, malnourished or living in high TB burden areas.[2] A recent population-based study of TB in children across 20 United States of America (USA) jurisdictions during 2005–2006 found that, compared with TB rates among USA-born children with US-born parents, rates were 32 times higher in foreign-born children and 6 times higher in US-born children with foreign-born parents.[8A]

In Australia, screening for TB infection is targeted at those at high risk of recent infection (e.g. contacts of persons with TB disease or recently arrived foreign-born migrants), those at high risk of progression because of underlying conditions (e.g. HIV or disorders requiring immune-modulating drugs) or those with signs of possible past untreated TB disease.[9] The two currently available tests for LTBI in Australia are the TST and the QuantiFERON-TB Gold test, the only commercially available IGRA in Australia.

While the use of IGRAs for the detection of infection with *M. tuberculosis* among adults has shown promising results, the evidence base is still lacking in children. A recently published review on diagnostic approaches for LTBI in children found that studies assessing IGRA performance in children are limited.[2] Several studies have shown a higher percentage of indeterminate results in young and or immunocompromised children.[2] A position statement endorsed by the National Tuberculosis Advisory Committee in 2012 stated that IGRA should not replace TST for detection of LTBI in children.[10] However, IGRA may have additional value over TST in children that received bacille Calmette-Guérin vaccination after the first year of life.[10]

Whatever the diagnostic test, according to the US Pediatric Tuberculosis Collaborative Group, any child with LTBI should receive treatment.[11] This is because young age is a major risk factor for progression to active disease and infection in children is likely to have been more recent (another risk factor for reactivation of infection). Moreover, children have more years ahead of them to develop active TB. Preventive therapy should also be given to an child under 5 years of age (even if TST or IGRA negative) who is in close contact with an infectious adult until re-evaluation sometime after first contact, as this age group are most at risk of progression to active disease.[2]

Recommendations for the treatment of LTBI in children vary among countries; Australian guidelines for first line treatment of LTBI suggest 6–12 months of isoniazid monotherapy.[12,13]

Latent tuberculosis infection treatment in children in immigration detention

Children in immigration detention are at increased risk of both active TB disease and latent TB infection as they mostly come from high incidence countries and prior experiences may have included poverty, overcrowding and poor access to clinical and public health services.[14] By virtue of being in detention in a congregate setting, they are also at increased risk of exposure from adults with active TB. Between 2010 and 2012 the Northern Territory and Western Australia TB Units notified 57 cases

of tuberculosis among Irregular Maritime Arrivals in Australian immigration detention facilities (unpublished data).

In this context, ideally all children would be screened and treated for LTBI. Prioritisation, however, should be given to those at greatest risk of disease progression (e.g. contacts of active TB cases) and to those children attending school, as a public health initiative.

Recommendations

1. The following children (aged 6 months–18 years) in immigration detention should be screened for TB infection:
   a. All contacts of TB cases (priority should be given to contacts of cases who have smear positive pulmonary TB cases and/or extensive lung involvement);
   b. All children attending school (this should be undertaken optimally prior to attending school but at the latest, within one month of starting school);
2. The screening test for LTBI is the TST.
3. All children who have a positive TST and/or have symptoms or signs of TB should have a detailed history, clinical examination and chest X-ray performed to rule out active TB. This should preferably be carried out at a specialised TB Unit.
4. Children diagnosed with LTBI or children under 5 years of age who have had close contact with TB should be offered preventive treatment as per jurisdictional guidelines. Children under 5 years of age who have had contact with TB and who are TST negative should have another TST performed 3 months after their first contact. If this second test is negative, then preventive treatment can generally be ceased.
5. Children who are released from immigration detention and have not been screened for LTBI (i.e. they were not contacts or attending school) should be referred to a local TB Unit to be screened and managed according to local guidelines.

Acknowledgements

National Tuberculosis Advisory Committee members (in alphabetical order): Associate Professor Anthony Allworth, Dr Ral Antic, Dr Ivan Bastian, Mr Philip Clift, Dr Jo Cochrane, Dr Chris Coulter (Chair), Dr Paul Douglas, Dr Justin Denholm, Dr Steve Graham, Clinical Associate Professor Mark Hurwitz, Dr Vicki Krause, Mr Chris Lowbridge, Ms Rhonda Owen, Dr Richard Stapledon, Dr David Stock, Mr Peter Trevan, Dr Justin Waring and the NTAC Secretariat from the Department of Health

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References

1. Tadolini M, Migliori GB. Chapter 18. The WHO strategy for TB control and elimination. *European Respiratory Monograph* 2012;58:242–253.
2. Amanatidou V, Syridou G, Mavrikou M, Tsolia MN. Latent tuberculosis infection in children: diagnostic approaches. *Euro J Clin Microbiol Infect Dis* 31(7):1285–1294.
3. World Health Organization. *Global tuberculosis report 2013*. Geneva: World Health Organization; 2013.
4. Diel R, Nienhaus A. Chapter 6. Prevention of TB in areas of low incidence. *European Respiratory Monograph* 2012;58:72–83.
5. Rutherford ME, Hill PC, Triasih R, Sinfield R, van Crevel R, Graham SM, et al. Preventive therapy in children exposed to *Mycobacterium tuberculosis*: problems and solutions. *Trop Med Int Health* 2012;17(10):1264–1273.
6. Munoz FM, Starke JR. Tuberculosis in children. In: Reichman LB, Hershfield ES, eds. *Tuberculosis: A Comprehensive International Approach*. New York: Marcel Dekker, Inc; 2000. p. 553–586.
7. Marais BJ, Gie RP, Schaaf HS, Hesseling AC, Obihara CC, Starke JJ, et al. The natural history of childhood intra-thoracic tuberculosis: a critical review of literature from the pre-chemotherapy era. *Int J Tuberc Lung Dis* 2004;8(4):392–402.
8. Pang J, Teeter LD, Katz DJ, Davidow AL, Miranda W, Wall K, et al. Epidemiology of tuberculosis in young children in the United States. *Pediatrics* 2014;133(3):e494–e504.
9. National Tuberculosis Advisory Committee. The strategic plan for control of tuberculosis in Australia: 2011–2015. *Commun Dis Intell* 2012;36(3):E286–E293.
10. National Tuberculosis Advisory Committee. Position statement on interferon-γ release assays in the detection of latent tuberculosis infection. *Commun Dis Intell* 2012;36(1):125–131.
11. Targeted tuberculin skin testing and treatment of latent tuberculosis infection in children and adolescents. *Pediatrics* 2004;114(Suppl 4):1175–1201.
12. Victorian Government Department of Health. Tuberculosis management, prevention and control of tuberculosis: Guidelines for health care providers 2002–2005. 2013. Accessed on 25 November. Available from: http://ideas.health.vic.gov.au/diseases/tuberculosis-management-guide.asp
13. Northern Territory Department of Health, Centre for Disease Control. Guidelines for the control of tuberculosis in the Northern Territory. Darwin: Centres for Diseases Control, Department of Health; 2008.
14. Kimbrough W, Saliba V, Dahab M, Haskew C, Checchi F. The burden of tuberculosis in crisis-affected populations: a systematic review. *Lancet Infect Dis* 2012;12(1):950–965.

REVISED SURVEILLANCE CASE DEFINITIONS

This report provides the revised Surveillance case definitions approved by the Communicable Diseases Network Australia (CDNA) since 1 July 2015.

The Case Definitions Working Group (CDWG) is a subcommittee of the CDNA and comprises members representing all states and territories, the Australian Government Department of Health, the Public Health Laboratory Network, OzFoodNet, the Kirby Institute, the National Centre for Immunisation Research and Surveillance and other communicable disease experts. CDWG develops and revises surveillance case definitions for all diseases reported to the National Notifiable Diseases Surveillance System. Surveillance case definitions incorporate laboratory, clinical and epidemiological elements as appropriate.

The following case definitions have been reviewed by CDWG and endorsed by CDNA.

These case definitions will be implemented on 1 January 2016 and supersede any previous versions.

Barmah Forest virus infection case definition

Reporting

Both **confirmed cases** and **probable cases** should be notified.

Confirmed case

A confirmed case requires **laboratory definitive evidence** only.

Probable case

A probable case requires **laboratory suggestive evidence** only.

Laboratory definitive evidence

Isolation of Barmah Forest virus

OR

Detection of Barmah Forest virus by nucleic acid testing

OR

IgG seroconversion or a significant increase in IgG antibody level (e.g. fourfold or greater rise in titre) to Barmah Forest virus.

Laboratory suggestive evidence

Detection of Barmah Forest virus IgM AND Barmah Forest virus IgG EXCEPT if Barmah Forest IgG is known to have been detected in a specimen collected greater than 3 months earlier.

Barmah Forest virus infection case definition changes	**New probable category** Laboratory **definitive** evidence now only includes detection by PCR and demonstrated seroconversions. A single IgM will no longer be included in this category. Laboratory **suggestive** evidence will require an IgM in the presence of IgG on the same specimen. Single IgM positive results will no longer meet the confirmed or probable case definition.

Ross River virus infection case definition

Reporting

Both **confirmed cases** and **probable cases** should be notified.

Confirmed case

A confirmed case requires **laboratory definitive evidence** only.

Probable case

A probable case requires **laboratory suggestive evidence** only.

Laboratory definitive evidence

Isolation of Ross River virus

OR

Detection of Ross River virus by nucleic acid testing

OR

IgG seroconversion or a significant increase in IgG antibody level (e.g. fourfold or greater rise in titre) to Ross River virus.

Laboratory suggestive evidence

Detection of Ross River virus IgM AND Ross River virus IgG EXCEPT if Ross River IgG is known to have been detected in a specimen collected greater than 3 months earlier.

Ross River virus infection case definition changes	**New probable category** Laboratory **definitive** evidence now only includes detection by PCR and demonstrated seroconversions. A single IgM will no longer be included in this category. Laboratory **suggestive** evidence will require an IgM in the presence of IgG on the same specimen. Single IgM positive results will no longer meet the confirmed or probable case definition.

Congenital rubella infection case definition

Congenital rubella infection is reported based on relevant evidence from a live or stillborn infant, miscarriage or pregnancy termination. Congenital rubella syndrome is reported as a subset of congenital rubella infection.

Reporting

Both **confirmed cases** and **probable cases** should be notified.

Confirmed case

A **confirmed case** requires **laboratory definitive evidence (fetal)**

OR

Laboratory definitive evidence (infant) AND **epidemiological evidence**

Laboratory definitive evidence

Fetal

Isolation or detection of rubella virus from an appropriate clinical sample (i.e. fetal blood or tissue, amniotic fluid, chorionic villus sample) by culture or nucleic acid testing

Infant

Isolation or detection of rubella virus from an appropriate clinical sample in an infant, by culture or nucleic acid testing.

OR

Detection of rubella-specific IgM antibody in the serum of the infant.

Epidemiological evidence

The mother has confirmed rubella infection during pregnancy (see definition for Rubella – non-congenital).

Probable case

A probable case requires

Epidemiological evidence (1st trimester infection)

OR

Epidemiological evidence (2nd and 3rd trimester infection) AND **laboratory suggestive evidence (infant)**

Laboratory suggestive evidence

Infant

High/rising rubella-specific IgG level in first year of life

Congenital rubella syndrome case definition

Reporting

Both **confirmed cases** and **probable cases** should be reported.

Confirmed case

A confirmed case requires **laboratory definitive evidence (fetal or infant),** as described above AND **clinical evidence**

Clinical evidence

A live or stillborn infant with ANY of the following compatible defects: cataract, congenital glaucoma, congenital heart disease, hearing defect, microcephaly, pigmentary retinopathy, developmental delay, purpura, hepatosplenomegaly, meningoencephalitis, radiolucent bone disease or other defect not better explained by an alternative diagnosis.

Probable case

A probable case requires **laboratory suggestive evidence (infant)** OR **epidemiological evidence,** as described above AND **clinical evidence**

Clinical evidence

(as for confirmed CRS case).

Congenital rubella infection changes	Case definition has been renamed 'Congenital Rubella Infection', with a subcategory of 'Congenital Rubella Syndrome'. Laboratory definitive evidence separated into fetal and infant. Laboratory suggestive evidence (maternal) reframed as epidemiological evidence and separated into 1st trimester versus 2nd/3rd trimester. Laboratory evidence criteria throughout amended to be consistent with PHLN case definition.

Salmonellosis case definition

Reporting

Only **confirmed cases** should be notified.

Confirmed case

A confirmed case requires **laboratory definitive evidence** only.

Laboratory definitive evidence

Isolation or detection of *Salmonella* species (excluding serotypes captured under the case definitions for typhoid and paratyphoid)

Salmonellosis changes	Revised to reflect the creation of a separate case definition for paratyphoid.

New surveillance case definition

Paratyphoid case definition

Reporting

Only **confirmed cases** should be notified

Confirmed case

A confirmed case requires **laboratory definitive evidence** only.

Laboratory definitive evidence

Isolation or detection of *Salmonella* Paratyphi A or *S.* Paratyphi B (excluding *S.* Paratyphi B biovar Java) or *S.* Paratyphi C.

Paratyphoid	Initial case definition (2015).

Annual reports

Influenza viruses received and tested by the Melbourne WHO Collaborating Centre for Reference and Research on Influenza annual report, 2014

Sheena G Sullivan, Michelle K Chow, Ian G Barr, Anne Kelso

Abstract

The WHO Collaborating Centre for Reference and Research on Influenza in Melbourne is part of the World Health Organization's (WHO) Global Influenza Surveillance and Response System. In 2014 the Centre received a total of 5,374 influenza samples from laboratories primarily in the Asia–Pacific region. Viruses were characterised by their antigenic, genetic and antiviral drug resistance properties. Of the viruses successfully analysed 52% were A(H1N1)pdm09 viruses. The majority of these were antigenically and genetically similar to the WHO recommended reference strain for the 2014 Southern Hemisphere influenza vaccine. Results for A(H3N2) and B/Yamagata viruses suggested that circulating viruses of this subtype and lineage, respectively, had undergone antigenic and/or genetic changes, consistent with the decision by WHO to change recommended strains for the 2015 Southern Hemisphere vaccine. A small number of A(H1N1)pdm09 and B/Victoria viruses had highly reduced inhibition to the neuraminidase inhibitors oseltamivir and zanamivir. The Centre also undertook primary isolation of vaccine candidate viruses directly into eggs. A total of 38 viruses were successfully isolated in eggs, of which 1 (B/Phuket/3073/2013) was included in the 2015 Southern Hemisphere influenza vaccine. *Commun Dis Intell* 2015;39(4):E584–E593.

Introduction

The WHO Collaborating Centre for Reference and Research on Influenza in Melbourne (the Centre) is part of the World Health Organization's Global Influenza Surveillance and Response System (WHO GISRS). The GISRS network, established in 1952, monitors changes in influenza viruses with the aim of reducing the impact of influenza through the use of vaccines and antiviral medications. The Centre in Melbourne was first designated as a collaborating centre in 1992, the third such centre in the world. There are now 5 collaborating centres (Atlanta, Beijing, London, Melbourne and Tokyo) that analyse influenza viruses currently circulating in the human population. Virus samples are submitted to WHO collaborating centres by WHO National Influenza Centres and other hospital and regional laboratories. Based on data and advice from the 5 collaborating centres and other experts, the WHO makes biannual recommendations on suitable influenza strains to be included in the next seasonal vaccine (in February for the Northern Hemisphere and in September for the Southern Hemisphere). To this end, the Centre in Melbourne conducts a range of virological surveillance activities. This report summarises those surveillance activities undertaken in 2014.

Virological surveillance performed by the Centre

Two types of influenza cause significant disease in humans: types A and B. Influenza A viruses are further classified into subtypes, based on their surface proteins, haemagglutinin (H) and neuraminidase (N). Thus, currently in circulation are subtypes A(H1N1)pdm09 and A(H3N2), although a number of subtypes have been known to infect humans and birds. Influenza B viruses are not classified into subtypes. However, there are currently two co-circulating lineages, B/Victoria/2/87 (B/Victoria) and B/Yamagata/16/88 (B/Yamagata). In addition, each year some cases of influenza C are isolated from humans, but as these viruses tend not to cause severe disease, they are not a focus of surveillance.

Virus isolation

All virus isolates received at the Centre were re-passaged in cell culture (Madin-Darby Canine Kidney [MDCK] cells) and virus isolation was also attempted on a selection of original clinical specimens received. In addition, influenza-positive original clinical samples were directly inoculated into eggs as potential vaccine strains.

Antigenic analysis

The antigenic properties of influenza viral isolates were analysed using the haemagglutination

inhibition (HI) assay as previously described.[1] In HI assays, viruses were tested for their ability to agglutinate red blood cells in the presence of ferret antisera previously raised against reference viruses. Isolates were identified as antigenically similar to the reference strain if the test samples had a titre that was no more than 4-fold different from the titre of the homologous reference strain. During 2014, results were reported by reference to the A/California/7/2009 (H1N1pdm09)-like, A/Victoria/361/2012 (H3N2)-like, B/Massachusetts/2/2012-like (Yamagata lineage), and B/Brisbane/60/2008-like (Victoria lineage) viruses that were recommended for the 2014 influenza vaccine.

Genetic analysis

A subset of all influenza viruses analysed at the Centre underwent genetic analysis by sequencing the viral RNA genes. Viruses exhibiting evidence of antigenic variation or reduced antiviral drug susceptibility, as well as representative viruses from different time periods and geographic locations, were selected for sequencing of the haemagglutinin gene and/or the neuraminidase gene. Routine sequencing of the matrix protein for influenza A viruses and non-structural protein genes for influenza B viruses was also performed. In addition, the full genomes of a smaller subset of viruses were sequenced.

For sequencing, RNA was extracted from isolates or original clinical specimens using the QIAGEN Xtractor Gene robot, followed by reverse transcription polymerase chain reaction (RT-PCR) using the BIOLINE MYTAQ one step RT-PCR kit according to the manufacturer's recommendations with gene specific haemagglutinin primers (primer sequences available on request). Conventional Sanger sequencing was carried out on PCR product using an Applied Biosystems 3500 XL sequencer, and sequence assembly performed using the Seqman Pro Module of DNASTAR Lasergene version 9.1.0 software (DNAStar, Madison, WI, USA). Phylogenetic analysis was performed using MEGA 5.[2]

Antiviral drug resistance testing

As the potential evolution of influenza viruses to develop resistance to antiviral drugs is of ongoing concern, circulating viruses were tested for their sensitivity to the currently used neuraminidase inhibitors oseltamivir (Tamiflu) and zanamivir (Relenza). The neuraminidase inhibition (NAI) assay used was a functional fluorescence-based assay in which the susceptibility of test viruses to the antiviral drugs was measured in terms of the drug concentration needed to reduce the neuraminidase enzymatic activity by 50% (IC_{50}), and compared to values obtained with sensitive reference viruses of the same subtype or lineage. NAI assays were performed as previously described.[3] For the purposes of reporting, reduced inhibition of influenza A viruses was defined as a 10–99 fold increase in IC_{50}, while highly reduced inhibition was defined as a $\geq$ 100-fold increase in IC_{50} in a NAI assay. For influenza B viruses, these figures were 5–49-fold and $\geq$ 50-fold increases, respectively. However, it should be noted that the relationship between the IC_{50} value and the clinical effectiveness of a neuraminidase inhibitor is not well understood and reduced inhibition may not be clinically significant.

Viruses found to have highly reduced inhibition by either oseltamivir or zanamivir underwent further analysis to determine the presence of amino acid substitutions in the neuraminidase protein that were associated with the reduction of inhibition by NAI assays. For example, a change from histidine to tyrosine at position 275 (H275Y) of the neuraminidase protein of A(H1N1)pdm09 viruses is known to reduce inhibition by oseltamivir, as does the H273Y NA mutation in influenza B viruses.[4]

Candidate vaccine strains

The viruses used to produce human vaccines are required to be isolated and passaged in embryonated hen's eggs or certified cell lines. The Centre undertook primary isolation of selected viruses from clinical samples directly into eggs, using previously described methods.[5] These isolates were then analysed by HI assay and genetic sequencing.

Results

During 2014, a total of 5,374 clinical specimens and/or virus isolates were received by the Centre from 36 laboratories in 14 countries (Figure 1). Most of the samples were provided by laboratories in the Asia–Pacific region. Figure 2 shows the temporal distribution of samples sent to the Centre by subtype and lineage. Most samples were received during the Australian influenza season.

Influenza A(H1N1)pdm09

Virus isolation was attempted for 4,899 (91%) of the samples received and was successful in 3,382 cases (69%). Of these, 3,374 were characterised by HI assay (Table). In addition, 27 samples were characterised by RT-PCR. Some 444 haemagglutinin genes were sequenced. Full genome sequencing using Sanger sequencing techniques was performed on 63 viruses. Of the samples for which results could be obtained (n=3,404), 52%

were identified as A(H1)pdm09, 29% were A(H3) viruses, 17% were B/Yamagata, 2% were B/Victoria and there was 1 sample with mixed H3/B viruses.

Of the 1,761 A(H1)pdm09 isolates analysed by HI assay in 2014, the majority (99.5%) were antigenically similar to the vaccine reference strain A/California/7/2009 (Table).

Sequencing and phylogenetic analysis of haemagglutinin genes from 168 viruses showed that A(H1N1)pdm09 viruses sent to the Centre during 2014 contained some minor genetic changes compared with A/California/7/2009 (Figure 3). However, the antigenic behaviour of these viruses was not affected by these changes.

Figure 1: Geographic spread of influenza laboratories sending viruses to the Centre during 2014

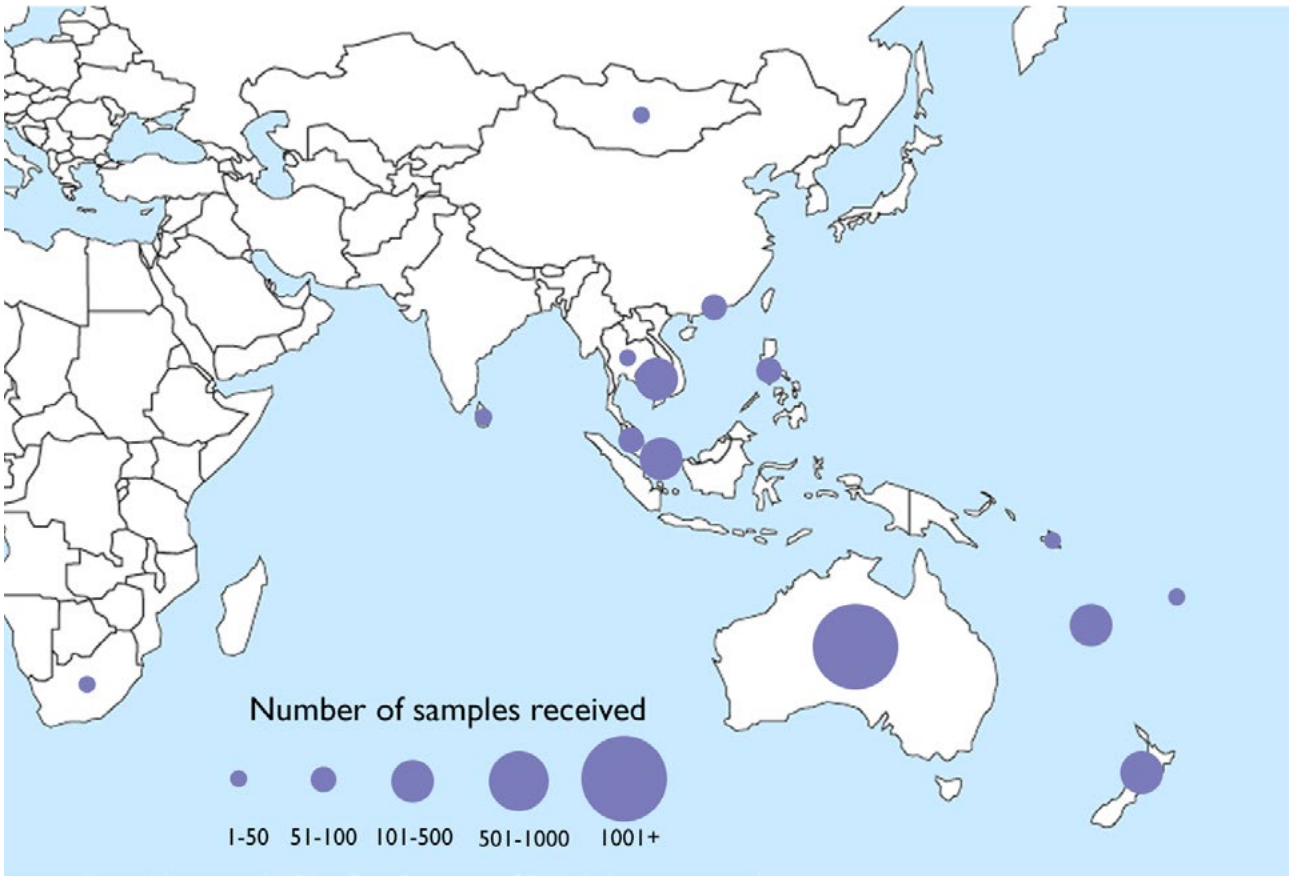
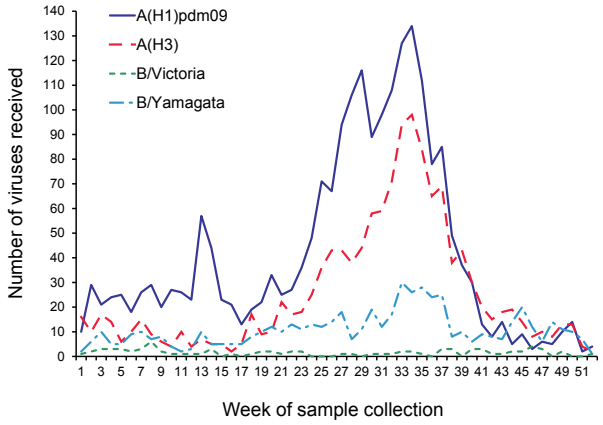


Table: Antigenic analysis of viruses received by the Centre, by country of origin

Region	A(H1)pdm09: A/California/7/2009 (cell)		A(H3) : A/Texas/50/2012 (cell)		B/Victoria: B/Brisbane/60/2008 (cell)		B/Yamagata: B/Massachusetts/2/2012 (cell)	
	Like	Low reactor (%)	Like	Low reactor (%)	Like	Low reactor (%)	Like	Low reactor (%)
Australasia	1,492	7 (0.5%)	647	41 (5.9%)	32	0	131	243 (65%)
Pacific	54	0	185	5 (2.6%)	2	0	3	6 (67%)
South East Asia	175	1 (0.6%)	30	17 (3.6%)	29	1 (3.3%)	98	66 (40%)
East Asia	25	1 (3.8%)	18	2 (10%)	16	0	5	12 (71%)
South Asia	2	0	8	0	0	0	0	0 (0%)
Africa	4	0	11	0	0	0	1	2 (67%)
Total	1,752	9 (0.5%)	899	65 (6.7%)	79	1 (1.3%)	238	329 (58%)

Figure 2: Number of samples received at the Centre, 2014, by week of sample collection



Nineteen viruses were inoculated into eggs for vaccine candidate strain isolation. Of these, 10 (53%) were successfully isolated and included at least 1 virus from each of the 3 clades represented in the dendrogram (Figure 3).

Of 1,743 H1 viruses tested, seven exhibited highly reduced inhibition by oseltamivir. These viruses were from Australia (Perth, n=3), Macau, Malaysia, Thailand, and New Caledonia. All of these viruses had the H275Y mutation. No H1 viruses showed highly reduced inhibition by zanamivir.

Influenza A(H3N2)

Antigenic analysis of 964 A(H3) subtype isolates showed that only 7% were low reactors to the cell-propagated reference strain A/Texas/50/2012 (Table). However, 24% of viruses were low reactors to the egg-propagated strain A/Texas/50/2012.

A total of 155 haemagglutinin genes from A(H3N2) viruses were sequenced. Phylogenetic analysis indicated that recently circulating viruses had undergone significant genetic changes compared with A/Texas/50/2012 (Figure 4). These viruses fell into one of 3 clades, designated 3C.2a and 3C.3a and 3C.3b, while A/Texas/50/2012 was in clade 3C.1.

Seventy-eight viruses were inoculated into eggs, of which 23 (29%) grew successfully. These viruses were representative of each of the clades identified in Figure 4 and included at least 4 viruses from clade 3C.2a and 2 viruses from clade 3C.3a.

None of the 969 H3 viruses tested had highly reduced inhibition by either of the neuraminidase inhibitors.

Influenza B

There are currently 2 antigenically and genetically distinct lineages of influenza B virus in circulation, the B/Victoria/2/87 lineage (represented by B/Brisbane/60/2008) and the B/Yamagata/16/88 lineage (represented by the southern hemisphere 2014 vaccine strain B/Massachusetts/2/2012). Among influenza B viruses received at the Centre during 2014, the B/Yamagata lineage was predominant (Table).

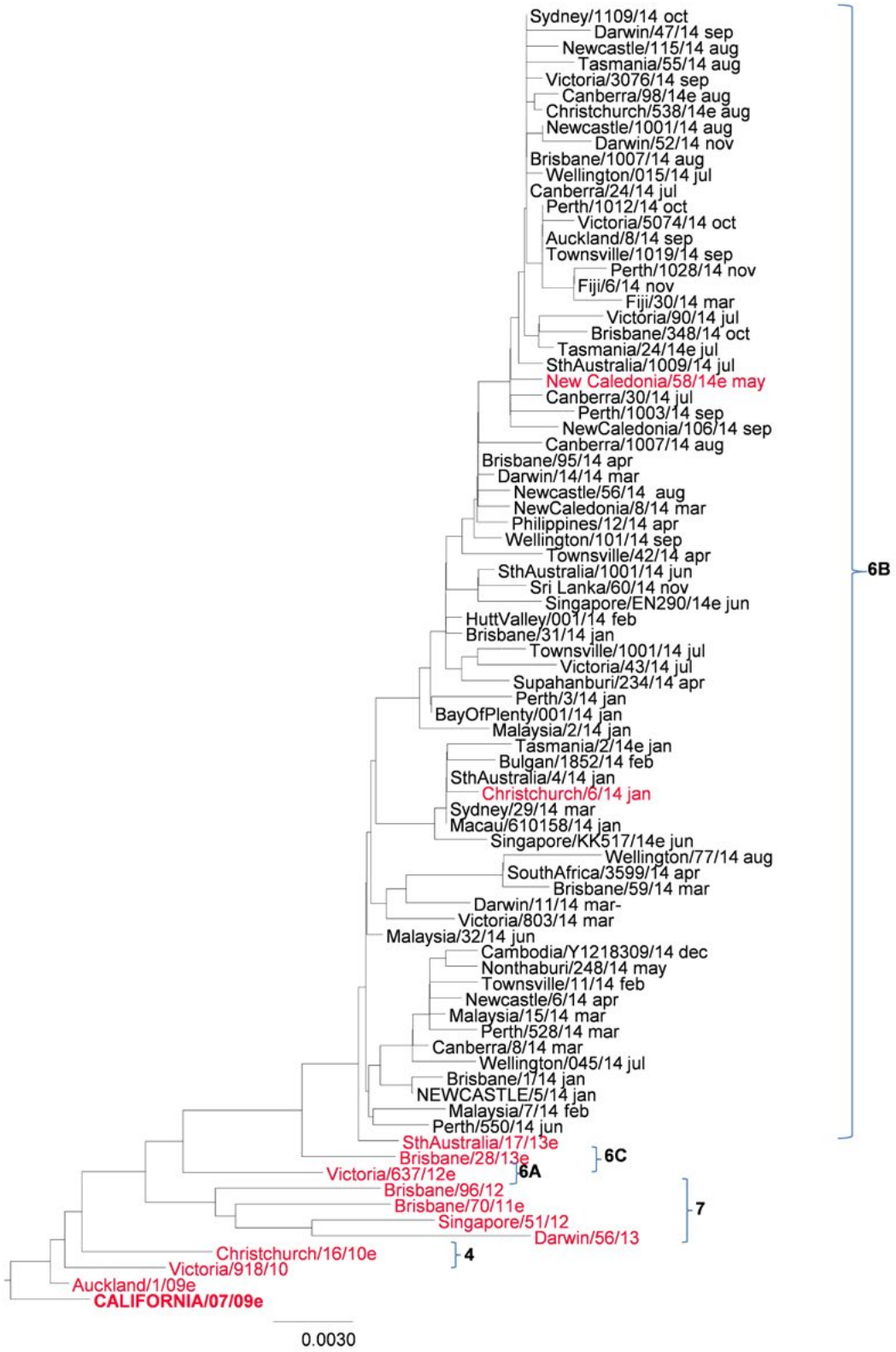
A total of 647 influenza B viruses were characterised by HI assay. All but one of the 80 B/Victoria viruses received and analysed were similar to B/Brisbane/60/2008 (Table). The majority (58%) of the 567 B/Yamagata lineage viruses analysed were low reactors to the B component of the 2014 vaccine, B/Massachusetts/2/2012 (Table). In contrast, roughly 66% were antigenically similar to the 2013 vaccine strain, B/Wisconsin/1/2010.

Sequencing was performed on 121 haemagglutinin genes from B viruses, the majority being B/Yamagata viruses. All of the viruses of B/Victoria lineage were genetically similar to the B/Brisbane/60/2008 reference virus (Figure 5). The majority of B/Yamagata lineage viruses belonged to Clade 3 and were antigenically and genetically distinct from the 2014 vaccine strain B/Massachusetts/2/2012 (Clade 2; Figure 6). Clade 3 includes B/Phuket/3073/2013, which is the B strain included in the 2015 vaccine, as well as B/Wisconsin/1/2010, which was the B strain used in the 2013 influenza vaccine.

Egg isolation was attempted for 7 B/Victoria and 16 B/Yamagata viruses. None of the B/Victoria viruses were successfully isolated, while 31% of the B/Yamagata viruses were isolated in eggs. One of these was the B/Phuket/3073/2013, the virus that was included in the 2015 Southern Hemisphere influenza vaccine.

Of 80 B/Victoria viruses tested, one from Mongolia displayed highly reduced inhibition by both oseltamivir and zanamivir. This virus had the G104R mutation in the NA gene. None of the 566 B/Yamagata viruses tested showed highly reduced inhibition by the neuraminidase inhibitors.

Figure 3: Phylogenetic tree of representative haemagglutinin genes of A(H1N1)pdm09 viruses, 2014



2015 Southern Hemisphere vaccine strain is presented in capital letters

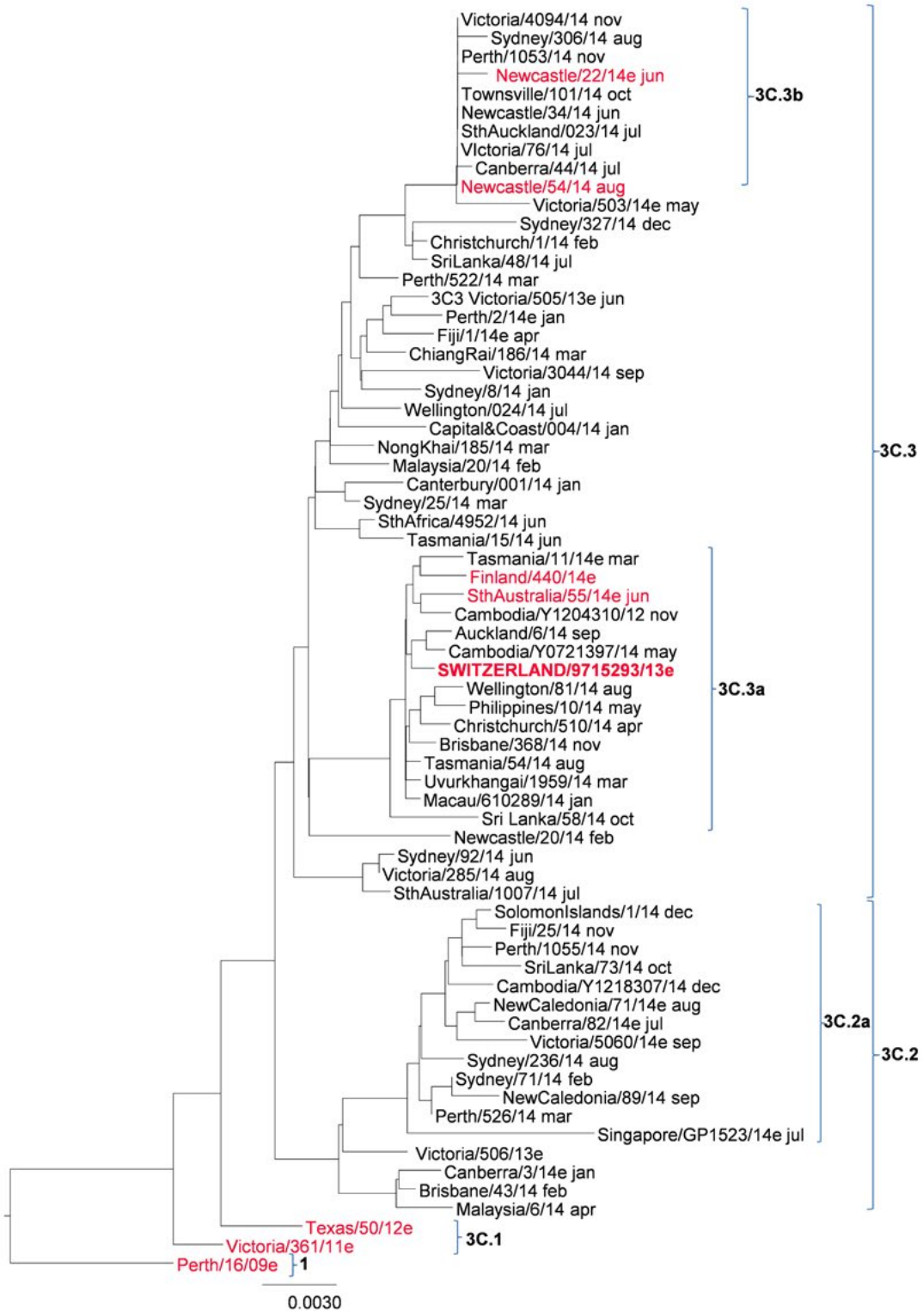
Reference virus is indicated in red text

e: egg isolate

Scale bar represents 0.3% nucleotide sequence difference between viruses

} Braces indicate clades

Figure 4. Phylogenetic tree of representative haemagglutinin genes of A(H3N2) viruses, 2014



2015 Southern Hemisphere vaccine strain is presented in capital letters

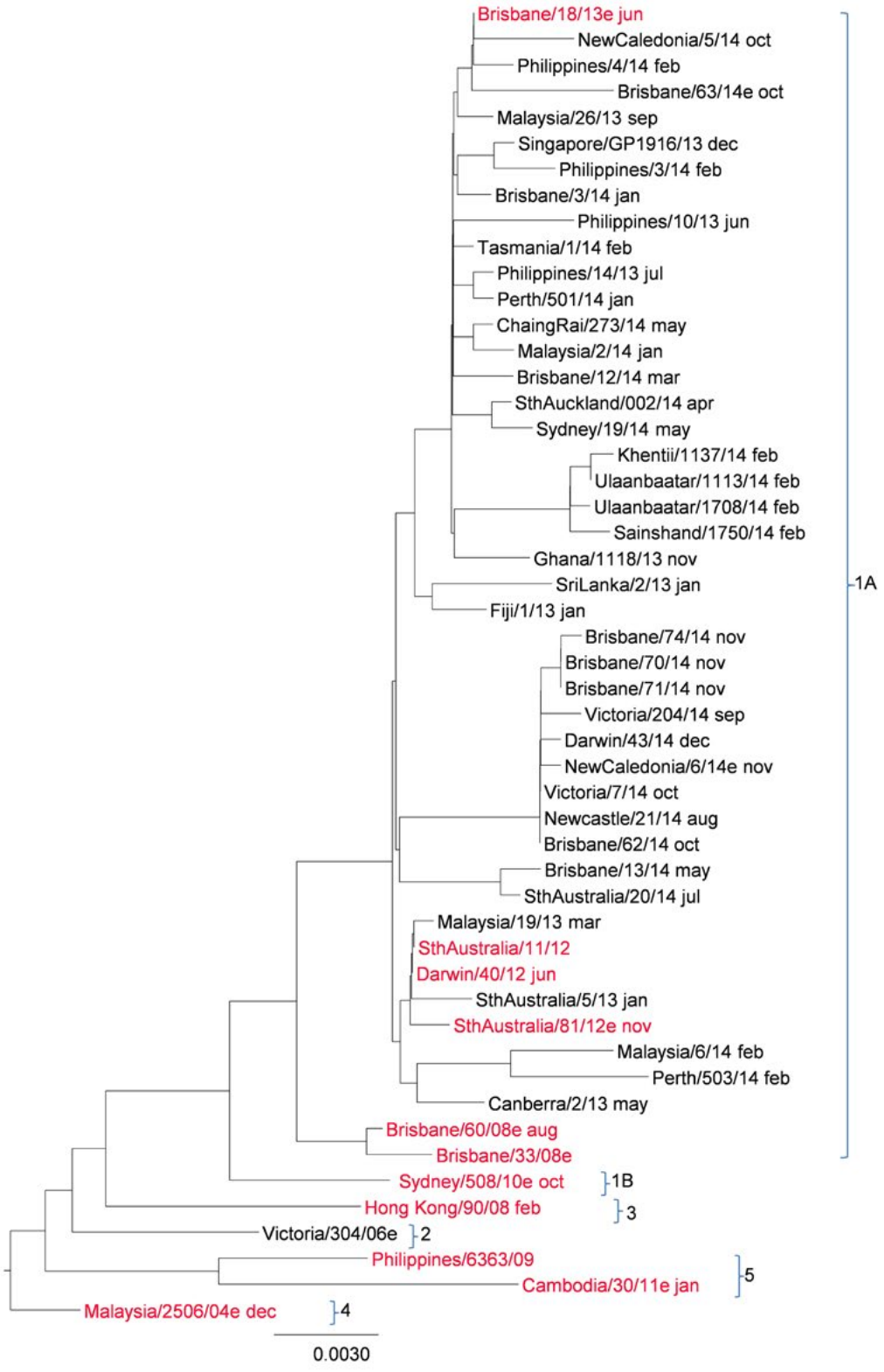
Reference virus is indicated in red text

e: egg isolate

Scale bar represents 0.3% nucleotide sequence difference between viruses

} Braces indicate clades

Figure 5. Phylogenetic tree of representative haemagglutinin genes of B/Victoria viruses, 2014



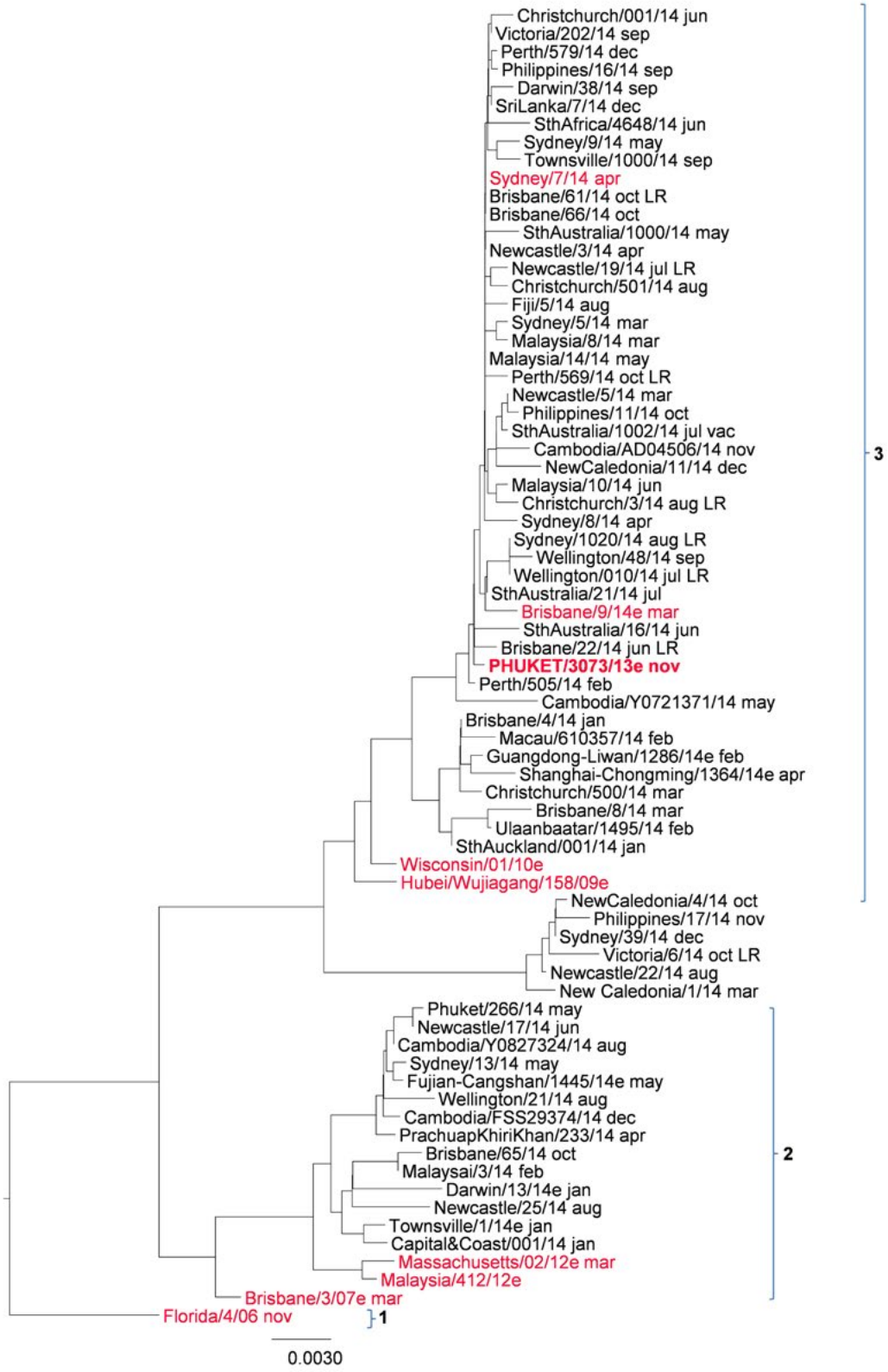
Reference virus is indicated in red text

e: egg isolate

Scale bar represents 0.3% nucleotide sequence difference between viruses

} Braces indicate clades

Figure 6. Phylogenetic tree of representative haemagglutinin genes of B/Yamagata viruses, 2014



2015 Southern Hemisphere vaccine strain is presented in capital letters

Reference virus is indicated in red text

e: egg isolate

Scale bar represents 0.3% nucleotide sequence difference between viruses

} Braces indicate clades

Discussion

The viruses analysed by the Centre were heavily dominated by samples received from Australian laboratories. The National Notifiable Diseases Surveillance System (NNDSS) data indicated that in 2014 Australia had the highest influenza activity on record, with 67,709 notifications of laboratory confirmed influenza.[6] This was reflected in the large number of samples received at the Centre, second only to 2009, the year of the A(H1N1)pdm09 pandemic, when 6,435 samples were received.

National Influenza Centre data indicated that A(H1)pdm09 viruses predominated in Australia in 2014.[7] This was reflected in the samples received by the Centre, where the majority of viruses received were A(H1)pdm09. Antigenic and genetic data indicated a good match between the vaccine strain and the circulating strains. Indeed, interim estimates from New Zealand, which also used the WHO-recommended Southern Hemisphere vaccine composition, observed that the vaccine's effectiveness was good (73% [95%CL: 50,85]).[8]

Despite the overall predominance of A(H1N1) pdm09 viruses during 2014, influenza A(H3N2) predominated in New South Wales and the Australian Capital Territory, and towards the end of the year an increasing number of A(H3N2) viruses from Queensland, Western Australia, the Northern Territory and Tasmania were reported to NNDSS.[7] As a result, the proportion of H3 viruses received at the Centre among all viruses increased towards the end of the 2014 season.

HI assays performed at the Centre showed that at least 75% of H3 viruses tested were antigenically similar to the egg-grown A/Texas/50/2011 strain. However, recently there have been considerable challenges in the antigenic characterisation of H3 viruses. Evolutionary changes in this subtype have made it difficult to detect antigenic changes using the HI assay.[9] Although other assays (e.g. virus neutralisation) are being used to examine the antigenicity of H3 viruses, these methods are more labour intensive and cannot easily replace the HI assay. Studies are continuing to determine the most appropriate method for detecting antigenic changes in recent H3 viruses, but more emphasis is being placed on genetic analysis. Genetic data from the Centre indicated that many 2014 H3 viruses fell in clades 3C.2a, 3C.3a and 3C.3b (the 2015 Southern Hemisphere vaccine strain, A/Switzerland/9715293/2013, lies in clade 3C.3a). The clades appear to be antigenically distinct. Reports in early 2015 from several Northern Hemisphere countries suggested low effectiveness against A(H3N2) for the vaccines containing A/Texas/50/2012.[10–13] This has been attributed to increased circulation of clade 3C.2a viruses, while the Texas strain is a clade 3C.1 virus.[10]

A further challenge in the development of H3 vaccine candidates has arisen in recent years. When isolated in eggs, human A(H3N2) viruses rapidly acquire adaptive changes in the haemagglutinin, which may alter antigenicity. This has been a problem for a long time but was a particular issue with the A/Victoria/361/2011 vaccine virus included in the 2013 vaccine.[14] This has stimulated a major effort to obtain H3 egg isolates that retain the antigenic properties of their corresponding cell isolates.

Finally, a majority of influenza B/Yamagata-lineage viruses received by the Centre appeared to be antigenically distant from the vaccine virus, B/Massachusetts/2/2012. This virus was selected for the vaccine in light of evidence that B/Massachusetts/2/2012-like viruses were increasingly prevalent in 2013. However, viruses that were antigenically more similar to the 2013 vaccine strain B/Wisconsin/1/2010 unexpectedly dominated in 2014. These 2 strains are both B/Yamagata viruses, but sit within different clades in the phylogenetic tree. There are no published vaccine effectiveness estimates available for influenza B during the 2014 or 2014/15 seasons. Genetic analysis by the Centre showed that many of the viruses were genetically similar to the egg-propagated B/Phuket/3073/2013 virus isolated at the Centre and this virus was subsequently recommended for inclusion in the Southern Hemisphere 2015 vaccine.

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References

1. Hobson D, Curry RL, Beare AS, Ward-Gardner A. The role of serum haemagglutination-inhibiting antibody in protection against challenge infection with influenza A2 and B viruses. *J Hyg (Lond)* 1972;70(4):767–777.
2. Tamura K, Peterson D, Peterson N, Stecher G, Nei M, Kumar S. MEGA5: molecular evolutionary genetics analysis using maximum likelihood, evolutionary distance, and maximum parsimony methods. *Mol Biol Evol* 2011;28(10):2731–2739.

3. Hurt AC, Okomo-Adhiambo M, Gubareva LV. The fluorescence neuraminidase inhibition assay: a functional method for detection of influenza virus resistance to the neuraminidase inhibitors. *Methods Mol Biol* 2012;865:115–125.
4. Takashita E, Meijer A, Lackenby A, Gubareva L, Rebelo-de-Andrade H, Besselaar T, et al. Global update on the susceptibility of human influenza viruses to neuraminidase inhibitors, 2013–2014. *Antiviral Res* 2015;117:27–38.
5. WHO Global Influenza Surveillance Network. *Manual for the laboratory diagnosis and virological surveillance of influenza*. Geneva: World Health Organization; 2011.
6. Australian Government Department of Health. National Notifiable Diseases Surveillance System. 2015. Accessed on 6 May 2015. Available from: http://www9.health.gov.au/cda/source/cda-index.cfm
7. Australian Government Department of Health. Australian Influenza Surveillance Report. No. 8, 2014, 27 September to 10 October 2014; 2014.
8. Turner N, Pierse N, Huang QS, Radke S, Bissielo A, Thompson MG, et al. Interim estimates of the effectiveness of seasonal trivalent inactivated influenza vaccine in preventing influenza hospitalisations and primary care visits in Auckland, New Zealand, in 2014. *Euro Surveill* 2014;19(42) pii: 20934.
9. Barr IG, Russell C, Besselaar TG, Cox NJ, Daniels RS, Donis R, et al. WHO recommendations for the viruses used in the 2013–2014 Northern Hemisphere influenza vaccine: Epidemiology, antigenic and genetic characteristics of influenza A(H1N1)pdm09, A(H3N2) and B influenza viruses collected from October 2012 to January 2013. *Vaccine* 2014;32(37):4713–4725.
10. Skowronski DM, Chambers C, Sabaiduc S, De Serres G, Dickinson JA, Winter AL, et al. Interim estimates of 2014/15 vaccine effectiveness against influenza A(H3N2) from Canada s Sentinel Physician Surveillance Network, January 2015. *Euro Surveill* 2015;20(4): pii: 21022.
11. Pebody R, Warburton F, Ellis J, Andrews N, Thompson C, von Wissmann B, et al. Low effectiveness of seasonal influenza vaccine in preventing laboratory-confirmed influenza in primary care in the United Kingdom: 2014/15 mid-season results. *Euro Surveill* 2015;20(5):21025.
12. McNeil SA, Andrew MK, Ye L, Haguinet F, Hatchette TF, ElSherif M, et al. Interim estimates of 2014/15 influenza vaccine effectiveness in preventing laboratory-confirmed influenza-related hospitalisation from the Serious Outcomes Surveillance Network of the Canadian Immunization Research Network, January 2015. *Euro Surveill* 2015;20(5):21024.
13. Flannery B, Clippard J, Zimmerman RK, Nowalk MP, Jackson ML, Jackson LA, et al. Early estimates of seasonal influenza vaccine effectiveness—United States, January 2015. *MMWR Morb Mortal Wkly Rep* 2015;64(1):10–15.
14. Skowronski DM, Janjua NZ, De Serres G, Sabaiduc S, Eshaghi A, Dickinson JA, et al. Low 2012–13 influenza vaccine effectiveness associated with mutation in the egg-adapted H3N2 vaccine strain not antigenic drift in circulating viruses. *PLoS One* 2014;9(3):e92153.

Quarterly report

OzFoodNet quarterly report, 1 January to 31 March 2014

The OzFoodNet Working Group

Introduction

The Australian Government Department of Health established the OzFoodNet network in 2000 to collaborate nationally to investigate foodborne disease. In each Australian state and territory OzFoodNet epidemiologists investigate outbreaks of enteric infection. OzFoodNet conducts studies on the burden of illness and coordinates national investigations into outbreaks of foodborne disease. This quarterly report documents investigations of outbreaks of gastrointestinal illness and clusters of disease potentially related to food, which commenced in Australia between 1 January and 31 March 2014.

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

During the 1st quarter of 2014, OzFoodNet sites reported 465 outbreaks of enteric illness, including those transmitted by contaminated food. Outbreaks of gastroenteritis are often not reported to health agencies or the reports may be delayed, meaning that these figures under-represent the true burden of enteric disease outbreaks. In total, these outbreaks affected 7,233 people, of whom 299 were hospitalised. There were 20 deaths reported during these outbreaks. The majority of outbreaks (n=335) were due to person-to-person transmission (Table 1), with 46% (155/335) of these occurring in child care facilities or schools and 42% (142/335) occurring in aged care facilities.

Foodborne and suspected foodborne disease outbreaks

There were 49 outbreaks during this quarter where consumption of contaminated food was suspected or confirmed as being the primary mode of transmission (Appendix). These outbreaks affected 721 people and resulted in 125 hospitalisations. Two deaths were reported during these outbreaks. This was an increase on the number of outbreaks that were reported in the 1st quarter of 2013 (n=34) and an increase on the 5-year mean for the 1st quarter between 2009 and 2013 (n=44). A limitation of the outbreak data provided by OzFoodNet sites for this report was the potential for variation in the categorisation of the features of outbreaks depending on circumstances and investigator interpretation. Changes in the number of foodborne outbreaks should be interpreted with caution due to the small number each quarter.

Salmonella Typhimurium was identified as, or suspected to be, the aetiological agent in 67% (33/49) of foodborne or suspected foodborne outbreaks during this quarter, a higher proportion than the number from the same quarter in 2013 (50%, 17/34). The aetiological agents for the remaining outbreaks included: ciguatoxin in 5 outbreaks; *Campylobacter* species and suspected bacterial enterotoxins in 2 outbreaks each; and *Salmonella*

Table 1: Outbreaks and clusters of gastrointestinal illness and number ill reported by OzFoodNet, 1 January to 31 March 2014, by mode of transmission

Transmission mode	Number of outbreaks and clusters	Per cent of total*	Number ill
Foodborne and suspected foodborne	49	11	721
Suspected waterborne	1	<1	16
Person-to-person	335	72	5,514
Unknown (*Salmonella* cluster)	13	3	301
Unknown (Other pathogen cluster)	2	<1	17
Unknown	65	14	664
Total	465	100	7,233

* Percentages do not add to 100 due to rounding.

sub species 1, *S.* Singapore, Shiga-toxin producing *Escherichia coli* (STEC), *Shigella sonnei*, histamine and *Escherichia coli* for 1 outbreak each. For 1 outbreak the aetiological agent was unknown. The 6 outbreaks associated with fish toxins affected 45 people, with 4 occurring in Queensland and the remaining two occurring in New South Wales. In comparison, there was only 1 fish toxin outbreak (ciguatoxin) recorded in the 1st quarter of 2013, which affected 4 people.

Nineteen outbreaks (39% of all the foodborne or suspected foodborne outbreaks) reported in this quarter were associated with food prepared in restaurants (Table 2), which was the same as the average number associated with foodborne or suspected foodborne outbreaks in the 1st quarter from 2009 to 2013 (39%, 85/218).

To investigate these outbreaks, sites conducted 9 cohort studies, 1 case control study and collected descriptive case series data for 34 investigations, while for 5 outbreaks no individual patient data were collected. The evidence used to implicate food vehicles included analytical evidence in 1 outbreak, microbiological evidence in 8 outbreaks, and descriptive evidence in 40 outbreak investigations.

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

Australian Capital Territory

There were 3 outbreaks of foodborne or suspected foodborne illness reported in the Australian Capital Territory during this quarter. The aetiological agents identified were *E. coli*, *Campylobacter jejuni* and suspected bacterial enterotoxin.

Description of key outbreaks

An outbreak was investigated in January following a complaint to public health authorities of gastroenteritis among 3 diners who had eaten from the same takeaway kebab store. Symptoms included diarrhoea, vomiting and abdominal pain, with onsets occurring 12 to 30 hours after consuming kebabs. Elevated levels of *E. coli* were found in multiple samples of tabouli obtained during 3 separate inspections of the premises, resulting in a prohibition order being issued. It is suspected that foods eaten by cases had been either cross contaminated or contained pathogenic *E. coli*, with parsley used in the tabouli being a likely source.

An outbreak was investigated in February following reports of gastroenteritis among diners at a restaurant. There were 2 laboratory-confirmed cases of *C. jejuni* infection and 1 clinical case, with one of the confirmed cases being hospitalised. All cases had consumed the liver parfait. An inspection of the premises found that the liver parfait served on the day the cases reported their exposure had been prepared using chicken livers due to the unavailability of duck livers.

Table 2: Outbreaks of foodborne or suspected foodborne disease and number ill reported by OzFoodNet, 1 January to 31 March 2014, by food preparation setting

Food preparation setting	Outbreaks	Per cent of total	Number ill
Restaurant	19	39	475
Private residence	11	22	55
Takeaway	4	8	17
Bakery	3	6	60
Commercial caterer	2	4	29
Aged care facility	1	2	14
Private residence/restaurant	1	2	2
Camp	1	2	4
Cruise/airline	1	2	3
Institution	1	2	10
Community	1	2	12
Fair/festival	1	2	8
Other*	1	2	8
Unknown	2	4	24
Total	49	100	721

* Other - food prepared in a cooking class.

Food samples, including duck liver parfait, were taken for analysis, with the duck parfait sample found to be positive for *S.* Typhimurium phage type (PT) 9. *Campylobacter* spp. was not detected. No further campylobacteriosis cases or salmonellosis cases with links to the restaurant were identified. Undercooked chicken liver used in the parfait is the suspected cause of the campylobacteriosis cases.

New South Wales

There were 10 outbreaks of foodborne or suspected foodborne illness reported in New South Wales during this quarter. The aetiological agents were identified as *S.* Typhimurium for 6 outbreaks, ciguatoxin for 2 outbreaks and *Shigella sonnei and* STEC for 1 outbreak each.

Description of key outbreaks

An outbreak was investigated in January after 2 cases of gastroenteritis reported eating Vietnamese rolls from the same café. Active case finding via emergency department notifications and salmonellosis notifications identified a total of 24 people who all ate at the same café; 16 cases of *S.* Typhimurium multiple-locus variable number tandem repeat analysis (MLVA) profile 03-17-10-11-523 infection and another 8 clinical cases. An inspection of the premises identified that sanitiser was not in use for utensils and equipment. A sample of pâté taken at the time of the inspection was positive for *S.* Typhimurium MLVA profile 03-17-10-11-523. The pâté was made on site and it is possible the chicken liver was not cooked to a temperature necessary to kill any *Salmonella* present, so may have been the source of the outbreak.

An outbreak was investigated in February after half (8/16) of a group who ate food prepared during a cooking class developed gastroenteritis. Five of the 8 cases were positive for *S.* Typhimurium (4 MLVA profile 03-10-07-12-523 and 1 MLVA profile 03-11-07-12-523). All cases consumed various fish dishes with a raw egg mayonnaise. The food authority traced the eggs back to a producer and inspected the egg farm. *Salmonella* was detected on environmental swabs at the egg farm, including 1 boot swab of the egg laying shed positive for *S.* Typhimurium with the same MLVA profile as the cases. The farm was found to be in very good running order and no further improvements were suggested.

An outbreak was investigated in March after 10 cases of *S.* Typhimurium MLVA 03-09-07-12-523 infection clustered in time and location were identified, with 7 cases reporting to have eaten food from the same bakery. A range of foods were consumed including sandwiches with sliced deli meat, bread, sweets and hot foods. A food safety investigation identified a number of food handling issues including temperature abuse, cross contamination and inadequate equipment sanitisation. Two open food samples (sliced silverside and sliced roast beef) were positive for *S.* Typhimurium with a MLVA profile identical to the confirmed cases. It appears that cross contamination and poor temperature control likely resulted in a number of foods being contaminated. It is uncertain how the pathogen was initially introduced into the bakery.

Northern Territory

There were 2 outbreaks of suspected foodborne illness reported in the Northern Territory during this quarter. The aetiological agents were identified as *S.* Typhimurium for 1 outbreak and unknown for the other.

Queensland

There were 8 outbreaks of foodborne or suspected foodborne illness reported in Queensland during this quarter. The aetiological agents were identified as *S.* Typhimurium for 4 outbreaks, ciguatoxin for 3 outbreaks and histamine for 1 outbreak.

Description of key outbreak

A suspected foodborne outbreak was investigated in January after reports of gastrointestinal illness among 7 guests and 3 food handlers at a resort restaurant. Six cases were confirmed with *S.* Typhimurium PT 135a, MLVA profile 03-12-12-09-524 infection. Cases reported consumption of chicken (either chicken focaccia sandwiches and/or roasted chicken breast) at the resort restaurant and both meals were served with a raw egg-based sauce. The investigation identified that multiple sauces had been left out of temperature control for extended time periods as well as being used over consecutive days prior to this outbreak occurring. Several food samples including aioli, tartare sauce, ham, chicken, eggs and passionfruit cream were collected for microbiological testing. *S.* Typhimurium MLVA profile 03-12-12-09-524 was isolated from the sample of passionfruit cream; however, no cases had reported the consumption of this food item. This finding was considered an indicator of poor food handling practices that had occurred within the kitchen environment and was a contributing factor for this outbreak.

South Australia

There were 5 outbreaks of foodborne or suspected foodborne illness reported in South Australia during this quarter. The aetiological agents were identified as *S.* Typhimurium for 4 outbreaks and *Salmonella* sub species I for the other.

Description of key outbreaks

An outbreak was investigated in February following a notification of *S.* Typhimurium PT 135 infection reported in a food handler at a café and reports of another ill co-worker. A total of 4 confirmed cases of *S.* Typhimurium MLVA profile 03-12-09-11-523 infection reported consuming baguettes from this café. This included the 2 food handlers, who had multiple exposures within their incubation period. All baguettes made at the café contained a raw egg aioli. Samples of the eggs used to make aioli were taken and *S.* Typhimurium with an MLVA profile identical to the confirmed cases was found on the inside and outside of the egg.

An outbreak was investigated in March after authorities received complaints of gastroenteritis after people ate at a café. Active case finding identified 33 people who reported feeling unwell after eating at the café, with 19 confirmed as *S.* Typhimurium PT 9 (MLVA profile 03-26-15-09-523 and 03-26-11-09-523) infection. A case-control study was conducted with 1 meal (containing eggs, haloumi, pesto, avocado, rye bread, tomatoes and lime) significantly associated with illness, but the odds ratio (OR) was undefined as none of the well people ate the meal (95% confidence intervals [CI] 3.4 to undefined; P value=0.001). Consuming pesto in any meal was also significantly associated with illness with an undefined OR (95% CI 3.8 to undefined, P value=0.001). The environmental investigation found that the owners had recently changed and the new owners had limited knowledge of food safety. The stab mixer used to make the pesto was also used to regularly mix scrambled eggs, without being sanitised afterwards. A sample of the pesto was taken for testing and was positive for *S.* Typhimurium PT 9 MLVA profile 03-26-15-09-523.

Tasmania

There was 1 outbreak of suspected foodborne illness reported in Tasmania during this quarter. The aetiological agent was *S.* Typhimurium.

Victoria

There were 17 outbreaks of foodborne or suspected foodborne illness reported in Victoria during this quarter. The aetiological agents were identified as *S.* Typhimurium for 15 outbreaks, and *Campylobacter* spp. and a suspected bacterial enterotoxin for 1 outbreak each.

Description of key outbreak

An investigation was initiated in February after authorities received numerous complaints of gastroenteritis after people had eaten at a gourmet hamburger restaurant. A total of 242 people reported illness, with 143 cases confirmed as *Salmonella* infection (142 confirmed as *S.* Typhimurium PT 9 infection). The investigation identified that up to 20 litres of raw egg mayonnaise had been prepared at the restaurant on the Friday evening and served throughout the weekend on most hamburgers and as a dipping sauce for chips. *S.* Typhimurium PT 9 was detected on a spoon used to serve the mayonnaise in the restaurant. Eggs used to make the mayonnaise were traced back to an egg farm that was implicated in 5 other outbreaks in 2004, 2005 and 2010, and all of these outbreaks were caused by *S.* Typhimurium PT 9. Authorities visited the implicated farm where *S.* Typhimurium PT 135 was detected in one of the initial drag swabs and *S.* Typhimurium PT 9 was found in a drag swab collected on a later date. The predominant MLVA profile for the *S.* Typhimurium PT 9 found in cases in this and the 2010 outbreak; on the mayonnaise serving spoon and in the drag swabs from the farm was 03-24-13-12-525. As a result of this outbreak investigation, complaints of gastroenteritis in the community and active surveillance for notified cases, a further 7 outbreaks involving at least 56 cases (51 of which were confirmed as *S.* Typhimurium PT 9 infection) were found to have been associated with food premises using eggs from the same egg supplier.

Western Australia

There were 3 outbreaks of foodborne or suspected foodborne illness reported in Western Australia during this quarter. The aetiological agents were identified as *S.* Typhimurium in 2 outbreaks and *S.* Singapore for the other.

Cluster investigations

During the quarter, OzFoodNet sites conducted investigations into 15 clusters of infection for which no common food vehicle or source of infection could be identified. Aetiological agents identified during the investigations included 8 *S.* Typhimurium clusters, and 1 cluster each of *S.* Agona, *S.* Oslo, *S.* subspecies, *S.* Virchow, *S.* Wangata, *Listeria monocytogenes* and suspected norovirus.

Comments

The majority of reported outbreaks of gastrointestinal illness in Australia are due to person-to-person transmission, and in this quarter 72% of outbreaks (n=335) were transmitted via this route, which was slightly lower than the same quarter in 2013 (n=385) but higher than the 5-year mean (1st quarter 2009–2013) of 271 outbreaks.

S. Typhimurium was identified as the aetiological agent in 67% (33/49) of foodborne or suspected foodborne outbreaks during the quarter (Table 2). Of the 9 confirmed foodborne outbreaks for which an analytical and/or microbiological link to a food vehicle was established, seven were due to *S.* Typhimurium and five of these were associated with the consumption of raw or minimally cooked egg dishes.

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Appendix: Outbreaks of foodborne or suspected foodborne disease reported by OzFoodNet sites, 1 January to 31 March 2014 (n=49)

State or territory	Month*	Setting prepared	Agent responsible	Number affected	Hospitalised	Evidence	Responsible vehicles
ACT	Jan	Takeaway	*Escherichia coli* (suspected)	3	0	D	Kebabs and tabouli (parsley)
ACT	Feb	Restaurant	*Campylobacter jejuni*	3	1	D	Liver parfait
ACT	Feb	Takeaway	Suspected foodborne enterotoxin (*Bacillus cereus*)	3	0	D	Fried rice
NSW	Jan	Bakery	*Salmonella* Typhimurium MLVA profile 03-17-10-11-523	24	9	M	Vietnamese rolls
NSW	Jan	Cruise/airline	*S.* Typhimurium MLVA profile 03-12-13-09-523	3	1	D	Unknown
NSW	Jan	Restaurant	*S.* Typhimurium MLVA profile 03-24-12-12-523	2	0	D	Raw egg Caesar salad dressing
NSW	Jan	Takeaway	Shiga toxin-producing *Escherichia coli*	6	5	D	Kebabs: multiple ingredients
NSW	Feb	Bakery	*S.* Typhimurium MLVA profile 03-16-09-12-523	26	3	M	Vietnamese rolls with raw egg butter
NSW	Feb	Commercial caterer	*Shigella sonnei* biotype f	7	1	D	Unknown
NSW	Feb	Other	*S.* Typhimurium MLVA 03-10/11-07-12-523	8	2	D	Raw egg mayonnaise
NSW	Feb	Restaurant	Ciguatoxin	5	3	D	Spanish mackerel
NSW	Mar	Bakery	*S.* Typhimurium MLVA profile 03-09-07-12-523	10	7	M	Multiple foods
NSW	Mar	Private residence	Ciguatoxin	9	9	D	Spanish mackerel
NT	Jan	Private residence	*S.* Typhimurium PT 6	3	0	D	Turkey
NT	Mar	Restaurant	Unknown	2	0	D	Chicken Caesar salad
Qld	Jan	Institution	*S.* Typhimurium MLVA profile 03-09-04-12-524	10	3	D	Unknown
Qld	Jan	Private residence	Histamine	2	0	M	Bonito Fish Stew
Qld	Jan	Restaurant	*S.* Typhimurium PT 135a MLVA profile 03-12-12-09-524	10	1	D	Suspected raw egg sauce
Qld	Jan	Restaurant	*S.* Typhimurium MLVA profile 04-15-09-09-490	18	3	D	Unknown
Qld	Feb	Community	*S.* Typhimurium MLVA profile 03-17-09-11-524	12	3	D	Bakery products (various)
Qld	Feb	Private residence	Ciguatoxin	2	0	D	Mackerel
Qld	Feb	Private residence	Ciguatoxin	9	0	D	Spanish mackerel
Qld	Feb	Unknown	Ciguatoxin	18	Unknown	D	Spanish mackerel
SA	Jan	Restaurant	*S.* Typhimurium PT 9	4	0	D	Unknown
SA	Jan	Takeaway	*S.* Typhimurium PT 9	5	2	D	Unknown
SA	Feb	Restaurant	*S.* Typhimurium PT 135	4	2	M	Raw egg aioli
SA	Mar	Fair/festival	*Salmonella* subsp 1 ser 4, 5, 12: i:-	8	0	D	Unknown
SA	Mar	Restaurant	*S.* Typhimurium PT 9	33	5	M	Suspected raw egg contamination of pesto
Tas.	Feb	Private residence	*S.* Typhimurium PT44	3	0	D	Unknown

Appendix (cont'd): Outbreaks of foodborne or suspected foodborne disease reported by OzFoodNet sites, 1 January to 31 March 2014 (n=49)

State or territory	Month*	Setting prepared	Agent responsible	Number affected	Hospitalised	Evidence	Responsible vehicles
Vic.	Jan	Private residence	*S.* Typhimurium PT 9	12	3	D	Unknown
Vic.	Jan	Restaurant	*S.* Typhimurium PT 135	94	17	D	Unknown
Vic.	Feb	Camp	*S.* Typhimurium PT 9	4	0	D	Lightly cooked eggs and/or hollandaise sauce
Vic.	Feb	Commercial caterer	Suspected foodborne enterotoxin (*Clostridium perfringens*)	22	0	A	Suspect dahl
Vic.	Feb	Private residence/ restaurant	*S.* Typhimurium PT 9	2	1	D	Unknown
Vic.	Feb	Restaurant	*S.* Typhimurium PT 135a	2	0	D	Lightly cooked eggs and/or hollandaise sauce
Vic.	Feb	Restaurant	*S.* Typhimurium PT 9	2	1	D	Raw egg aioli
Vic.	Feb	Restaurant	*S.* Typhimurium PT 9	3	1	D	Undercooked eggs
Vic.	Feb	Restaurant	*S.* Typhimurium PT 9	6	0	D	Undercooked eggs
Vic.	Feb	Restaurant	*S.* Typhimurium PT 9	15	26	M	Raw egg mayonnaise
Vic.	Feb	Restaurant	*S.* Typhimurium PT 9	13	1	D	Suspect raw egg aioli
Vic.	Feb	Restaurant	*S.* Typhimurium PT 9	242	5	D	Lightly cooked eggs
Vic.	Mar	Aged care facility	*Campylobacter*	14	0	D	Suspect chicken patties
Vic.	Mar	Private residence	*S.* Typhimurium PT 44	6	3	D	Suspect undercooked eggs in pasta dish
Vic.	Mar	Private residence	*S.* Typhimurium PT 9	3	0	D	Raw egg tiramisu
Vic.	Mar	Restaurant	*S.* Typhimurium PT 9	3	2	D	Raw egg aioli
Vic.	Mar	Restaurant	*S.* Typhimurium PT 9	14	5	M	Undercooked eggs in hollandaise sauce
WA	Jan	Private residence	*S.* Typhimurium PFGE 39	3	0	D	Multiple foods
WA	Feb	Private residence	*S.* Typhimurium PFGE 526	3	0	D	Unknown
WA	Feb	Unknown	*S.* Singapore	6	0	D	Unknown
Total				721	125		

* Month of outbreak is the month of onset of first case or month of notification/investigation of the outbreak.

The number of people affected and hospitalised relate to the findings of the outbreak investigation at the time of writing and not necessarily in the month specified or in this quarter.

A Analytical epidemiological association between illness and 1 or more foods.

D Descriptive evidence implicating the suspected vehicle or suggesting foodborne transmission.

M Microbiological confirmation of aetiological agent in the suspected vehicle and cases.

PT Phage type.

MLVA Multi-locus variable number tandem repeat analysis profile.

PFGE Pulsed-field gel electrophoresis.

National Notifiable Diseases Surveillance System, 1 July to 30 September 2015

A summary of diseases currently being reported by each jurisdiction is provided in Table 1. There were 122,283 notifications to the National Notifiable Diseases Surveillance System (NNDSS) between 1 July to 30 September 2015 (Table 2). The notification rate of diseases per 100,000 population for each state or territory is presented in Table 3.

Table 1: Reporting of notifiable diseases by jurisdiction

Disease	Data received from:
Bloodborne diseases	
Hepatitis (NEC)	All jurisdictions
Hepatitis B (newly acquired)	All jurisdictions
Hepatitis B (unspecified)	All jurisdictions
Hepatitis C (newly acquired)	All jurisdictions except Queensland
Hepatitis C (unspecified)	All jurisdictions
Hepatitis D	All jurisdictions
Gastrointestinal diseases	
Botulism	All jurisdictions
Campylobacteriosis	All jurisdictions except New South Wales
Cryptosporidiosis	All jurisdictions
Haemolytic uraemic syndrome	All jurisdictions
Hepatitis A	All jurisdictions
Hepatitis E	All jurisdictions
Listeriosis	All jurisdictions
STEC, VTEC*	All jurisdictions
Salmonellosis	All jurisdictions
Shigellosis	All jurisdictions
Typhoid fever	All jurisdictions
Quarantinable diseases	
Cholera	All jurisdictions
Highly pathogenic avian influenza (human)	All jurisdictions
Plague	All jurisdictions
Rabies	All jurisdictions
Severe acute respiratory syndrome	All jurisdictions
Smallpox	All jurisdictions
Viral haemorrhagic fever	All jurisdictions
Yellow fever	All jurisdictions
Sexually transmissible infections	
Chlamydia	All jurisdictions
Donovanosis	All jurisdictions
Gonococcal infection	All jurisdictions
Syphilis - congenital	All jurisdictions
Syphilis <2 years duration	All jurisdictions
Syphilis >2 years or unspecified duration	All jurisdictions

Table 1 continued: Reporting of notifiable diseases by jurisdiction

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

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

NEC Not elsewhere classified.

Table 2: Notifications of diseases received by state and territory health authorities, 1 July to 30 September 2015, by date of diagnosis*

Disease	State or territory								Total 3rd quarter 2015	Total 2nd quarter 2015	Total 3rd quarter 2014	Last 5 years mean 3rd quarter	Ratio	Year to date 2015	Last 5 years YTD mean
	ACT	NSW	NT	Qld	SA	Tas.	Vic.	WA							
Bloodborne diseases															
Hepatitis (NEC)	0	0	0	0	0	0	0	0	0	0	0	0.0	0.0	0	0.0
Hepatitis B (newly acquired)†	0	4	1	15	2	0	6	6	34	39	37	47.2	0.7	113	148.2
Hepatitis B (unspecified)‡	21	712	50	314	81	12	325	159	1,674	1,548	1,690	1,751.4	1.0	4,799	5,002.2
Hepatitis C (newly acquired)†	4	6	1	0	6	8	35	57	117	108	106	99.4	1.2	328	323.0
Hepatitis C (unspecified)‡	45	894	52	627	104	63	201	213	2,199	2,500	2,645	2,682.6	0.8	7,227	7,710.0
Hepatitis D	0	1	0	8	3	0	0	0	12	12	14	12.6	1.0	34	36.0
Gastrointestinal diseases															
Botulism	0	0	0	0	0	0	0	0	0	1	0	0.4	0.0	2	1.4
Campylobacteriosis	159	NN	99	1,806	478	242	270	654	3,708	4,087	4,761	4,083.8	0.9	13,229	12,225.0
Cryptosporidiosis	5	88	8	141	48	7	159	21	477	988	419	342.8	1.4	2,945	2,097.4
Haemolytic uraemic syndrome	0	3	0	2	0	0	0	1	6	4	4	3.2	1.9	14	11.8
Hepatitis A	1	11	2	4	1	0	5	4	28	28	51	45.6	0.6	144	154.8
Hepatitis E	0	6	0	0	0	0	1	0	7	4	10	6.2	1.1	21	30.6
Listeriosis	0	3	0	2	1	0	4	4	14	19	18	13.6	1.0	49	57.6
STEC, VTEC§	0	3	0	6	5	0	5	0	19	35	22	32.6	0.6	83	89.0
Salmonellosis	41	581	93	671	220	35	727	306	2,674	4,186	2,743	2,222.6	1.2	13,272	9,670.0
Shigellosis	2	42	20	41	15	2	132	17	271	263	250	140.8	1.9	868	466.8
Typhoid fever	0	6	0	5	2	1	3	2	19	29	22	19.0	1.0	91	95.4
Quarantinable diseases															
Cholera	0	0	0	0	0	0	0	0	0	1	0	1.0	0.0	1	3.4
Highly pathogenic avian influenza (human)	0	0	0	0	0	0	0	0	0	0	0	0.0	0.0	0	0.0
Plague	0	0	0	0	0	0	0	0	0	0	0	0.0	0.0	0	0.0
Rabies	0	0	0	0	0	0	0	0	0	0	0	0.0	0.0	0	0.0
Severe acute respiratory syndrome	0	0	0	0	0	0	0	0	0	0	0	0.0	0.0	0	0.0
Smallpox	0	0	0	0	0	0	0	0	0	0	0	0.0	0.0	0	0.0
Viral haemorrhagic fever	0	0	0	0	0	0	0	0	0	0	0	0.0	0.0	0	0.0
Yellow fever	0	0	0	0	0	0	0	0	0	0	0	0.0	0.0	0	0.5

Table 2 *continued:* Notifications of diseases received by state and territory health authorities, 1 July to 30 September 2015, by date of diagnosis*

Disease	State or territory								Total 3rd quarter 2015	Total 2nd quarter 2015	Total 3rd quarter 2014	Last 5 years mean 3rd quarter	Ratio	Year to date 2015	Last 5 years YTD mean
	ACT	NSW	NT	Qld	SA	Tas.	Vic.	WA							
Sexually transmissible infections															
Chlamydia[||,¶]	318	5,375	683	5,254	1,285	395	0	2,747	16,057	16,363	20,752	20,076.6	0.8	54,591	61,894.8
Donovanosis	0	0	0	0	0	0	0	0	0	0	0	0.4	0.0	0	0.6
Gonococcal infection[¶]	28	1,434	406	691	134	12	82	612	3,399	4,593	3,647	3,183.2	1.1	12,621	10,038.6
Syphilis – congenital	0	0	0	2	0	0	0	0	2	2	4	2.2	0.9	4	3.8
Syphilis <2 years duration[¶]	6	180	64	182	17	4	196	52	701	637	541	399.0	1.8	1,939	1,163.6
Syphilis >2 years or unspecified duration[‡,¶]	1	164	5	61	26	2	219	21	499	490	479	396.0	1.3	1,474	1,157.2
Vaccine preventable diseases															
Diphtheria	0	0	0	1	0	0	0	0	1	0	1	0.4	2.5	2	1.4
Haemophilus influenzae type b	0	2	0	3	0	0	2	1	8	4	6	6.0	1.3	14	14.2
Influenza (laboratory confirmed)	920	25,650	348	22,330	11,725	1,152	12,639	3,650	78,414	9,788	53,023	25,726.8	3.0	92,685	31,708.8
Measles	0	0	0	11	0	0	7	1	19	19	57	58.8	0.3	65	149.4
Mumps	3	12	5	8	4	0	10	150	192	101	47	40.2	4.8	352	132.6
Pertussis	129	3,211	13	293	317	13	1,131	489	5,596	4,219	3,138	6,086.4	0.9	13,839	16,698.0
Pneumococcal disease – invasive	9	205	18	98	44	16	126	71	587	402	588	643.0	0.9	1,176	1,326.8
Poliovirus infection	0	0	0	0	0	0	0	0	0	0	0	0.0	0.0	0	0.0
Rubella	0	1	0	1	0	0	0	2	4	5	3	10.0	0.4	14	30.4
Rubella – congenital	0	0	0	0	0	0	0	0	0	0	0	0.2	0.0	0	0.4
Tetanus	0	0	0	0	0	0	0	0	0	1	0	0.6	0.0	1	2.6
Varicella zoster (chickenpox)	16	NN	29	126	101	19	9	150	450	534	545	596.6	0.8	1,510	1,419.8
Varicella zoster (shingles)	57	NN	83	9	498	56	9	346	1,058	1,564	1,323	1,051.8	1.0	4,207	3,232.2
Varicella zoster (unspecified)	40	NN	3	1,672	62	39	5	343	2,164	3,251	3,035	2,516.8	0.9	8,462	7,185.8
Vectorborne diseases															
Barmah Forest virus infection	0	23	5	44	0	1	1	10	84	194	89	335.4	0.3	540	1,613.4
Chikungunya virus infection	0	7	1	0	0	0	3	2	13	27	17	14.6	0.9	95	50.2
Dengue virus infection	6	67	5	33	12	3	66	64	256	426	282	286.4	0.9	1,425	1,129.2
Flavivirus infection (unspecified)	0	0	0	0	0	0	0	1	1	2	2	3.8	0.3	7	11.8
Japanese encephalitis virus infection	0	0	0	0	0	0	0	0	0	0	1	0.6	0.0	2	1.2
Kunjin virus infection	0	0	0	0	0	0	0	0	0	0	0	0.0	0.0	0	0.8
Malaria	2	11	6	10	2	0	17	11	59	47	84	99.4	0.6	169	289.0
Murray Valley encephalitis virus infection	0	0	0	0	0	0	0	0	0	1	0	0.0	0.0	2	3.2
Ross River virus infection	0	179	67	398	11	0	32	129	816	2,066	879	595.4	1.4	8,543	3,920.0

Table 2 continued: Notifications of diseases received by state and territory health authorities, 1 July to 30 September 2015, by date of diagnosis

Disease	State or territory								Total 3rd quarter 2015	Total 2nd quarter 2015	Total 3rd quarter 2014	Last 5 years mean 3rd quarter	Ratio	Year to date 2015	Last 5 years YTD mean
	ACT	NSW	NT	Qld	SA	Tas.	Vic.	WA							
Zoonoses															
Anthrax	0	0	0	0	0	0	0	0	0	0	0	0.0	0.0	0	0.2
Australian bat lyssavirus infection	0	0	0	0	0	0	0	0	0	0	0	0.0	0.0	0	0.2
Brucellosis	0	1	1	2	0	0	0	0	4	7	5	6.6	0.6	15	18.0
Leptospirosis	0	4	0	11	0	0	1	1	17	18	19	20.2	0.8	60	107.2
Lyssavirus infection (NEC)	0	0	0	0	0	0	0	0	0	0	0	0.0	0.0	0	0.0
Ornithosis	0	0	0	0	0	0	3	0	3	2	10	13.0	0.2	7	38.4
Q fever	0	75	0	59	3	0	12	4	153	141	118	98.0	1.6	450	303.6
Tularaemia	0	0	0	0	0	0	0	0	0	0	0	0.0	0.0	0	0.3
Other bacterial infections															
Legionellosis	0	23	1	20	8	2	14	22	90	103	109	103.0	0.9	285	289.6
Leprosy	0	0	1	1	0	0	1	2	5	3	0	2.4	2.1	9	7.4
Meningococcal infection – invasive**	1	16	0	9	14	1	17	8	66	49	57	67.8	1.0	141	157.2
Tuberculosis	4	111	2	47	22	2	93	25	306	309	353	356.6	0.9	894	964.8
Total	1,818	39,111	2,072	35,018	15,251	2,087	16,568	10,358	122,283	59,220	102,006			248,820	

* The date of diagnosis is the onset date or where the date of onset was not known, the earliest of the specimen collection date, the notification date, or the notification receive date. For hepatitis B (unspecified), hepatitis C (unspecified), leprosy, syphilis (> 2 years or unspecified duration) and tuberculosis, the public health unit notification receive date was used.

† Newly acquired hepatitis includes cases where the infection was determined to be acquired within 24 months prior to diagnosis. Queensland reports hepatitis C newly acquired under hepatitis unspecified.

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

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

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

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

** 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 July to 30 September 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	1.3	0.5	0.0	0.4	0.9	0.6
Hepatitis B (unspecified)§	21.8	37.9	81.8	26.6	19.2	9.3	22.3	24.8	28.5
Hepatitis C (newly acquired)‡	4.2	0.3	1.6	0.0	1.4	6.2	2.4	8.9	2.0
Hepatitis C (unspecified)§	46.7	47.6	85.0	53.1	24.7	49.0	13.8	33.2	37.5
Hepatitis D	0.0	0.1	0.0	0.7	0.7	0.0	0.0	0.0	0.2
Gastrointestinal diseases									
Botulism	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
Campylobacteriosis	165.0	NN	161.9	153.0	113.4	188.1	18.5	102.0	92.9
Cryptosporidiosis	5.2	4.7	13.1	11.9	11.4	5.4	10.9	3.3	8.1
Haemolytic uraemic syndrome	0.0	0.2	0.0	0.2	0.0	0.0	0.0	0.2	0.1
Hepatitis A	1.0	0.6	3.3	0.3	0.2	0.0	0.3	0.6	0.5
Hepatitis E	0.0	0.3	0.0	0.0	0.0	0.0	0.1	0.0	0.1
Listeriosis	0.0	0.2	0.0	0.2	0.2	0.0	0.3	0.6	0.2
STEC,VTEC‖	0.0	0.2	0.0	0.5	1.2	0.0	0.3	0.0	0.3
Salmonellosis	42.5	30.9	152.1	56.8	52.2	27.2	49.8	47.7	45.6
Shigellosis	2.1	2.2	32.7	3.5	3.6	1.6	9.0	2.7	4.6
Typhoid fever	0.0	0.3	0.0	0.4	0.5	0.8	0.2	0.3	0.3
Quarantinable diseases									
Cholera	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
Human pathogenic avian influenza in humans	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¶,**	330.0	286.1	1,116.9	445.1	305.0	307.0	0.0	428.3	273.6
Donovanosis	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
Gonococcal infection**	29.1	76.3	663.9	58.5	31.8	9.3	5.6	95.4	57.9
Syphilis – congenital	0.0	0.0	0.0	0.2	0.0	0.0	0.0	0.0	0.0
Syphilis < 2 years duration**	6.2	9.6	104.7	15.4	4.0	3.1	13.4	8.1	11.9
Syphilis > 2 years or unspecified duration§,**	1.0	8.7	8.2	5.2	6.2	1.6	15.0	3.3	8.5
Vaccine preventable diseases									
Diphtheria	0.0	0.0	0.0	0.1	0.0	0.0	0.0	0.0	0.0
Haemophilus influenzae type b	0.0	0.1	0.0	0.3	0.0	0.0	0.1	0.2	0.1
Influenza (laboratory confirmed)	954.6	1,365.1	569.1	1,891.8	2,782.7	895.3	865.8	569.1	1,336.1
Measles	0.0	0.0	0.0	0.9	0.0	0.0	0.5	0.2	0.3
Mumps	3.1	0.6	8.2	0.7	0.9	0.0	0.7	23.4	3.3
Pertussis	133.9	170.9	21.3	24.8	75.2	10.1	77.5	76.2	95.4
Pneumococcal disease – invasive	9.3	10.9	29.4	8.3	10.4	12.4	8.6	11.1	10.0
Poliovirus infection	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
Rubella	0.0	0.1	0.0	0.1	0.0	0.0	0.0	0.3	0.1
Rubella – congenital	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
Tetanus	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0

Table 3 continued: Notification rates of diseases, 1 July to 30 September 2015, by state or territory. (Annualised rate per 100,000 population)*,†

Disease	State or territory								Aust.
	ACT	NSW	NT	Qld	SA	Tas.	Vic.	WA	
Vaccine preventable diseases, (cont'd)									
Varicella zoster (chickenpox)	16.6	NN	47.4	10.7	24.0	14.8	0.6	23.4	11.3
Varicella zoster (shingles)	59.1	NN	135.7	0.8	118.2	43.5	0.6	53.9	26.5
Varicella zoster (unspecified)	41.5	NN	4.9	141.7	14.7	30.3	0.3	53.5	54.2
Vectorborne diseases									
Barmah Forest virus infection	0.0	1.2	8.2	3.7	0.0	0.8	0.1	1.6	1.4
Chikungunya virus infection	0.0	0.4	1.6	0.0	0.0	0.0	0.2	0.3	0.2
Dengue virus infection	6.2	3.6	8.2	2.8	2.8	2.3	4.5	10.0	4.4
Flavivirus infection (unspecified)	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.2	0.0
Japanese encephalitis virus infection	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
Kunjin virus infection	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
Malaria	2.1	0.6	9.8	0.8	0.5	0.0	1.2	1.7	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	0.0	9.5	109.6	33.7	2.6	0.0	2.2	20.1	13.9
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	1.6	0.2	0.0	0.0	0.0	0.0	0.1
Leptospirosis	0.0	0.2	0.0	0.9	0.0	0.0	0.1	0.2	0.3
Lyssavirus infection (NEC)	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
Ornithosis	0.0	0.0	0.0	0.0	0.0	0.0	0.2	0.0	0.1
Q fever	0.0	4.0	0.0	5.0	0.7	0.0	0.8	0.6	2.6
Tularaemia	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
Other bacterial diseases									
Legionellosis	0.0	1.2	1.6	1.7	1.9	1.6	1.0	3.4	1.5
Leprosy	0.0	0.0	1.6	0.1	0.0	0.0	0.1	0.3	0.1
Meningococcal infection – invasive††	1.0	0.9	0.0	0.8	3.3	0.8	1.2	1.2	1.1
Tuberculosis	4.2	5.9	3.3	4.0	5.2	1.6	6.4	3.9	5.2

* The date of diagnosis is the onset date or where the date of onset was not known, the earliest of the specimen collection date, the notification date, or the notification receive date. For hepatitis B (unspecified), hepatitis C (unspecified), leprosy, syphilis (> 2 years or unspecified duration) and tuberculosis, the public health unit notification receive date was used.

† Rate per 100,000 of population. Annualisation Factor was 4.0

‡ Newly acquired hepatitis includes cases where the infection was determined to be acquired within 24 months prior to diagnosis. Queensland reports hepatitis C newly acquired under hepatitis C unspecified.

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

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

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

** The national case definitions for 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 April 2014 to 31 March 2015, assessed as at 30 June 2015

Brynley P Hull for the National Centre for Immunisation Research and Surveillance of Vaccine Preventable Diseases

Introduction

The National Centre for Immunisation Research and Surveillance of Vaccine Preventable Diseases (NCIRS) provides commentary on the trends in ACIR data. For further information please contact NCIRS at: telephone +61 2 9845 1435, email: brynley.hull@health.nsw.gov.au

Tables 1, 2 and 3 provide the latest quarterly report on childhood immunisation coverage from the Australian Childhood Immunisation Register (ACIR) for all children.

The data show the percentage of all children 'fully immunised' at 12 months, 24 months and 60 months, for four 3-month birth cohorts of children assessed at the stated ages between 1 April 2014 and 31 March 2015 using ACIR data up to 30 June 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 PRP-OMP containing *Haemophilus influenzae* type b (Hib) vaccine or 3 doses of any other *Haemophilus influenzae* type b (Hib) vaccine, 3 doses of hepatitis B vaccine, and 3 doses of 13-valent pneumococcal conjugate vaccine. 'Fully immunised' at 24 months of age is defined as a child having a record on the ACIR of 3 doses of a DTP-containing vaccine, 3 doses of polio vaccine, 3 or 4 doses of PRP-OMP Hib vaccine or 4 doses of any other Hib vaccine, 3 doses of hepatitis B vaccine, 1 dose of a measles, mumps and rubella-containing (MMR) vaccine, 1 dose of meningococcal C vaccine, and 1 dose of varicella vaccine. 'Fully immunised' at 60 months of age is defined as a child having a record on the ACIR of 4 doses of a DTP-containing vaccine, 4 doses of polio vaccine, and 2 doses of an MMR-containing vaccine.

A full description of the basic methodology used can be found in *Communicable Diseases Intelligence* 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 0.3 of a percentage point to 91.3% (Table 1). Most jurisdictions experienced small increases in the percentage of children 'fully immunised' by 12 months of age. For individual vaccines due by 12 months of age all jurisdictions achieved coverage greater than 90%.

The rolling annualised percentage of all children 'fully immunised' by 24 months of age for Australia decreased again for the 2nd consecutive report by 0.9 percentage points to 89.2% (Table 2). This decrease is due to the immunisation coverage assessment algorithm for the 24-month milestone

Table 1. Percentage of children immunised at 12 months of age for the birth cohort 1 April 2013 to 31 March 2014, preliminary results, by disease and state or territory; assessment date 30 June 2015

Vaccine	State or territory								Aust
	ACT	NSW	NT	Qld	SA	Tas.	Vic.	WA	
Total number of children	5,657	98,744	3,795	62,947	20,104	5,971	76,090	34,034	307,342
Diphtheria, tetanus, pertussis (%)	94.1	91.9	91.0	92.4	91.8	91.3	92.1	92.4	92.1
Poliomyelitis (%)	94.0	91.9	91.0	92.4	91.8	91.3	92.1	92.3	92.1
Haemophilus influenzae type b (%)	93.7	91.8	90.9	92.3	91.7	91.2	91.9	92.1	92.0
Hepatitis B (%)	93.5	91.6	91.0	92.2	91.6	91.1	91.7	91.9	91.8
Pneumococcal	93.8	91.6	91.1	92.2	91.5	91.2	91.8	91.9	91.8
Fully immunised (%)	92.9	91.1	90.5	91.9	91.2	90.7	91.2	91.4	91.3

being amended in July 2014 to include a dose of meningococcal vaccine, a dose of varicella vaccine and a 2nd dose of MMR in the assessment of 'fully immunised'. All jurisdictions experienced similar decreases in fully immunised coverage for this age group. Coverage for individual vaccines due by 24 months remained high in all jurisdictions, except that coverage in all jurisdictions again decreased for the measles, mumps and rubella vaccine (by 0.7 to 1.2 percentage points, and 0.9 nationally). This decrease is due to the immunisation coverage assessment algorithm for the MMR vaccine being amended in July 2014 to look for a 2nd dose of MMR vaccine, rather than a 1st dose.

The rolling annualised percentage of all children 'fully immunised' by 60 months of age for Australia increased marginally from the previous report by 0.1 of a percentage point to 92.3% (Table 3). This maintains the improvement in coverage for this age milestone. There were also only marginal changes in fully immunised coverage at 60 months of age in all jurisdictions. Coverage for individual vaccines due by 60 months remained greater than 90% in all jurisdictions.

Figure: Trends in vaccination coverage, Australia, 1997 to 31 March 2015, by age cohorts

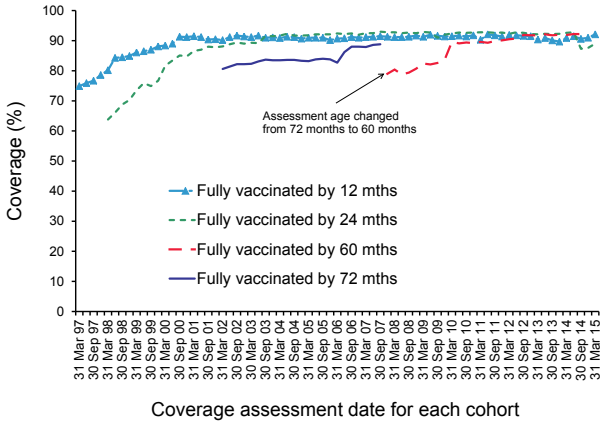


Table 2. Percentage of children immunised at 24 months of age for the birth cohort 1 April 2012 to 31 March 2013, preliminary results, by disease and state or territory; assessment date 30 June 2015

	State or territory								
Vaccine	ACT	NSW	NT	Qld	SA	Tas.	Vic.	WA	Aust
Total number of children	5,531	101,192	3,600	63,189	20,218	5,889	76,870	34,281	310,770
Diphtheria, tetanus, pertussis (%)	96.1	95.1	95.2	95.0	94.8	95.1	95.6	94.8	95.2
Poliomyelitis (%)	96.1	95.0	95.3	95.0	94.7	95.0	95.6	94.7	95.1
Haemophilus influenzae type b (%)	95.0	93.7	94.9	94.2	93.4	93.5	94.4	93.5	94.0
Measles, mumps, rubella (%)	93.1	91.0	92.1	91.9	90.4	90.2	91.5	89.8	91.2
Hepatitis B (%)	95.7	94.6	95.3	94.6	94.4	94.8	95.2	94.2	94.7
Meningococcal C (%)	94.7	93.5	94.4	94.1	92.6	93.7	93.9	92.7	93.6
Varicella (%)	94.4	91.2	90.4	91.5	90.3	89.1	91.9	90.1	91.3
Fully immunised (%)	91.4	88.8	89.3	90.4	87.9	87.5	89.6	87.7	89.2

Table 3. Percentage of children immunised at 60 months of age for the birth cohort 1 April 2009 to 31 March 2010, preliminary results, by disease and state or territory; assessment date 30 June 2015

	State or territory								
Vaccine	ACT	NSW	NT	Qld	SA	Tas.	Vic.	WA	Aust
Total number of children	5,559	101,107	3,517	65,254	20,290	6,385	76,035	34,222	312,369
Diphtheria, tetanus, pertussis (%)	93.8	93.2	93.1	92.8	91.5	93.4	93.2	91.2	92.8
Poliomyelitis (%)	93.8	93.2	93.1	92.8	91.5	93.3	93.2	91.2	92.8
Measles, mumps, rubella (%)	93.5	93.2	93.5	92.8	91.4	93.2	93.1	91.1	92.8
Fully immunised (%)	93.2	92.7	92.4	92.3	90.9	92.6	92.6	90.6	92.2

Australian Gonococcal Surveillance Programme, 1 April to 30 June 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 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.

Results

A summary of the proportion of isolates with decreased susceptibility to ceftriaxone, and the proportion resistant to penicillin, ciprofloxacin and azithromycin 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 ciprofloxacin, azithromycin and penicillin, Australia, 1 April to 30 June 2015, by state or territory

State or territory	Number of isolates tested quarter 2 2015	Decreased susceptibility		Resistance					
		Ceftriaxone		Ciprofloxacin		Azithromycin		Penicillin	
		n	%	n	%	n	%	n	%
Australian Capital Territory	23	0	0.0	1	4.3	0	0.0	11	47.8
New South Wales	439	6	1.4	133	30.3	7	1.6	112	25.5
Queensland	159	0	0.0	47	29.6	11	6.9	42	26.4
South Australia	54	0	0.0	19	35.2	0	0.0	7	13.0
Tasmania	8	0	0.0	0	0.0	0	0.0	1	12.5
Victoria	453	7	1.5	53	11.7	13	2.9	74	16.3
Northern Territory/Urban & Rural	46	0	0.0	0	0.0	0	0.0	0	0.0
Northern Territory/Remote	18	0	0.0	3	16.7	0	0.0	3	16.7
Western Australia/Urban & Rural	94	1	1.1	22	23.4	4	4.3	16	17.0
Western Australia/Remote	24	0	0.0	1	4.2	0	0.0	0	0.0
Australia	1,318	14	1.1	279	21.2	35	2.7	266	20.2

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 1st quarter of 2015 the only states that reported isolates with decreased susceptibility to ceftriaxone were New South Wales, Victoria and urban Western Australia. All reported a decrease in the proportion of *N. gonorrhoeae* isolates with decreased susceptibility to ceftriaxone when compared with the same quarter in 2014; and the annual data for 2014.[4]

From New South Wales there were 6 of 439 strains with decreased susceptibility to ceftriaxone. Of those, 2 (33%) were multi-drug resistant (MDR); all (100%) were from males; and 3 (50%) were isolated from extragenital sites (rectal and pharyngeal). From Victoria there were 7 of 453 strains with decreased susceptibility to ceftriaxone and of those, all (100%) were MDR; all (100%) were from males; and 4 (57%) were isolated from extragenital sites (rectal and pharyngeal). From urban Western Australia one of the 94 strains tested had decreased susceptibility to ceftriaxone. This strain was from a male, was MDR and not from an extragenital site (rectal or pharyngeal).

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 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 MDR gonococcal strain (A8806) with a ceftriaxone MIC of 0.5 mg/L, the highest ever reported in Australia, which 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 0.06–0.125 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 extragenital 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, and 1 April to 30 June 2015, by state or territory

Ceftriaxone MIC mg/L	2010	2011	2012	2013	2014	2015 Q1	2015 Q2
0.06	4.6	3.2	4.1	8.2	4.8	1.6	1.1
0.125	0.1	0.1	0.3	0.6	0.6	0.1	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 annual report, 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 Mar;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. Australian Sexual Health Alliance. *The Australian Sexually Transmitted Infection Management Guidelines 2014*. Available from: http://www.sti.guidelines.org.au

Australian Meningococcal Surveillance Programme, 1 July to 30 September 2015

Monica M Lahra, Rodney P Enriquez for the Australian Meningococcal Surveillance Programme

Introduction

The reference laboratories of the Australian Meningococcal Surveillance Programme (AMSP) report data on the number of cases confirmed by laboratory testing using culture and by non-culture based techniques. Culture positive cases, where *Neisseria meningitidis* is grown from a normally sterile site or skin lesions, and non-culture based diagnoses, derived from results of nucleic acid amplification assays and serological techniques, are defined as invasive meningococcal disease (IMD) according to Public Health Laboratory Network definitions. Data contained in quarterly reports are restricted to a description of the number of cases by jurisdiction and serogroup, where known. Some minor corrections to data in the Table may be made in subsequent reports if additional data are received. A full analysis of laboratory confirmed cases of IMD in each calendar year is contained in the AMSP annual reports published in *Communicable Diseases Intelligence*. For more information see *Commun Dis Intell* 2015;39(1):E179.

Results

Laboratory confirmed cases of invasive meningococcal disease for the period 1 July to 30 September 2015 are shown in the Table. In this quarter there has been a notable increase in IMD cases attributed to MenW and MenY, particularly in Victoria. The National Neisseria Network, which coordinates the AMSP is conducting investigations including whole genome sequencing and continuing to monitor and evaluate the situation.

Table: Number of laboratory confirmed cases of invasive meningococcal disease, Australia, 1 July to 30 September 2015, by serogroup and state or territory

State or territory	Year	Serogroup													
		A		B		C		Y		W135		ND		All	
		Q3	YTD	Q3	YTD	Q3	YTD	Q3	YTD	Q3	YTD	Q3	YTD	Q3	YTD
Australian Capital Territory	2015	0	0	0	1	0	0	1	1	0	0	0	0	1	2
	2014	0	0	0	1	0	0	1	1	0	0	0	0	1	2
New South Wales	2015	0	0	4	16	0	1	5	7	4	5	0	0	13	29
	2014	0	0	5	15	0	0	0	6	0	3	0	0	5	24
Northern Territory	2015	0	0	0	1	0	0	0	0	0	0	0	0	0	1
	2014	0	0	1	3	0	0	0	0	0	0	0	0	1	3
Queensland	2015	0	0	6	19	0	0	1	2	3	4	0	0	10	25
	2014	0	0	11	24	1	1	1	1	2	3	0	2	15	31
South Australia	2015	0	0	12	18	0	0	0	0	0	0	0	0	12	18
	2014	0	0	12	21	0	0	0	0	0	0	0	0	12	21
Tasmania	2015	0	0	0	0	0	0	0	0	0	0	0	0	0	0
	2014	0	0	1	1	0	0	0	0	1	1	0	0	2	2
Victoria	2015	0	0	8	23	0	0	4	6	6	12	0	1	18	42
	2014	0	0	12	20	0	0	1	2	0	2	0	0	13	24
Western Australia	2015	0	0	3	7	0	0	1	2	3	3	0	0	7	12
	2014	0	0	3	10	0	2	1	1	0	1	0	0	4	14
Total	2015	0	0	33	85	0	1	12	18	16	24	0	1	61	129
	2014	0	0	45	95	1	3	4	11	3	10	0	2	53	121

Australian Sentinel Practices Research Network, 1 July to 30 September 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.

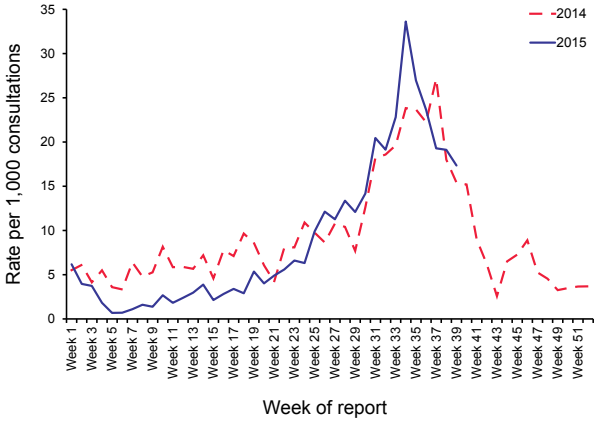
The list of conditions reported is reviewed annually by the ASPREN management committee. In 2015, 4 conditions are being monitored. They include ILI, gastroenteritis and varicella infections (chickenpox and shingles). Definitions of these conditions are described in Surveillance systems reported in CDI, 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 260 general practitioners regularly contributed data to ASPREN in the 3rd quarter of 2015. Each week an average of 230 general practitioners provided information to ASPREN at an average of 18,322 (range 16,122–19,971) consultations per week and an average of 361 (range 260–538) notifications per week.

ILI rates reported from 1 July to 30 September 2015 averaged 19.5 cases per 1,000 consultations (range 11.3–33.6 cases per 1,000 consultations). This was higher than in the same reporting period in 2014, which averaged 17.56 cases per 1,000 consultations (range 7.7–27.2 cases per 1,000 consultations, Figure 1). ILI rates peaked in week 34 at a rate of 33.6 ILI cases per 1,000 consultations.

Figure 1: Weighted consultation rates for influenza-like illness, ASPREN, 2014 and 1 January to 30 September 2015, by year and week of report

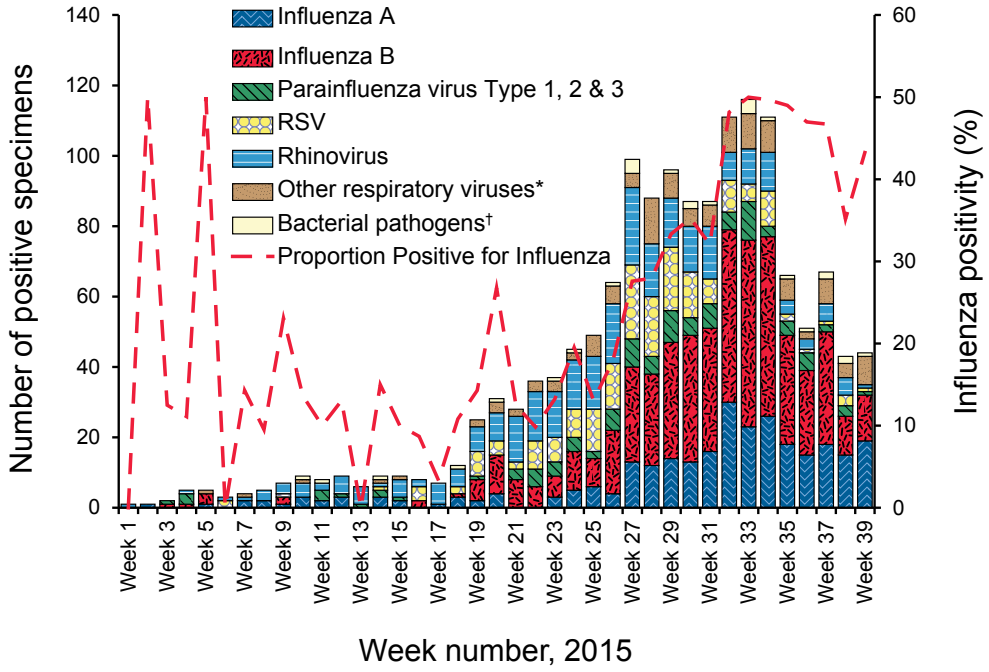


* Results are weighted to account for population size differences between jurisdictions, using population estimates from the 2011 Australian Census.

The ASPREN ILI swab testing program continued in 2015 with 1,629 tests being undertaken from 1 July to 30 September. The most commonly reported virus during this reporting period was influenza B (25.8% of all swabs performed, Figure 2), with the second most common virus being influenza A (14.2% of all swabs performed).

From the beginning of 2015 to the end of week 39 there were 785 cases of influenza detected with 505 of these typed as influenza B (19.8% of all swabs performed) and the remaining 280 being influenza A (11%) (Figure 2). Influenza positivity for 2015 was higher at 30.8% compared with 27% for the same period last year, with lower influenza A positivity and higher influenza B positivity being seen in 2015 compared with 2014 (23.4% and 3.6% of all swabs performed in 2014, respectively).

Figure 2: Swab testing results, ASPREN, 1 January to 30 September 2015, proportion positivity by virus, by week of report



* Includes human metapneumovirus and adenovirus.

† Includes *Bordetella pertussis* and *Mycoplasma pneumoniae*.

During this reporting period, consultation rates for gastroenteritis averaged 4.03 cases per 1,000 consultations (range 2.5–6.4 cases per 1,000, Figure 3). This was similar to the rates in the same reporting period in 2014 where the average was 4.02 cases per 1,000 consultations (range 2.2–8.3 cases per 1,000).

Varicella infections were reported at a similar rate for the 3rd quarter of 2015 compared with the same period in 2014. From 1 July to 30 September 2015, recorded rates for chickenpox averaged 0.21 cases per 1,000 consultations (range 0.08–0.59 cases per 1,000 consultations, Figure 4).

Figure 3: Consultation rates for gastroenteritis, ASPREN, 2014 and 1 January to 30 September 2015, by year and week of report

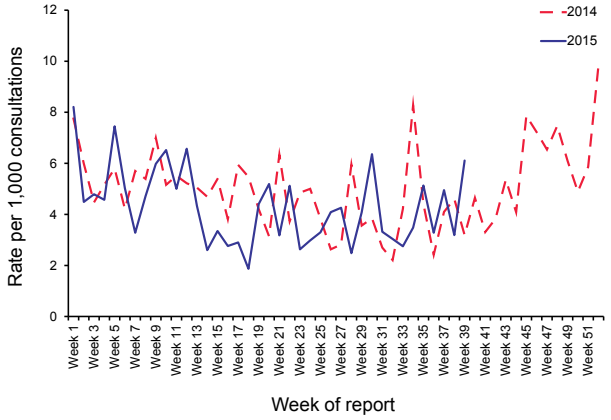
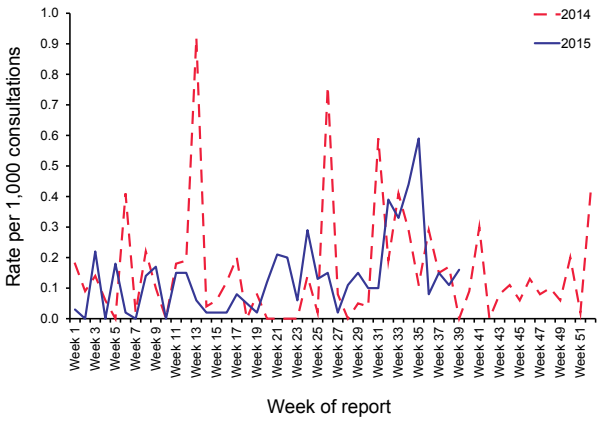
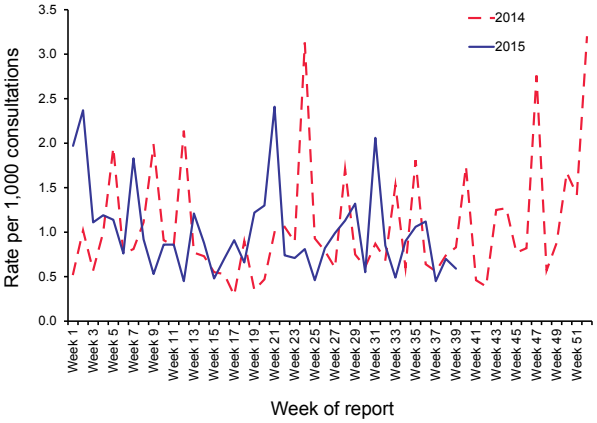


Figure 4: Consultation rates for chickenpox, ASPREN, 2014 and 1 January to 30 September 2015, by year and week of report



In the 3rd quarter of 2015, reported rates for shingles averaged 0.94 cases per 1,000 consultations (range 0.45–2.06 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 0.92 cases per 1,000 consultations (range 0.60–1.81 cases per 1,000 consultations).

Figure 5: Consultation rates for shingles, ASPREN, 2014 and 1 January to 30 September 2015, by year and week of report



HIV surveillance, 1 January to 31 March 2014

The Kirby Institute

Introduction

National surveillance for HIV infection is coordinated by the Kirby Institute, in collaboration with state and territory health authorities and the Australian Government Department of Health. Cases of HIV infection are notified to the National HIV Registry on the first occasion of diagnosis in Australia, by either the diagnosing laboratory (Australian Capital Territory, New South Wales, Tasmania, Victoria) or by a combination of laboratory and doctor sources (Northern Territory, Queensland, South Australia, Western Australia). Diagnoses of HIV infection are notified with the person's date of birth and name code, to minimise duplicate notifications while maintaining confidentiality.

Tabulations of newly diagnosed HIV infections are based on data available 3 months after the end of the reporting interval indicated, to allow for reporting delay and to incorporate newly available information. More detailed information on diagnoses of HIV infection is published in the quarterly Australian HIV Surveillance Report, and annually in the *HIV, Viral Hepatitis and Sexually Transmissible Infections in Australia, Annual Surveillance Report*. The reports are available from the Kirby Institute, University of New South Wales, SYDNEY NSW 2052. Internet: www.kirby.unsw.edu.au Telephone: +61 2 9385 0900. Facsimile: +61 2 9385 0920. For more information see *Commun Dis Intell* 2015;39(1):E181.

Results

Newly diagnosed cases of HIV infection reported for 1 January to 31 March 2014, are shown in Tables 1 and 2.

Table 1: Number of new diagnoses of HIV infection, 1 January to 31 March 2014, by sex and state or territory of diagnosis

Sex	ACT	NSW	NT	Qld	SA	Tas	Vic	WA	Total 1st qrt 2014	Total 1st qrt 2013	YTD 2014	YTD 2013
Male	9	99	3	56	11	3	75	21	277	211	277	211
Female	0	2	0	6	1	2	4	5	20	20	20	20
Total	9	102	3	62	12	5	79	26	298	231	298	231

* Totals include people whose sex was reported as transgender.

Table 2: Cumulative number of new diagnoses of HIV infection since the introduction of HIV antibody testing, 1985 to 31 March 2014, by sex and state or territory

Sex	ACT	NSW	NT	Qld	SA	Tas	Vic	WA	Aust
Male	358	16,579	199	3,965	1,231	175	7,173	1,694	31,374
Female	44	1,098	36	411	152	27	534	329	2,631
Not reported	0	226	0	0	0	0	22	0	248
Total*	402	17,946	235	4,385	1,384	202	7,755	2,030	34,339

* Totals include people whose sex was reported as transgender.

HIV surveillance, 1 April to 30 June 2014

The Kirby Institute

Introduction

National surveillance for HIV infection is coordinated by the Kirby Institute, in collaboration with state and territory health authorities and the Australian Government Department of Health. Cases of HIV infection are notified to the National HIV Registry on the first occasion of diagnosis in Australia, by either the diagnosing laboratory (Australian Capital Territory, New South Wales, Tasmania, Victoria) or by a combination of laboratory and doctor sources (Northern Territory, Queensland, South Australia, Western Australia). Diagnoses of HIV infection are notified with the person's date of birth and name code, to minimise duplicate notifications while maintaining confidentiality.

Tabulations of newly diagnosed HIV infections are based on data available 3 months after the end of the reporting interval indicated, to allow for reporting delay and to incorporate newly available information. More detailed information on diagnoses of HIV infection is published in the quarterly Australian HIV Surveillance Report, and annually in the *HIV, Viral Hepatitis and Sexually Transmissible Infections in Australia, Annual Surveillance Report*. The reports are available from the Kirby Institute, University of New South Wales, SYDNEY NSW 2052. Internet: www.kirby.unsw.edu.au Telephone: +61 2 9385 0900. Facsimile: +61 2 9385 0920. For more information see *Commun Dis Intell* 2015;39(1):E181.

Results

Newly diagnosed cases of HIV infection reported for 1 April to 30 June 2014, are shown in Tables 1 and 2.

Table 1: Number of new diagnoses of HIV infection, 1 April to 30 June 2014, by sex and state or territory of diagnosis

Sex	ACT	NSW	NT	Qld	SA	Tas	Vic	WA	Total 2nd qrt 2014	Total 2nd qrt 2013	YTD 2014	YTD 2013
Male	3	69	3	53	8	4	63	22	225	234	502	445
Female	0	9	0	9	2	0	7	5	32	30	52	50
Total*	3	78	3	62	10	4	70	27	257	264	555	495

* Totals include people whose sex was reported as transgender.

Table 2: Cumulative number of new diagnoses of HIV infection since the introduction of HIV antibody testing, 1985 to 30 June 2014, by sex and state or territory

Sex	ACT	NSW	NT	Qld	SA	Tas	Vic	WA	Aust
Male	361	16,648	202	4,018	1,239	179	7,236	1,716	31,599
Female	44	1,107	36	420	154	27	541	334	2,663
Not reported	0	226	0	0	0	0	22	0	248
Total*	405	18,024	238	4,447	1,394	206	7,825	2,057	34,596

* Totals include people whose sex was reported as transgender.

HIV surveillance, 1 July to 30 September 2014

The Kirby Institute

Introduction

National surveillance for HIV infection is coordinated by the Kirby Institute, in collaboration with state and territory health authorities and the Australian Government Department of Health. Cases of HIV infection are notified to the National HIV Registry on the first occasion of diagnosis in Australia, by either the diagnosing laboratory (Australian Capital Territory, New South Wales, Tasmania, Victoria) or by a combination of laboratory and doctor sources (Northern Territory, Queensland, South Australia, Western Australia). Diagnoses of HIV infection are notified with the person's date of birth and name code, to minimise duplicate notifications while maintaining confidentiality.

Tabulations of newly diagnosed HIV infections are based on data available 3 months after the end of the reporting interval indicated, to allow for reporting delay and to incorporate newly available information. More detailed information on diagnoses of HIV infection is published in the quarterly Australian HIV Surveillance Report, and annually in the *HIV, Viral Hepatitis and Sexually Transmissible Infections in Australia, Annual Surveillance Report*. The reports are available from the Kirby Institute, University of New South Wales, SYDNEY NSW 2052. Internet: www.kirby.unsw.edu.au Telephone: +61 2 9385 0900. Facsimile: +61 2 9385 0920. For more information see *Commun Dis Intell* 2015;39(1):E181.

Results

Newly diagnosed cases of HIV infection reported for 1 July to 30 September 2014, are shown in Tables 1 and 2.

Table 1: Number of new diagnoses of HIV infection, 1 July to 30 September 2014, by sex and state or territory of diagnosis

Sex	ACT	NSW	NT	Qld	SA	Tas	Vic	WA	Total 3rd qrt 2014	Total 3rd qrt 2013	YTD 2014	YTD 2013
Male	1	75	1	54	5	3	80	26	245	256	747	701
Female	2	6	2	6	2	0	3	5	26	22	78	72
Total*	3	81	3	60	7	3	83	32	272	279	827	774

* Totals include people whose sex was reported as transgender.

Table 2: Cumulative number of new diagnoses of HIV infection since the introduction of HIV antibody testing, 1985 to 30 September 2014, by sex and state or territory

Sex	ACT	NSW	NT	Qld	SA	Tas	Vic	WA	Aust
Male	362	16,723	203	4,072	1,244	182	7,316	1,742	31,844
Female	46	1,113	38	426	156	27	544	339	2,689
Not reported	0	226	0	0	0	0	22	0	248
Total*	408	18,105	241	4,507	1,401	209	7,908	2,089	34,868

* Totals include people whose sex was reported as transgender.

HIV surveillance, 1 October to 31 December 2014

The Kirby Institute

Introduction

National surveillance for HIV infection is coordinated by the Kirby Institute, in collaboration with state and territory health authorities and the Australian Government Department of Health. Cases of HIV infection are notified to the National HIV Registry on the first occasion of diagnosis in Australia, by either the diagnosing laboratory (Australian Capital Territory, New South Wales, Tasmania, Victoria) or by a combination of laboratory and doctor sources (Northern Territory, Queensland, South Australia, Western Australia). Diagnoses of HIV infection are notified with the person's date of birth and name code, to minimise duplicate notifications while maintaining confidentiality.

Tabulations of newly diagnosed HIV infections are based on data available 3 months after the end of the reporting interval indicated, to allow for reporting delay and to incorporate newly available information. More detailed information on diagnoses of HIV infection is published in the quarterly Australian HIV Surveillance Report, and annually in the *HIV, Viral Hepatitis and Sexually Transmissible Infections in Australia, Annual Surveillance Report*. The reports are available from the Kirby Institute, University of New South Wales, SYDNEY NSW 2052. Internet: www.kirby.unsw.edu.au Telephone: +61 2 9385 0900. Facsimile: +61 2 9385 0920. For more information see *Commun Dis Intell* 2015;39(1):E181.

Results

Newly diagnosed cases of HIV infection reported for 1 October to 31 December 2014, are shown in Tables 1 and 2.

Table 1: Number of new diagnoses of HIV infection, 1 October to 31 December 2014, by sex and state or territory of diagnosis

Sex	ACT	NSW	NT	Qld	SA	Tas	Vic	WA	Total 4th qrt 2014	Total 4th qrt 2013	YTD 2014	YTD 2013
Male	3	76	0	60	5	3	64	17	228	219	975	920
Female	0	8	0	2	5	0	6	5	26	33	104	105
Total*	3	84	0	62	10	3	70	22	254	254	1,081	1,028

* Totals include people whose sex was reported as transgender.

Table 2: Cumulative number of new diagnoses of HIV infection since the introduction of HIV antibody testing, 1985 to 31 December 2014, by sex and state or territory

Sex	ACT	NSW	NT	Qld	SA	Tas	Vic	WA	Aust
Male	365	16,799	203	4,132	1,249	185	7,380	1,759	32,072
Female	46	1,121	38	428	161	27	550	344	2,715
Not reported	0	226	0	0	0	0	22	0	248
Total*	411	18,189	241	4,569	1,411	212	7,978	2,111	35,122

* Totals include people whose sex was reported as transgender.

Invasive pneumococcal disease surveillance Australia, 1 July to 30 September 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 3rd quarter of 2015 was more than the previous quarter and similar to the number of notified cases in the 3rd 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.

Results

In the 3rd quarter of 2015, there were 586 cases of IPD reported to the NNDSS. This was a small reduction on the number of cases reported for the same period in 2014 (n=588) (Table 1). Serotype 3 was the most commonly reported cause of IPD in this quarter (Table 2).

In non-Indigenous Australians, the number of notified cases was highest in the 65–69 years age group followed by the under 5 years age group. In Indigenous Australians, notified cases were highest in the 50–54 years and 35–39 years age groups followed by the 40–49 years age group (Table 3). The proportion of cases reported as Indigenous was similar to the 3rd quarter of 2014 (http://www.health.gov.au/internet/main/publishing.nsf/Content/cda-cdi3901n.htm).

There were 58 cases of IPD reported in children under 5 years of age. Of those cases with known serotype, 53% (n=21) were due to a serotype included in either the 7vPCV or the 13vPCV (Figure 1) compared with 39% (n=22) of cases in the 3rd quarter of 2014. Serotype 19A, which is included in the 13vPCV, continued to be the most common serotype affecting this age group (Table 2). The number of cases in this age group was 11% less than the 3rd quarter of 2014 (n=65).

In the 3rd quarter of 2015, there were 16 cases reported in fully vaccinated children aged less than 5 years who were considered to be 13vPCV failures. Serotype 19A was reported as the cause of disease in 56% (n=9) of these cases (Table 4).

There were 19 cases of IPD reported in Indigenous Australians aged 50 years and over. Of those cases with a reported serotype, 59% (n=10) were due to a serotype included in the 23-valent polysaccharide pneumococcal vaccine (23vPPV) (Figure 2). The number of notified cases of IPD in this age group was 9.5% fewer than the previous quarter (n=21) and identical to that recorded in the same quarter of 2014 (n=19). Compared with the previous quarter, the proportion of 23vPPV serotypes increased slightly from 57% to 59% of cases with a reported serotype.

Table 1: Notified cases of invasive pneumococcal disease, Australia, 1 July to 30 September 2015, by Indigenous status, serotype completeness and state or territory

Indigenous status	ACT	NSW	NT	Qld	SA	Tas.	Vic.	WA	Q3 2015	Q2 2015	Q3 2014	YTD 2015
Indigenous	0	14	14	14	5	0	0	16	63	60	62	–
Non-Indigenous	9	166	4	82	39	16	78	55	449	297	470	–
Not stated/ Unknown	0	25	0	2	0	0	47	0	74	42	56	–
Total	9	205	18	98	44	16	125	71	586	399	588	1,174
Indigenous status completeness* (%)	100	88	100	98	100	100	62	100	87			–
Serotype completeness† (%)	100	86	94	94	68	88	95	97	90			–

* 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 July to 30 September 2015, by age group

Serotype	Age group			Serotype total*
	Under 5 years	5–64 years	Over 65 years	
3	6	31	24	61
22F	2	25	21	48
19A	9	17	15	41
19F	5	14	17	36
7F		27	4	31
9N		21	7	28
23B	3	12	10	25
15A	1	13	6	20
8		16	3	19
23A		5	13	18
6C	1	6	11	18
38	3	3	11	17
11A	1	10	5	16
33F	1	10	4	15
10A	1	5	8	14
35B	1	5	8	14
15B	1	3	7	11
15C	3	1	4	8
16F		3	5	8
17F		5	3	8
31		3	5	8
4		5	3	8
12F		5		5
14	1	3	1	5
Other	1	28	15	44
Serotype unknown	18	27	15	60
Total	58	303	225	586

* 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.

There were 211 cases of IPD reported in non-Indigenous Australians aged 65 years or over. Of those cases with a reported serotype, 61% (n=120) were due to a serotype included in the 23vPPV (Figure 3). The number of notified cases of IPD in this age group was 8% higher than in the 3rd quarter of 2014 (n=195) and 75% higher than the previous quarter (n=122). Compared with the previous quarter, the proportion of IPD due to 23vPPV serotypes increased from 55% to 61% of cases with a reported serotype.

In this quarter there were 30 deaths attributed to 15 different IPD serotypes. There was 1 death reported in a child aged under 5 years, which was associated with serotype 35B.

Notes

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

In Australia, pneumococcal vaccination is recommended as part of routine immunisation for children, the medically at risk, and older Australians. More information on the scheduling of the pneumococcal vaccination can be found on the Immunise Australia Program web site (www.immunise.health.gov.au).

Table 3: Notified cases of invasive pneumococcal disease, Australia, 1 July to 30 September 2015, by Indigenous status and age group

Age group	Indigenous status			Total
	Indigenous	Non-Indigenous	Not reported	
0–4	5	50	3	58
5–9	4	11	7	22
10–14	2	4	1	7
15–19	2	5	2	9
20–24	4	4	2	10
25–29	4	5	2	11
30–34	3	9	8	20
35–39	8	10	6	24
40–44	6	17	16	39
45–49	6	16	10	32
50–54	8	26	4	38
55–59	3	47	2	52
60–64	4	33	2	39
65–69	2	51	1	54
70–74	0	34	3	37
75–79	0	44	4	48
80–84	1	35	2	38
85+	1	47	0	48
Total	63 (11%)	449 (76%)	74 (13%)	586

Figure 1: Notifications (2004 to 30 September 2015) and annual rates (2004 to 2014) of invasive pneumococcal disease in children aged less than 5 years, Australia, by vaccine serotype group

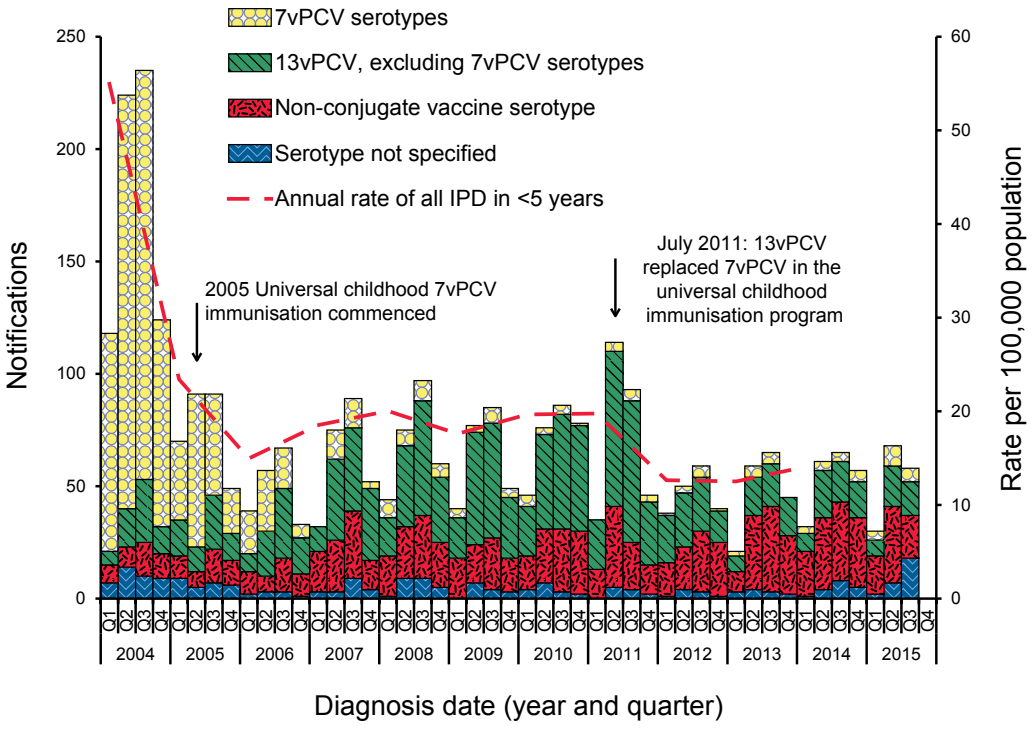
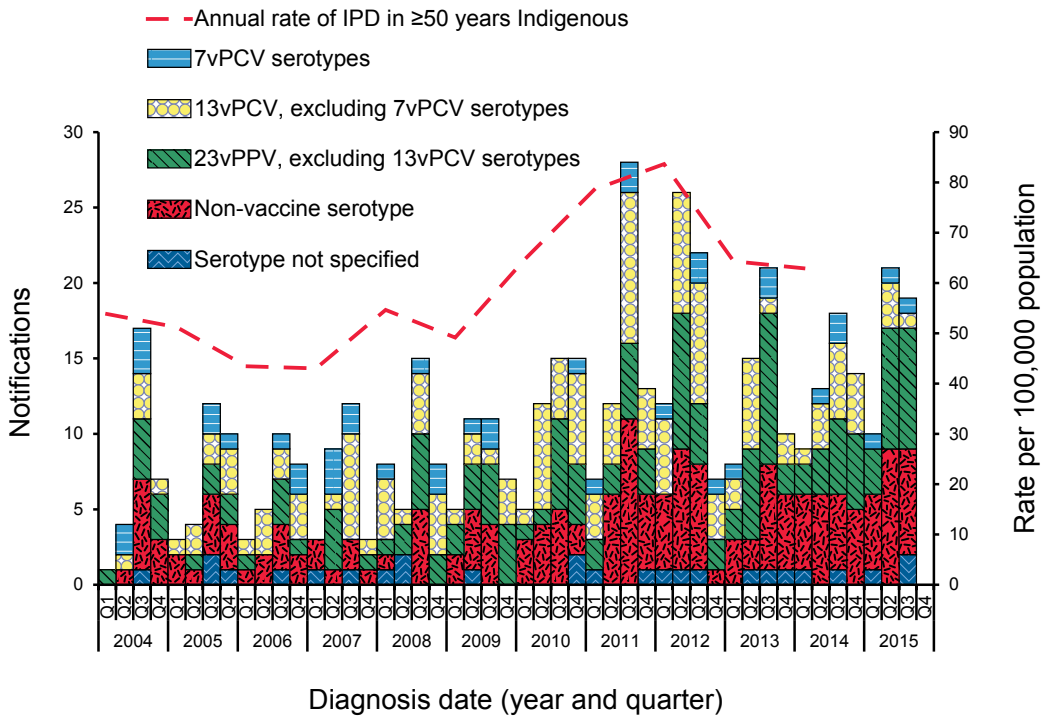


Table 4: Characteristics of 13vPCV failures in children aged less than 5 years, Australia, 1 July to 30 September 2015

Age	Indigenous status	Serotype	Clinical category	Risk factor/s
1 year	Non-Indigenous	19F	Other sterile site	Other/childcare attendee
1 year	Non-Indigenous	19F	Pneumonia	No data available
1 year	Non-Indigenous	19A	Pneumonia	No data available
11 months	Non-Indigenous	19A	Septic arthritis	Childcare attendee
1 year	Non-Indigenous	19A	Bacteraemia	Childcare attendee
2 years	Non-Indigenous	19A	Other sterile site	Childcare attendee
1 year	Non-Indigenous	19A	Pneumonia	No risk factor identified
1 year	Indigenous	19A	Bacteraemia	Other/childcare attendee
3 years	Unknown	19A	No data available	No data available
2 years	Non-Indigenous	19A	No data available	No data available
2 years	Non-Indigenous	19A	No data available	No data available
2 years	Non-Indigenous	3	Other sterile site	Childcare attendee
1 year	Non-Indigenous	3	Pneumonia and bacteraemia	No risk factor identified
9 months	Non-Indigenous	3	Pneumonia	No risk factor identified
1 year	Non-Indigenous	3	Pneumonia	Premature (<37 weeks gestation)
3 years	Non-Indigenous	3	Pneumonia	Chronic illness

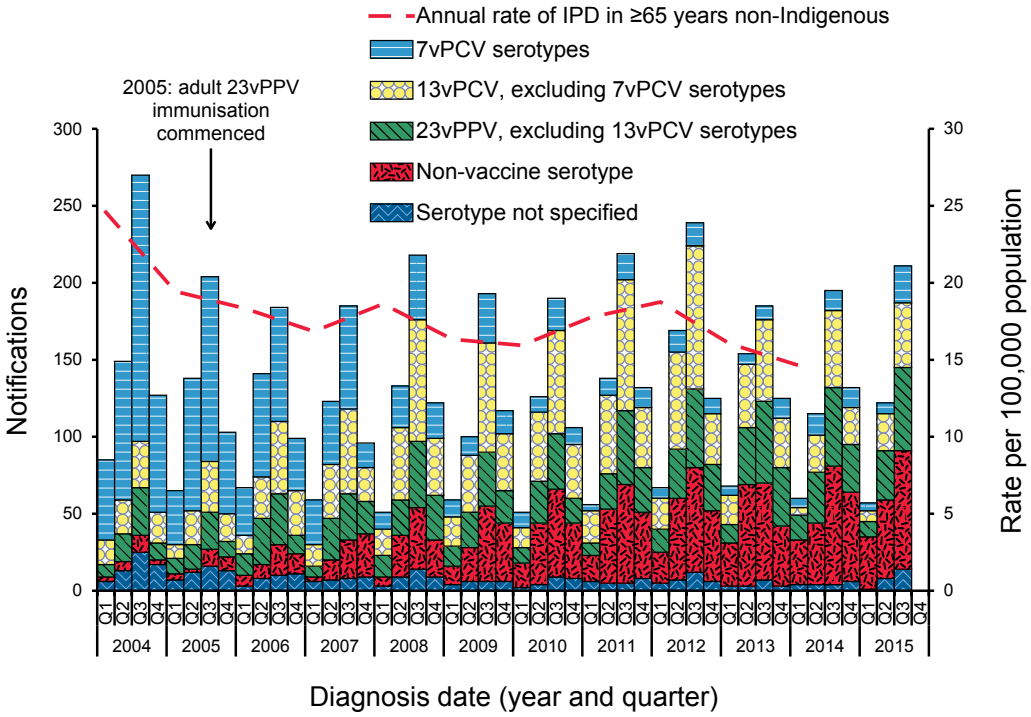
Figure 2: Notifications (2004 to 30 September 2015) and annual rates of all invasive pneumococcal disease (2004 to 2014) in Indigenous Australians aged 50 years or over, Australia, by vaccine serotype group



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). In this report serotype analysis is generally grouped according to vaccine composition.

Figure 3: Notifications (2004 to 30 September 2015) and annual rates of all invasive pneumococcal disease (2004 to 2014) in non-Indigenous Australians aged 65 years or over, Australia, by vaccine serotype group



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

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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.

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Reviewers for *Communicable Diseases Intelligence*, 2015

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