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Original article

Atypical outbreak of Q fever affecting low-risk residents of a remote rural town in New South Wales

Brett N Archer, Cathie Hallahan, Priscilla Stanley, Kathy Seward, Margaret Lesjak, Kirsty Hope, Anthony Brown

Abstract

We investigated an outbreak of Q fever in a remote rural town in New South Wales, Australia. Cases identified through active and passive case finding activities, and retrospective laboratory record review were interviewed using a standard questionnaire. Two sets of case–case analyses were completed to generate hypotheses regarding clinical, epidemiological and exposure risk factors associated with infection during the outbreak. Laboratory-confirmed outbreak cases (n=14) were compared with an excluded case group (n=16) and a group of historic Q fever cases from the region (n=106). In comparison with the historic case group, outbreak cases were significantly more likely to be female (43% vs. 18% males, $P = 0.04$) and identify as Aboriginal (29% vs. 7% non-Aboriginal, $P = 0.03$). Similarly, very few cases worked in high-risk occupations (21% vs. 84%, $P < 0.01$). Most outbreak cases (64%) reported no high-risk exposure activities in the month prior to onset. In comparison with the excluded case group, a significantly increased proportion of outbreak cases had contact with dogs (100% vs. 63%, $P = 0.02$) or sighted kangaroos on their residential property (100% vs. 60%, $P = 0.02$). High rates of tick exposure (92%) were also reported, although this was not significantly different from the excluded case group. While a source of this outbreak could not be confirmed, our findings suggest infections likely occurred via inhalation of aerosols or dust contaminated by *Coxiella burnetii,* dispersed through the town from either an unidentified animal facility or from excreta of native wildlife or feral animals. Alternatively transmission may have occurred via companion animals or tick vectors. *Commun Dis Intell* 2017;41(2):E125–E133.

Keywords: Q fever, *Coxiella burnetii,* disease outbreaks, epidemiology

Introduction

Q fever is the most commonly notified zoonosis in Australia.[1] It is caused by an obligate intracellular bacterium, *Coxiella burnetii,* which is environmentally stable and found widely across the animal kingdom.[2–4] Domestic and feral ruminants, including cattle, goats and sheep, are considered the main sources of human infections. However, a wide range of other animals including native and introduced wildlife, companion animals, and various species of ticks, birds and rodents have all been implicated in human infections.[3,5] Infected animals shed *C. burnetii* in placental tissues, birth fluids, milk, urine and faeces.[4] Transmission typically occurs via airborne dissemination of these materials, or through direct contact with infected animals or other contaminated materials (such as wool, straw or clothing).[4,6] As such, workers in animal-related industries are at increased risk of infection, and vaccination of 'at risk' individuals is routinely recommended.[7]

Disease in humans is challenging to diagnose, especially outside traditional risk settings. Subclinical seroconversion occurs in up to 60% of infected individuals.[8] In the remainder, acute Q fever usually manifests as a severe, 'influenza-like' illness lasting 2–6 weeks; although, presentations such as hepatitis or pneumonia are common, particularly in some parts of the world. Neurological manifestations, myocarditis or pericarditis, may occur in some cases, and pregnant women are at risk of various obstetric complications. A protracted chronic infection may develop in 10%–30% of acute cases, presenting as endocarditis, recrudescent granulomatous lesions or post-Q fever fatigue syndrome. Persons who are immunosuppressed, those with a pre-existing heart valve defect, and pregnant women are at highest risk for chronic infections.[4]

In February 2015, an astute infectious disease physician notified public health authorities of an increase in patients presenting with a febrile illness requiring hospitalisation, including 2 cases of Q fever, in the town of Lightning Ridge, New South Wales. An investigation was conducted with the aim of characterising the epidemiological and clinical profile of cases and identifying the source of infections to guide implementation of control measures.

Methods

Setting

Lightning Ridge is a remote rural town in north-western New South Wales within the Walgett Shire Local Government Area (LGA), near the southern border of Queensland. The town has a culturally-diverse population of approximately 2,400 people (2014 estimate), of which 29.7% are aboriginal people (census 2011 LGA estimate).[9] The area is classified as remote (ASGC-RA4), is semi-arid with long-term annual mean rainfalls of <470 mm, and is subject to regular droughts.[10] The town's industries include tourism, agriculture and opal-mining, with some 1,900 informal or formal mining camps located on opal mining fields in the vicinity of Lightning Ridge and Grawin/Glengarry areas.[11] Local agriculture is predominantly sheep grazing for wool and meat.[9]

Case finding and investigations

Case finding activities were both passive and active. Routine (passive) notifications were sourced from the NSW Notifiable Diseases Information Management System (NCIMS). Between 20 February and 31 May 2015, active surveillance for possible cases was undertaken through the Lightning Ridge Health Service, the sole local general practice and the local ambulance service, and by means of an alert to general practitioners in surrounding towns. Healthcare professionals in the area were asked to maintain a high index of suspicion for possible cases, undertake polymerase chain reaction (PCR) and serological testing for Q fever, and commence empiric treatment. A retrospective review was also conducted of all Q fever test requests received from Lightning Ridge and surrounding areas by the Centre for Infectious Diseases and Microbiology Laboratory Services, Institute for Clinical Pathology and Medical Research, Pathology West, Westmead, which is the primary reference laboratory for the region. Public health authorities in neighbouring jurisdictions were alerted to the outbreak and asked to report cases who travelled to the region.

A possible case was defined as any person with onset of an acute febrile illness, (which may include a temperature >38°C, severe headache, extreme fatigue, sweating/chills, myalgia/arthralgia, and/or malaise), from 1 December 2014 to 31 May 2015, who, in the 1 month prior illness onset, was resident in the vicinity of Lighting Ridge (±40 km), had a history of travel to the area, or had direct contact with livestock or wildlife from the area.

A confirmed case was defined as a possible case who met the Communicable Diseases Network Australia definition (i.e. clinically compatible illness plus laboratory-suggestive evidence, or laboratory-definitive evidence).[12] Cases with only laboratory-suggestive evidence (i.e. single detection of specific IgM), or a single negative serology test, were actively followed-up to obtain convalescent specimens to provide laboratory-definitive evidence of acute infection (i.e. demonstrated seroconversion), or exclude the case. Cases were excluded if they had consistently negative paired sera samples, had a non-compatible illness or alternate diagnosis with at least 1 negative serology test, evidence of being vaccinated against Q fever, or evidence of a previous diagnosis of Q fever or chronic/persistent infection.

Local public health unit staff interviewed all possible and confirmed outbreak cases by phone using a standardised questionnaire. Walgett Shire Council Rangers, North West Local Land Services, local veterinarians and the Department of Primary Industries were additionally consulted regarding recent animal stocking and transport activities.

A site visit to Lightning Ridge was undertaken to review environmental conditions during the outbreak, engage local stakeholders and identify potential sources of infection.

Hypotheses-generating analytic studies

Two sets of nested, case–case analyses were completed to generate hypotheses with respect to clinical, epidemiological and exposure risk factors associated with infection during the outbreak. In the first, outbreak cases were compared to an excluded case group, which was defined as all possible case notifications that tested negative for Q fever in consecutive serum samples, had no evidence of being vaccinated or laboratory evidence of past infection (i.e. otherwise susceptible to *C. burnetii* infection), and had completed an interview.

In the second, outbreak cases were compared with an historic profile of Q fever notifications (as at 25 June 2015) with a reported date of illness onset prior to the outbreak during 2010–2014 and usual place of residence within Western New South Wales and Far West Local Health Districts (LHDs) (hereafter the historic case group). Notification records were extracted NCIMS, which included a basic case history collected by local public health units as part of routine case investigations and captured within standardised, electronic surveillance templates.

Statistical analyses

Odds ratios (OR) and corresponding 95% confidence intervals (95% CI) were calculated to measure associations for categorical variables. Fisher's Exact Test was performed where the expected cell

frequency in a contingency table was ≤5. As age demonstrated significant deviations from normality using the Kolmogorov-Smirnov test, the non-parametric Mann-Whitney U Test was performed to measure significant differences between groups. The alpha level was set at 0.05 for all analyses. Year-specific incidence (notification) rates or annual average notification rates were calculated using annual mid-year population projections published by the NSW Ministry of Health.[13]

Environmental investigations

Data on daily and average monthly rainfall, and wind speed, for Lightning Ridge and Walgett for December 2014 to February 2015 were sourced from the Australian Government Bureau of Meteorology.[10]

Results

Walgett Shire LGA has historically experienced the highest annual incidence of Q fever among Far West and Western New South Wales LHDs, with an average annual incidence of 47 cases per 100,000 population in 2010–2014. In comparison, rates ranged from 0–40 cases per 100,000 over the same period within other LGAs in these LHDs. Between 1 December 2014 and 31 May 2015, the outbreak investigation revealed 44 possible cases in and around Lightning Ridge, of which 14 cases met the confirmed case definition: a diagnosis of acute Q fever infection was excluded in the remainder. These 14 confirmed outbreak cases represented a significant increase in disease incidence for the LGA to 165 per 100,000 from baseline ($P = 0.001$). In case–case analyses, confirmed outbreak cases were compared against 16 individuals and 106 historic Q fever cases in the excluded case group and historic case group, respectively.

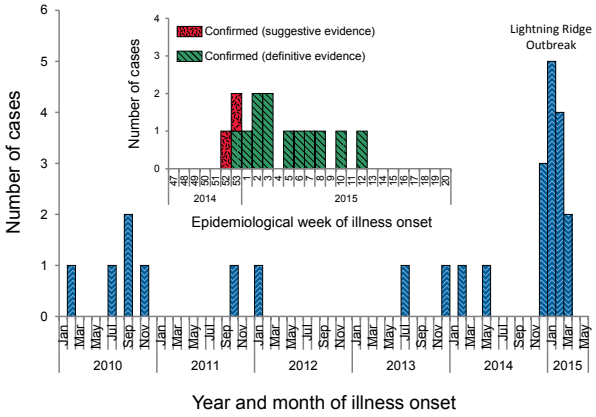
Demographics

The majority of confirmed outbreak cases were aged 40 years or over (79%), and were statistically similar in age to both the excluded case group and historic case groups (Table 1). A significantly larger proportion of outbreak cases were, however, female (43%) or identified as Aboriginal (29%) when compared with the historic Q fever cases (18% female, 7% Aboriginal people).

Clinical presentation and laboratory confirmation

Symptom onset dates for the 14 confirmed outbreak cases were clustered over a period of 12 weeks between 22 December 2014 and 26 March 2015 (Figure). Most confirmed outbreak cases reported symptoms typical of Q fever, including: high fever, rigors with profuse sweating, severe headache, malaise and extreme fatigue lasting several weeks (Table 1). With the exception of 1 case (for whom liver function tests were only performed post-recovery), all cases had abnormally elevated transaminases. Nine cases (64%) required hospitalisation; however, there were no reports of complications or deaths. When compared with the excluded case group, presenting with headache, malaise, fever, rigors, elevated transaminases and being hospitalised were all predictive of having a laboratory confirmed diagnosis.

Figure: Number of confirmed Q fever notifications, Walgett Shire Local Government Area, 1 January 2010 to 31 May 2015, by month, year and epidemiological week of illness onset



Of the 14 confirmed outbreak cases, 12 (86%) had laboratory-definitive evidence of *C. burnetii* infection: 1 by PCR only, 1 by PCR and demonstrated seroconversion, and 10 by seroconversion only. Two confirmed outbreak cases had a clinically compatible illness and laboratory-suggestive evidence of infection (detection of specific IgM in single serum sample), but did not submit convalescent samples. Of the 11 cases that demonstrated seroconversion, 6 (55%) had an initial, acute serum sample that was negative on serology tests performed, IgM negative by enzyme immunoassays, or Phase II antibody titre of less than 4 by compliment fixation testing or immunofluorescence assays.

Exposure history

The majority (79%) of confirmed outbreak cases were retired, unemployed, or worked in town-based, non-animal related occupations (Table 2). Outbreak cases were significantly less likely to work in a high-risk occupation than the historic case group (OR: 0.05, 95% CI: 0.01–0.22, $P < 0.01$), while being statistically similar to the excluded case group (OR: 1.6, 95% CI: 0.02–11.7, $P = 1.0$).

Table 1: Demographic and clinical characteristics of confirmed Q fever cases notified during an outbreak in Lightning Ridge, New South Wales compared with historic case and excluded case groups

	Outbreak cases (22 Dec 2014–26 Mar 2015)		Historic cases (1 Jan 2010 – 30 Nov 2015)		Excluded cases (3 Jan 2015–4 Mar 2015)		Outbreak vs historic cases		Outbreak vs excluded cases	
Demographics										
	Years*	IQR	Years	IQR	Years	IQR	*P* value		*P* value	
Median age	57.5	40	36.9	27	57.5	40	0.09		0.78	
	n/N*	%	n/N*	%	n/N*	%	OR (95% CI)	*P* value	OR (95% CI)	*P* value
Female	6/14	43	19/106	18	7/16	44	3.4 (1.1–11.1)	0.04†	1.0 (0.2–4.1)	0.75
Aboriginal	4/14	29	5/70	7	2/15	13	5.2 (1.2–22.7)	0.03†	2.6 (0.4–17.2)	0.39
Clinical presentation										
Headache	13/13	100	–		9/16	56	–	–	Inf.	<0.01†
Malaise	14/14	100	–		8/16	50	–	–	Inf.	<0.01†
Lethargy	14/14	100	–		14/16	88	–	–	Inf.	0.49
Fever	13/14	93	–		7/14	50	–	–	13.0 (1.3–128.1)	0.03†
Rigors (with sweating)	13/14	93	–		7/15	47	–	–	14.9 (1.5–144.2)	0.01†
Elevated transaminases	13/14	93	–		2/9	22	–	–	45.5 (3.5–594.7)	<0.01†
Hospitalised	9/14	64	–		2/16	13	–	–	12.6 (2.0–79.4)	<0.01†

IQR: Interquartile range.

OR (95% CI): Odds ratio and corresponding 95% confidence interval.

Inf.: Odds ratio approaches infinity, cannot be calculated.

* Denominators exclude cases with missing data.

† Statistically significant difference between groups at α=0.05.

– Not applicable, or data not available/unreliably reported.

Table 2: Occupations of confirmed Q fever cases notified during an outbreak in Lightning Ridge, New South Wales compared with historic case and excluded case groups

Occupation in the month prior to illness onset*	Outbreak cases (22 Dec 2014–26 Mar 2015)		Historic cases (1 Jan 2010–30 Nov 2015)		Excluded cases (3 Jan 2015–4 Mar 2015)	
	n	%	n	%	n	%
% High-risk occupation	3	21	53†	84	2	14
Farmer or resident on farm	2	14	38	60	1	7
Shearer or wool classer	1	7	1	2	0	0
Stockyard worker or stock transporter	0	0	7	11	0	0
Abattoir and other meat industry worker	0	0	5	8	0	0
Veterinary or wildlife worker	0	0	2	3	1	7
Low-risk (non-animal related) occupation	11	79	10	16	12	86
Retired or unemployed	5	36	0	0	6	43
Construction, maintenance, mining or similar	3	21	0	0	2	14
Office, school worker or similar	3	21	0	0	4	29
Other (or undefined) low-risk occupation	0	0	10	16	0	0
Total	14	100	63	100	14	100

* Occupation not reported (or age <16 years) for n=43 historic cases and n=2 excluded cases.

† Statistically significant difference in comparison WITH the outbreak group at α=0.05.

Five confirmed outbreak cases (36%) reported participating in one or more high-risk activities including, slaughtering, shooting, transporting, and/or shearing livestock or feral animals (Table 3). For the remaining 64% of cases, interviews did not identify any apparent high-risk exposure activities. In comparison with the excluded case group, however, a significantly increased proportion of confirmed outbreak cases reported owning or having direct contact with dogs (100% of cases vs. 63% of excluded cases, $P = 0.02$), or sighting kangaroos on their residential property (100% of cases vs. 60%, $P = 0.02$). Most confirmed outbreak cases additionally reported being exposed to ticks (92%) with many noting tick bites (60%); however, statistically similar rates of tick exposure were observed in the excluded case group.

Environmental investigations

Investigations by North West Local Land Services, the Department of Primary Industries and local public health units were unable to identify any specific animal intensive enterprises, stocking, transport, birthing, animal congregations such as shearing or similar events occurring in the area within the month before the outbreak that could be considered a possible point source of infection. Lightning Ridge was not considered a regular route or stop for livestock transport trucks, and cases did not reside along major roads.

Reports of a notable increase in kangaroo activity in and around town were supported by sightings of a large number of kangaroos on residential properties during site visits. Reports of increased tick activity and exposure by cases were supported by observations by local veterinarians.

Data from the Bureau of Meteorology confirmed a drought in Lightning Ridge in the months preceding the outbreak. In 2014, the area received a total of 256 mm of rain compared with an annual mean of 468 mm (measurements available since 1997). A total of 8 mm and 25 mm of rain fell in October and November respectively. This was followed by a slight increase to 49 mm in December 2014 (the highest for the year) amidst average daily temperatures of 36°C (max 41°C). Drought conditions persisted during January to March 2015, with just 4–11 mm of rain falling each month. The town itself has artesian water available and so presents as a green oasis for local wildlife. Although wind gust data were not available for Lightning Ridge, Walgett Airport (approximately 67 km away) reported gusts of up to 65–83 km/h during December 2014 to February 2015, which supported anecdotal reports of dust storms occurring in Lightning Ridge in the month preceding the outbreak.

Table 3: Exposure history of confirmed Q fever cases notified during an outbreak in Lightning Ridge, New South Wales compared with excluded case group

Exposures in the month prior to illness onset*	Outbreak cases (22 Dec 2014–26 Mar 2015)		Excluded cases (1 Jan 2010–30 Nov 2015)		OR (95% CI)	*P* value
	n/N	%	n/N	%		
Kangaroos on residential property	14/14	100	9/15	60	Inf.	0.02†
Direct contact with dogs	14/14	100	10/16	63	Inf.	0.02†
Direct contact with ticks	12/13	92	12/15	80	3.0 (0.3–33.1)	0.60
Tick bites	6/10	60	8/13	62	0.9 (0.2–5.1)	1.00
Travel	6/14	43	4/15	27	2.1 (0.4–9.8)	0.45
Direct contact with livestock or wildlife	5/14	36	2/15	13	3.6 (0.6–22.9)	0.21
Sheep	4/14	29	1/15	7	5.6 (0.5–57.9)	0.17
Cattle	2/14	14	0/15	0	Inf.	0.22
Goats	1/14	7	0/15	0	Inf.	0.48
Pigs	1/14	7	0/15	0	Inf.	0.48
Feral pigs	2/14	14	1/15	7	2.3 (0.2–29.0)	0.60
Feral goats	2/14	14	0/15	0	Inf.	0.22
Livestock transport	2/14	14	0/15	0	Inf.	0.22
Slaughtering	2/14	14	1/15	7	2.3 (0.2–29.0)	0.60
Shearing	1/14	7	1/15	7	1.1 (0.1–19.0)	1.00
Shooting	1/14	7	2/15	13	0.5 (0.0–6.2)	1.00
Animal birthing	0/14	0	1/15	7	–	1.00
Other veterinary practices	0/14	0	0/15	0	–	1.00
Visited meat processors, zoo or saleyard	0/14	0	0/16	0	–	1.00
Wool classing	0/14	0	0/16	0	–	1.00

OR (95% CI): Odds ratio and corresponding 95% confidence interval.

Inf.: Odds ratio approaches infinity, cannot be calculated.

* Exposure events are not mutually exclusive and denominators exclude cases with missing data.

† Statistically significant difference between groups at α=0.05.

– Not applicable, or data not available/unreliably reported.

Discussion

We observed an unusual outbreak of Q fever affecting residents of a small mining town in Western New South Wales. Cases had an epidemiological and risk profile that varied from the historic profile of Q fever notifications from the region. Outbreak cases were significantly more likely to be female and identify as Aboriginal. Moreover, the majority of outbreak cases were residents in town, did not work in a high-risk occupation, and did not participate in any high-risk activities prior to their illness onset.

We additionally observed that symptoms typical of Q fever (namely headache, malaise, fever, rigors) and having elevated transaminases were predictive of infection when compared with laboratory-excluded cases. The majority of cases (71%) were only confirmed through testing of paired acute and convalescent serum specimens, highlighting the critical need for clinical and public health authorities to follow up patients to collect repeat serum, even if they have since recovered. Moreover, initial serology tests were negative in 60% of seroconverted cases, suggesting a review of laboratory records to identify cases without convalescent serology should be routinely considered as part of Q fever outbreak investigations.

The epidemiological profile and lack of definitive exposures suggests a number of hypotheses about the sources of infection in this outbreak. Most plausible is the inhalation of aerosols or dust contaminated by *C. burnetii,* dispersed through the town either from unidentified animal facilities or from excreta of native wildlife or feral animals. This hypothesis is supported by case–case analyses, which suggest an association between kangaroo activity on residential properties prior to onset

and laboratory-confirmed infection. Moreover, the observed dry conditions and dust storms, and activities which may disturb animal faeces, may have played a role in facilitating aerosol transmission. Indeed, studies elsewhere have demonstrated that while infection in individuals without apparent occupational or incidental exposures are relatively infrequent,[14] high seroprevalence rates exist in putatively low-risk communities,[15] and the largest Q fever outbreak to date was attributed to community-wide dispersal of *C. burnetii* up to 5 km from source farms.[16] Kangaroos have been shown to carry and transmit Q fever,[17,18] and a recent New South Wales case series postulated kangaroo faeces may pose a risk (especially if aerosolised by wind or lawn mowing) with 7% of patients recalling macropod contact.[19] Studies have demonstrated the infectious dose of *C. burnetii* may be as little as one aerosolised rickettsia,[20] suggesting even low levels of environmental contamination may cause an outbreak.

Transmission via tick vectors offers a strong alternate hypothesis. This is supported by the findings of high rates of exposure to ticks. Moreover, shortly after the outbreak, we were notified of a confirmed case of Q fever in a Victorian woman who camped in Lighting Ridge in the 2 weeks prior to her illness onset, receiving multiple tick bites, with no other discernible high risk exposures. *C. burnetii* was detected by PCR in multiple ticks collected from the case's body (personal communication: Dr Andrew Fuller, Infectious Diseases Physician, The Alfred Hospital). Numerous field studies show that many macropod-associated tick species (including human biting ticks) carry *C. burnetii*.[5,19] While it is largely accepted ticks play a role in transmission between wild fauna and domestic ruminants, the vector capacity of ticks to transmit *C. burnetii* to humans has been contested and such cases remain infrequently reported.[4,5] Experimental systems, however, suggest a substantial risk posed by tick excreta, with potential human infections through inhalation (e.g. during removal and disposal of ticks, or aerosol-generating procedures such as shearing), direct contact (e.g. crushing ticks with bare hands), or tick bites.[4,5]

Transmission via companion animals offers a third hypothesis. Transmission from pets has been implicated in past outbreaks; however, this was restricted to individuals exposed during aerosol generating veterinary procedures.[3,4,21]

Our findings are subject to a number of limitations. Firstly, case identification was dependent on presentation to healthcare and clinical suspicion of infection prompting laboratory testing. Studies elsewhere demonstrate each Q fever notification corresponds to up to 12 infections in the community,[22] suggesting many asymptomatic and subclinical infections were missed. Secondly, the question must be asked if the detected increase may be attributed to active case finding activities alone. While it is likely that Q fever is underreported in routine surveillance data, the majority of confirmed outbreak cases were reported following hospital admission prior to the period of active surveillance, and no new cases were detected in the 2 months following the outbreak despite ongoing surveillance; supporting the existence of a true outbreak. Thirdly, our study lacked a robust comparison group, which may have resulted in the introduction of selection biases. Although the excluded case group were demonstrated by laboratory tests to be susceptible to, yet remained uninfected by *C. burnetii*, analyses were restricted to a subset of this group with completed interviews, and these individuals were symptomatic of another illness requiring healthcare and likely affecting their activities. Therefore, this comparison group may not have had the same distribution (risk) of exposures as cases. Fourthly, historic notification data may be skewed towards certain demographic groups and persons with high risk exposures, and characteristics such as Aboriginal status may be underreported. Aboriginal status and occupation were not available for 41% and 34% of historic notifications, respectively. Therefore, the observed risk difference in demographic and occupational profile of cases in comparison to historic trends may be subject to measurement bias and over exaggerated here. More robust epidemiological studies, ideally including a seroprevalence component, are therefore needed to adequately test the hypotheses proposed here.

The National Q Fever Vaccination Program was successful in reducing the incidence of disease in high-risk settings such as abbatoirs.[23] While the majority of cases notified in Far West and Western New South Wales LHDs continue to have occupational exposures, and could be prevented through current vaccination recommendations, this outbreak represents a deviation from the norm. The documentation of infections in persons who are typically at low-risk highlights that Q fever should not be excluded from the differential diagnosis based on risk history alone, especially in regional and remote settings, or communities neighbouring animal facilities. With the decline in incidence across the State, there is an increased capacity to detect and investigate infections in low-risk populations. Sporadic cases and outbreaks in rural/regional towns, similar to that investigated here, may become more commonplace. It is critical that such events be thoroughly investigated to better inform the transmission and source dynamics of Q fever, and build an evidence-base for future interventions.

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Demographic and geographical risk factors for gonorrhoea and chlamydia in greater Western Sydney, 2003–2013

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Abstract

Introduction: Notification rates of sexually transmitted infections (STIs) have increased in New South Wales as elsewhere in Australia. Understanding trends in chlamydia and gonorrhoea notifications at smaller geographical areas may assist public health efforts to deliver targeted STI interventions.

Methods: Routinely collected disease notification data from 2 local health districts within the greater Western Sydney area were analysed. De-identified notifications of gonorrhoea and chlamydia were extracted for people aged over 15 years during the period 1 January 2003 to 31 December 2013. Sex-specific and age-specific population notification rates for each infection were calculated. Incidence rate ratios were also calculated with age group, sex, year and local government area (LGA) of residence as explanatory variables.

Results: Rates of gonorrhoea and chlamydia increased among males and females over the period. Males had a 4-fold increased risk of gonorrhoea ($P<0.0001$). Compared with the 30–44 years age group, young people aged 15–29 years had a 70% increased risk of gonorrhoea and a 4-fold increased risk of chlamydia (P values <0.0001). Chlamydia notifications demonstrated smaller and more uniform annual increases across LGAs compared with gonorrhoea notifications, which appeared more highly clustered.

Conclusion: Analysis of notification rates of chlamydia and gonorrhoea in the greater Western Sydney area suggest that young people aged 15–29 years and residents of particular LGAs are at greater risk of infection. A limitation was the unknown effect of patterns of testing. Nevertheless, these results can support the planning of local sexual health clinical services as well as the design of targeted health promotion interventions. *Commun Dis Intell* 2017;41(2):E134–E141.

Keywords: gonorrhoea; *Neisseria gonorrhoeae*, *Chlamydia trachomatis*; Sydney; sexual health

Introduction

Australia has experienced a sharply increasing trend in detected cases of gonorrhoea (*Neisseria gonorrhoeae)*, particularly in men, since 2009.[1] Since chlamydia (*Chlamydia trachomatis*) became notifiable in Australia in 1999, overall rates have also increased each year until 2013, when a slight decrease was observed for the first time.[1] The increase in notification of these 2 infections over time are most likely due to multiple factors including increased screening, use of more sensitive tests to screen and diagnose as well as higher-risk sexual behaviours among subpopulations.[2]

In Australia, men who have sex with men (MSM) are a population particularly at risk of sexually transmitted infections (STIs) such as gonorrhoea, chlamydia, syphilis and HIV.[3] Since 2004, rates of unprotected anal intercourse with casual male partners have increased among MSM.[4] High rates of gonorrhoea observed in areas with large MSM populations, such as inner Sydney, support the theory that unprotected sex among MSM may be a significant driver of increased notifications.[5] A national study on gonorrhoea epidemiology demonstrated that a dual epidemic in Australia among Aboriginal people in remote areas, and non-Aboriginal men in metropolitan areas contributed substantially to the overall increase.[2] The rise in gonorrhoea notifications is especially concerning as it coincides with increasing drug resistance to currently used antimicrobials in Australia.[3]

The greater Western Sydney area is an outer metropolitan region with a population of over 2 million people, more ethnic and linguistic diversity compared with inner Sydney, and suburbs that are among the most socially disadvantaged in urban New South Wales.[6] It is unknown how cultural and behavioural factors might impact on the effectiveness of sexual health-related initiatives, especially for MSM for whom fewer specific services exist in this region compared with inner Sydney. Providing locally adapted clinical services and health promotion interventions pose unique challenges in this context.

Understanding local patterns of STI transmission can assist public health efforts to deliver targeted

interventions. An analysis was undertaken of routinely collected disease notification data from 2 local health districts within the greater Western Sydney area, Western Sydney Local Health District (WSLHD) and Nepean Blue Mountains Local Health District (NBMLHD).

WSLHD has a population of approximately 885,000 persons and covers the local government areas (LGAs) of Auburn, Blacktown, Holroyd, The Hills Shire and Parramatta. Around 43% of the WSLHD population were born overseas and 1.7% were Aboriginal or Torres Strait Islander people.[7] NBMLHD has a population of around 356,000 people and covers the LGAs of Penrith, Blue Mountains, Hawkesbury and Lithgow. Around 22% of the NBMLHD population were born overseas and 3.3% were Aboriginal or Torres Strait Islander people.[8,9]

The purpose of this study was to gain a better understanding of the population groups at greatest risk of STIs and to inform more targeted health promotion and clinical service delivery strategies. As gonorrhoea and chlamydia occur more commonly than other STIs such as syphilis and HIV, the study focussed on these 2 infections to allow meaningful analysis of trends and risk factors at the level of small geographical areas.

Methods

The Notifiable Conditions Information Management System (NCIMS) is an Internet based system used by New South Wales public health units and Health Protection NSW to register communicable disease notifications including gonorrhoea and chlamydia.[10] De-identified notifications of gonorrhoea or chlamydia were extracted from NCIMS for people aged over 15 years residing within boundaries of WSLHD or NBMLHD.

Notifications received during the 11-year period 1 January 2003 to 31 December 2013 were extracted for analysis in August 2014. Fields from the dataset included date of first notification, age, sex, Aboriginal and Torres Strait Islander status, Local Health District (LHD) of residence, LGA of residence, site of infection and laboratory testing method. If sex or age was recorded as missing the notification was excluded from further analysis. Data on sexual exposure were not routinely collected in the New South Wales database between 2003 and 2013.

Annual counts of all notifications of gonorrhoea and chlamydia were assembled separately and used to calculate sex specific population notification rates for each STI over the 11-year period of analysis. Age specific notification rates were also calculated by age group (15–29, 30–44, 45–59 and 60 years or over). Mid-calendar year resident population estimates were used in rate calculations and were obtained from the Australian Bureau of Statistics (ABS).[11]

In the absence of data on sexual exposure in the NCIMS database, annual counts of any oropharyngeal or anorectal infection of gonorrhoea or chlamydia in males were calculated and used to calculate notification rates among all males over 15 years. This was done to provide an approximate indicator of the trend in notifications specific to MSM.

The incidence rate ratio of gonorrhoea was estimated to allow comparisons by variables of interest. A Poisson regression model was fitted to gonorrhoea notification data with age group, gender, year and LGA of residence as explanatory variables. An interaction term between year and LGA was also included to account for differing trends within LGAs.

Similarly, the incidence rate ratio of chlamydia was estimated by fitting a negative binomial regression model to chlamydia notification data. A Poisson model was not suitable because of over-dispersion of the data. As with the model for gonorrhoea, age group (as above), gender, year, LGA and an interaction term of year and LGA were included as explanatory variables. Population was included as an offset. Confidence intervals (95%) were calculated for each relative risk. Indicators of goodness of fit were assessed.

Non-identified data, as defined by the Australian National Health and Medical Research Council's National Statement on Ethical Conduct in Human Research were used for this study and ethics approval was not required. Analysis was conducted using SAS version 9.3 through SAS Enterprise Guide version 5.1.

Results

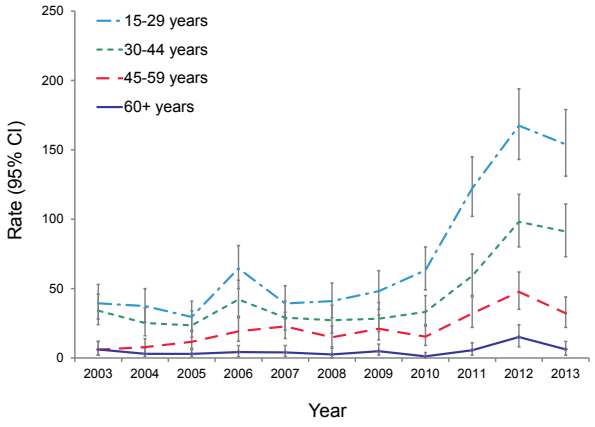
Descriptive analysis

A total of 2,513 notifications of gonorrhoea and 19,626 notifications of chlamydia were recorded among residents of WSLHD and NBMLHD between 1 January 2003 and 31 December 2013. Age or sex information was missing from 9 gonorrhoea notifications and 85 chlamydia notifications (0.4% of all notifications). These notifications were excluded from further analyses leaving a final number of 2,504 gonorrhoea notifications (80% male) and 19,541 chlamydia notifications (42% male) in the analysis.

The completeness of data on Aboriginal and Torres Strait Islander status was poor, with 56% of gonorrhoea notifications and 99% of chlamydia notifications recorded as 'unknown' or 'missing'. Data on Aboriginal and Torres Strait Islander status were therefore not included in further analysis. The greater completeness of gonorrhoea notifications can be explained by focused efforts by public health units to confirm Aboriginal and Torres Strait Islander status given the known disproportionate burden of gonorrhoea among Aboriginal and Torres Strait Islander peoples.

The rate of gonorrhoea in males showed a dramatic rise, particularly in 2011 and 2012. A steep upward trend was particularly marked in young men aged 15–29 years (Figure 1) who had a peak annual rate in 2012 of 167 per 100,000 population. There was a similar trend in females although of a lesser magnitude (Figure 2). While genitourinary infections in males accounted for the majority of notifications, there was an increasing trend in notifications of any anorectal or oropharyngeal gonorrhoea in men from 2010 (Figure 3) suggesting that transmission between MSM may account in part for the overall rise in notifications.

Figure 1: Notification rate of gonorrhoea in males, Western Sydney and Nepean Blue Mountains Local Health Districts, 2003 to 2013, by age group



Chlamydia rates steadily increased in both males and females throughout the study period although a steeper trend was evident from 2010. The highest rate and most marked increase was in females aged 15–29 years (Figure 4) followed by males aged 15–29 years (Figure 5). Anorectal or oropharyngeal chlamydia in males showed a rise from 2011 that peaked in 2012 and then slightly declined in 2013 (Figure 6).

Figure 2: Notification rate of gonorrhoea in females, Western Sydney and Nepean Blue Mountains Local Health Districts, 2003 to 2013, by age group

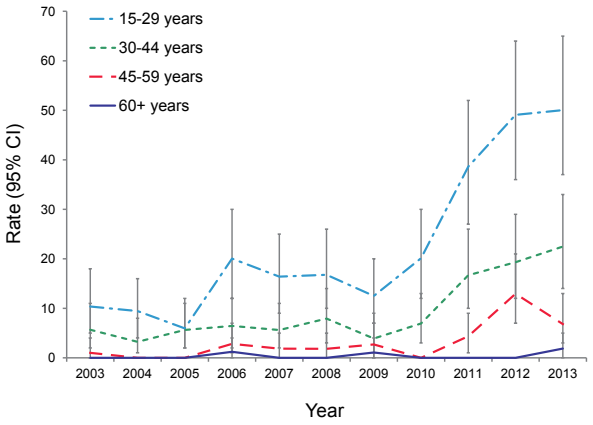


Figure 3: Notification rate of any anal, rectal oropharyngeal gonorrhoea in males, Western Sydney and Nepean Blue Mountains Local Health Districts, 2003 to 2013, by age group

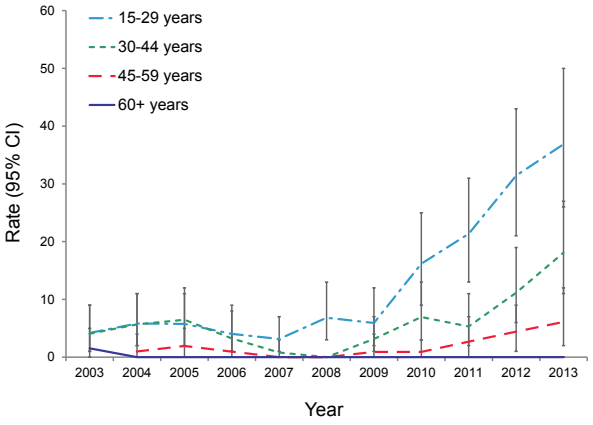


Figure 4: Notification rate of chlamydia in females, Western Sydney and Nepean Blue Mountains Local Health Districts, 2003 to 2013, by age group

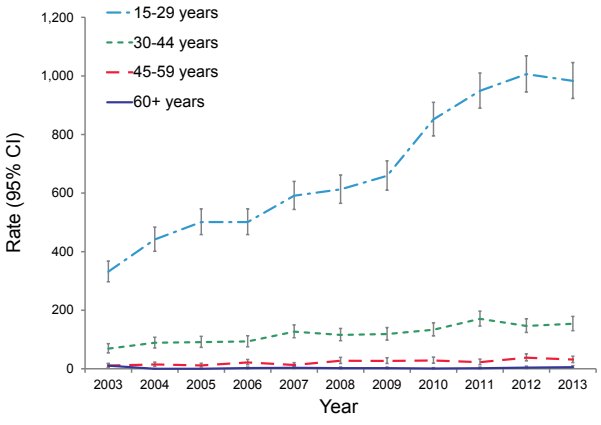


Figure 5: Notification rate of chlamydia in males, Western Sydney and Nepean Blue Mountains Local Health Districts, 2003 to 2013, by age group

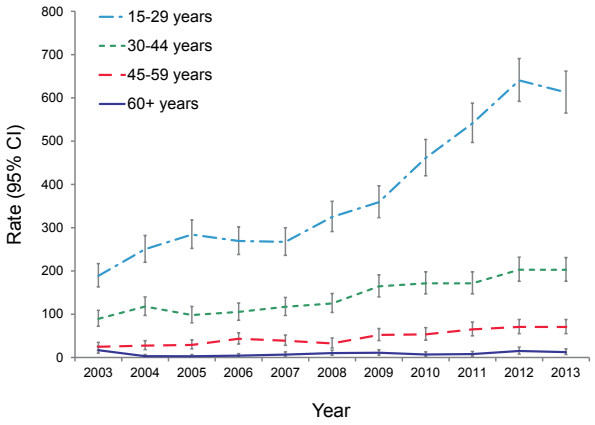
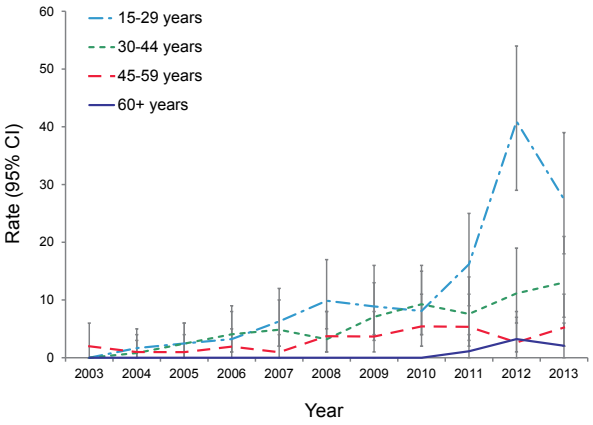


Figure 6: Notification rate of any anal, rectal oropharyngeal chlamydia in males, Western Sydney and Nepean Blue Mountains Local Health Districts, 2003 to 2013, by age group



Regression analysis

Tests for goodness of fit confirmed that the models were satisfactory for both the gonorrhoea data (scaled deviance/degrees of freedom = 1.21) and the chlamydia data (scaled deviance/degrees of freedom =1.14) (Table 1).

Controlling for age, LGA and year, males had a 4-fold increased risk of gonorrhoea ($P<0.0001$) but a reduced risk of chlamydia compared with females (IRR 0.93; $P=0.038$). Compared with those aged 30–44 years, young people aged 15–29 years had a 70% increased risk of gonorrhoea and a 4-fold increased risk of chlamydia when controlling for sex, LGA and year (P values < 0.0001).

Controlling for age and sex, the trend of gonorrhoea and chlamydia notifications by geographical area differed over the study period. Gonorrhoea notifications demonstrated a greater annual rate increase, particularly in Auburn LGA (25% per year, 95%CI 19% to 32% increase), Hawkesbury LGA (24% per year, 95%CI 16% to 33% increase), Penrith LGA (23% per year, 95%CI 19% to 27% increase) and Blacktown LGA (22% per year, 95%CI 19% to 24% increase). Chlamydia notifications demonstrated smaller and more uniform annual increases across LGAs (Table 2).

Discussion

A critical part of the public health response to STIs is to 'know the local epidemic'. This study illustrates the benefits and limitations of routinely collected data to support this aim.

The analysis demonstrates that gonorrhoea and chlamydia notification rates have increased overall and by LGA within the greater Western Sydney area. Young people aged 15–29 years and people residing in specific geographical areas appear to be at higher risk than other population groups. In addition, an analysis of data on site of infection, as a proxy measure for sexual exposure, suggests an increase in transmission between MSM. Importantly, our analysis could not provide further insight on key population groups especially Aboriginal people and Culturally and Linguistically Diverse communities, or on the influence of testing.

Table 1: Likelihood ratio statistics from regression models

Variable	Gonorrhoea			Chlamydia		
	Degrees of freedom	Chi-squared test statistic	*P*-value	Degrees of freedom	Chi-squared test statistic	*P*-value
Age	3	1495.76	<0.0001	3	1888.77	<0.0001
Sex	1	1164.5	<0.0001	1	4.12	0.0423
Local Government Area	8	108.67	<0.0001	8	130.7	<0.0001
Year	1	360.71	<0.0001	1	76.2	<0.0001
Year and Local Government Area interaction	8	51.13	<0.0001	8	57.43	<0.0001

Table 2: Association between demographic factors and gonorrhoea and chlamydia notifications

Variable	Gonorrhoea			Chlamydia		
	Rate ratio estimate	95% CI	*P* value	Rate ratio estimate	95% CI	*P* value
Sex						
Female	ref	ref	ref	ref	ref	ref
Male	3.98	3.63,4.36	<0.0001	0.93	0.87,0.99	0.038
Age						
15–29	1.71	1.59,1.84	<0.0001	3.92	3.65,4.2	<0.0001
30–44	ref	ref	ref	ref	ref	ref
45–59	0.47	0.41,0.52	<0.0001	0.27	0.25,0.3	<0.0001
60+	0.11	0.08,0.14	<0.0001	0.043	0.04,0.05	<0.0001
Local Government Area trend (per year)						
Blacktown	1.22	1.19,1.24	<0.0001	1.11	1.09,1.14	<0.0001
Auburn	1.25	1.19,1.32	<0.0001	1.12	1.08,1.15	<0.0001
Baulkham Hills	1.18	1.13,1.22	<0.0001	1.11	1.08,1.14	<0.0001
Blue Mountains	1.16	1.07,1.25	<0.0001	1.07	1.03,1.10	<0.0001
Hawkesbury	1.24	1.16,1.33	<0.0001	1.14	1.10,1.18	<0.0001
Holroyd	1.14	1.10,1.19	<0.0001	1.12	1.09,1.16	<0.0001
Lithgow	0.86	0.73,1.02	0.08	1.16	1.11,1.22	<0.0001
Parramatta	1.13	1.11,1.16	<0.0001	1.02	0.99,1.05	0.09
Penrith	1.23	1.19,1.27	<0.0001	1.15	1.11,1.18	<0.0001

Ref. = Reference group for negative binomial regression analysis

Previous studies exploring the spatial epidemiology of STIs identified gonorrhoea as the most highly clustered infection.[12,13] It has been suggested that the phase of the STI epidemic (growth, hyperendemic, decline or endemic) in conjunction with the density of the sexual network is associated with the geographical distribution of disease.[14] Incorporating geographical surveillance into routine STI surveillance has been proposed as an important tool in being able to implement geography-specific and phase-specific STI control strategies.[13] Our results support previous studies that identify gonorrhoea as having a more highly clustered geographical transmission pattern than chlamydia, and revealed particular LGAs in the greater Western Sydney area where the annual rate increase in gonorrhoea notifications was particularly high among residents (Auburn, Hawkesbury, Penrith and Blacktown). Further investigation is required to understand the higher rates of increase observed in these LGAs.

This study, using routinely collected administrative data, was limited by the lack of detailed data on characteristics to identify specific population groups of interest. Chlamydia and gonorrhoea notifications in New South Wales are notifiable by laboratories only and therefore demographic and risk factor information such as Aboriginal and Torres Strait Islander status, country of birth or sexual exposure is not routinely collected. We found poor recording of Aboriginal and Torres Strait Islander status in the data, which is an area that should be targeted for improvement given the known higher risk of both infections among Aboriginal and Torres Strait Islander peoples.

Given the particular concern around increasing STI rates among MSM, we analysed trends in notifications of any anorectal or oropharyngeal infections of gonorrhoea or chlamydia in males as a proxy measure for notifications among MSM. A steep rise, beginning in 2010 for gonorrhoea notifications and 2011 for chlamydia notifications, suggest that transmission between MSM may account in part for the overall rise in notifications.

Increasing notification rates of chlamydia or gonorrhoea could be explained by a true increase in incidence or by improved detection of cases through higher testing rates. Social marketing campaigns promoting testing may have contributed to increased testing and an associated increase in notifications although no campaign evaluation outcomes are available to assess if this occurred. In 2009, the New South Wales campaign 'Get tested, play safe' was launched and subsequently repeated in 2011. The campaign had a particular

focus on encouraging young people to test.[15] The Commonwealth government also had national campaigns in 2009 and 2010 encouraging people to get tested.[16]

Improved access to sexual health services in the greater Western Sydney area may have also resulted in increased notifications. However, there is no indication of significant local changes over the study period such as the opening of new sexual health clinics, improved general practitioner awareness or markedly changed STI testing guidelines that could explain the steep rise.

Notifications of chlamydia are particularly influenced by testing rates as infections are often asymptomatic. A study that analysed testing-adjusted chlamydia notification rates between 2000 and 2010 found that increased testing drove the increase in notifications in New South Wales over the period and that there was no evidence of a true increase in the prevalence of chlamydia.[17] In contrast, other analyses of Australian data have suggested that chlamydia prevalence modestly increased over a similar period among at-risk populations tested in sentinel clinics.[18,19] While the importance of interpreting notifications in the context of testing data including positivity rates is well recognised, data have not always been available to allow this to occur.

In New South Wales, the routine reporting of laboratory testing data commenced in January 2012 for selected notifiable conditions including chlamydia and gonorrhea.[20] This was an important step towards understanding state-wide notification trends. As a next step, more detailed testing data that would allow analysis of patterns of testing in subpopulations and geographical areas would be of value to enable STI transmission dynamics to be better understood.

Over the study period, nucleic acid amplification tests (NAAT) for gonorrhoea became more widely used to detect infections in non-genitourinary sites.[21–24] NAAT has greater sensitivity than culture,[25,26] and its increasing use may therefore have partly contributed to the overall increase in notifications. Furthermore, as NAAT for gonorrhea and chlamydia is a combined test, incidental findings of 1 infection when testing for the other may also have a role in the overall increase in notifications. However the extent to which this may affect notification rates is unclear.[27]

Finally, it is possible that over the study period there was a change in the profile of the population such that high-risk groups made up a greater proportion of the overall population in the Western Sydney area. However, similar trends across other regions of New South Wales suggest that these results are not explained by a shift in population demographics.[4]

This study suggests that within the greater Western Sydney area there are population groups at greater risk of STIs based on age, sex and place of residence. The apparent clustering of gonorrhoea notifications among residents of particular LGAs requires further investigation. Poor health literacy in relation to STI prevention and treatment among particular community groups, poor health seeking behaviour or inadequate training of local general practitioners may be contributing factors to be explored and addressed.

Surveillance systems should continue to be enhanced to combine testing data with more detailed geographic, demographic and sexual exposure data to obtain a clearer picture of local transmission dynamics. Improved recording of Aboriginal and Torres Strait Islander status is particularly important. This can inform timely health promotion strategies aimed at specific population groups and also support clinical services through targeted outreach programs and engagement with general practitioners.

Improving access to testing is important to diagnose and treat STIs early, especially for MSM of whom less than half currently receive comprehensive testing as recommended by guidelines.[28] In addition to traditional clinic-based services, novel approaches such as outreach initiatives show encouraging results in reaching high-risk populations.[29] Clinic-based and outreach services should continue to explore ways to improve their accessibility and acceptability, particularly by the culturally diverse community groups living in greater Western Sydney.

In conclusion, a better understanding of local STI epidemiology, including testing rates, is an important tool in the planning of clinical sexual health services and in the design of local health promotion interventions adapted to populations at greatest risk.

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Waterparks are high risk for cryptosporidiosis: A case-control study in Victoria, 2015

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Abstract

Background: An increase in notifications of cryptosporidiosis was observed in Victoria between March and April 2015. Cases mostly resided in one metropolitan region and hypothesis-generating interviews identified common exposures to aquatic facilities. We conducted a case-control study to determine exposure source(s) and facilitate control measures.

Methods: Laboratory-confirmed cases of cryptosporidiosis from the region of interest notified between 1 March and 23 April 2015 were included. Controls residing in the same region were recruited from participants in a population health survey and frequency matched (2 per case) by age group. Details of exposure to potential risk factors were collected using a standardised telephone questionnaire for the 14-days prior to illness for cases, and an analogous exposure period for controls. Univariable and multivariable logistic regression were used to determine risk factors associated with illness using STATA SE 13.1.

Results: Thirty cases and 66 controls were included in the study. Half the cases were less than 12 years of age and 62% were female. Illness was most strongly associated with recreational water exposure at any waterpark (adjusted odds ratio (aOR)=73.5; 95% confidence interval (CI):6.74–802), and specifically at Victorian waterparks (aOR=45.6; 95% CI:5.20–399). Cases were linked with attendance at either a waterpark in the region or an adjacent region. As a result of this investigation, hyperchlorination was completed at identified facilities and swim hygiene information distributed.

Conclusion: This study reinforces the potential for recreational water facilities, particularly waterparks, to act as a transmission source of *Cryptosporidium* infections. Continued communication to patrons is required to ensure healthy swimming practice in Victorian aquatic facilities. *Commun Dis Intell* 2017;41(2):E141–E149.

Keywords: cryptosporidiosis, *Cryptosporidium*, case-control study, waterborne disease

Introduction

Cryptosporidiosis is a gastrointestinal illness caused by a species of the parasite *Cryptosporidium*. *Cryptosporidium* infection manifests as a gastrointestinal illness approximately 2 to 14 days (average seven days) after exposure. Symptoms commonly include watery diarrhoea, often in conjunction with abdominal pain, fever, nausea and vomiting. Illness is usually mild and self-limiting, but may be more severe in children and those who are immunocompromised.[1]

In Australia, it is mandatory for doctors and laboratories to notify *Cryptosporidium* infections to health departments. Cryptosporidiosis is the 3rd most commonly notified gastrointestinal infection in Australia, with 2,405 cases reported in 2014 (10.2 cases per 100,000 population).[2]

A common transmission pathway of *Cryptosporidium* infection is via ingestion of drinking and recreational waters contaminated with human or animal faeces.[3] *Cryptosporidium* is commonly transmitted by the waterborne route as oocysts are shed in large numbers in the faeces of infected humans or animals, have a low infective dose, can remain viable for many months and are highly resistant to chlorine disinfection.[4,5] An Australian review of waterborne outbreaks of gastroenteritis between 2001 and 2007 found that 98% (41/42) of outbreaks were associated with *Cryptosporidium* contamination in swimming pools.[6] A 2015 review by Lal et al. of peer reviewed research, outbreak reports and surveillance summaries identified that risk factors for *Cryptosporidium* infection in Australia include exposure to contaminated public swimming pools and contact with other infected persons or livestock.[7]

In March and April 2015, the Victorian Department of Health and Human Services (DHHS) detected an increase in cases of *Cryptosporidium* infection, particularly in 1 of the 3 Melbourne metropolitan regions. Initial hypothesis generating interviews with 12 cases identified attendance at local swimming pools and a waterpark as common exposures.

This case-control study aimed to ascertain risk factors to explain the observed increase in cryptosporidiosis. Specifically, we sought to identify any recreational water exposure sites that may be a source of infection, in order to guide control and prevention strategies to reduce the incidence of *Cryptosporidium* infections.

Methods

Epidemiological investigation

We identified cases of cryptosporidiosis from the Victorian DHHS communicable disease surveillance system. For the case-control study, cases were defined as a person with laboratory-definitive evidence of *Cryptosporidium* spp. infection, who resided in the metropolitan region of interest whose onset of diarrhoea (defined as 3 or more loose stools in a 24-hour period) was between 17 February and 21 March 2015. Cases were excluded from the study if they were unable to be contacted after 6 attempts, were unable to recall the date of onset of their diarrhoea at interview or reported international travel for the entire 14 days prior to the onset of their illness. Controls who resided in the same region as cases were recruited from participants in the DHHS annual population health survey.[8] Controls were recruited after all case interviews had been completed. Two controls were recruited per case and were frequency matched by age group to cases (≤5 years, 6–12 years, 13–19 years, 20–29 years, 30–39 years, and 40–49 years). Controls were randomly allocated a 14-day exposure period, analogous to the exposure date range for all cases. Controls were excluded from interview if they had a gastrointestinal illness during their exposure period or reported international travel for the entire exposure period.

A standardised telephone questionnaire was developed and used to collect exposure information on potential risk factors for *Cryptosporidium* infection in the 14 days prior to the case's illness, or the assigned 14-day exposure period for controls, with reference to a calendar to aid recall. The risk factor information included person-to-person contact (household, childcare or high-risk occupations), drinking water sources, contact with pets, farm animals and manure and consumption of unpasteurised milk or milk products. Specific recreational water exposures explored were: public or private swimming pools; waterparks; public or private spas; home paddling pools; or any natural bodies of water. In addition, the questionnaire sought clinical information for cases. If the case or control was under 18 years of age, the parent, guardian or another person in the household most familiar with their routine was interviewed.

Univariable and multivariable regression analyses were used to determine risk factors significantly associated with illness after accounting for collinearity in exposure classification, with the strength of an association assessed by estimating age-adjusted Mantel-Haenszel odds ratios, 95% confidence intervals and 2-sided Fisher's exact probabilities. Statistical tests for associations were determined to be significant when $P < 0.05$. All data cleaning and analysis were completed using STATA/SE 13.1 (StataCorp, Texas, USA). In order to focus our analysis to identify unknown primary risk factors for illness, due to the high transmissibility of cryptosporidiosis and the ease of person-to-person spread within a household, we deemed a secondary case to be any case who had a household member who was also a confirmed case in this study but had an earlier onset date of more than 1 incubation period. These secondary cases were excluded from any further risk factor analysis.

This study was conducted as a Public Health Investigation under Section 188 of the Victorian *Public Health and Wellbeing Act 2008,* and approval from a Human Research Ethics Committee (HREC) was not required. Contact with the control bank was encompassed by DHHS HREC approval of the population health survey in which they initially participated. Verbal consent was obtained prior to each interview, and if the case or control was under 18 years of age the consent of their parent or guardian was sought.

Environmental investigation

Public aquatic facilities are regulated in Victoria by the *Public Health and Wellbeing Act 2008* and the *Public Health and Wellbeing Regulations 2009.* An aquatic facility outbreak was defined when 2 or more cases of cryptosporidiosis had swum at the same facility during their exposure period and case onset dates were less than 28 days apart. When linked cases were identified, facility information was referred to the DHHS Water Program and to local government environmental health officers for investigation and control activities. Control activities were guided by the DHHS *Cryptosporidiosis Prevention and Outbreak Response Plan*[9] and the *Pool Operators' Handbook*.[10] To ensure any *Cryptosporidium* oocysts present in the aquatic facility were inactivated, hyperchlorination was requested.[11] Water sampling is not incorporated in Victoria's outbreak protocol and response plan due to measurement challenges associated with detection of *Cryptosporidium* oocysts, which are often in low abundance in the aquatic environment.[12]

Results

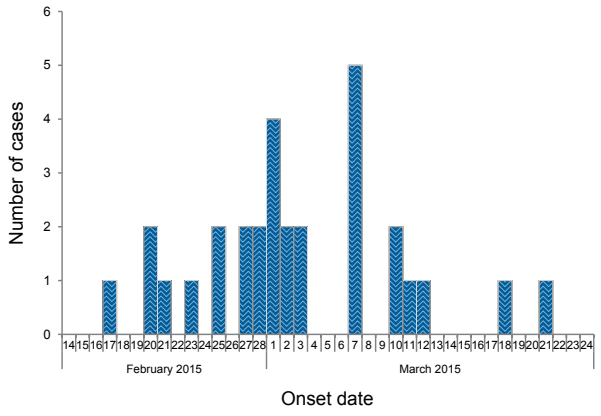
Epidemiological investigation

Descriptive epidemiology

There were 70 notifications of cryptosporidiosis from the region of interest received in the period 1 March to 23 April 2015, which was higher than the 5-year average (2010–2014) for the same

period (55 notifications). Thirty-nine interviews were completed. Case exclusion was due to: non-contact (n=10); privacy reasons (n=11); refusal to participate (n=3); and inability to recall onset date (n=7). An additional 9 cases were excluded post interview as they either had an onset date after 21 March 2015 or were identified as secondary cases. Thirty cases and 66 controls were recruited. There were several peaks of onset of illness for included cases between 17 February and 21 March 2015, indicating the potential for multiple sources of infection (Figure). The median duration of diarrhoea for 27 of 30 cases was 13 days (range: 3–31 days); 3 cases still had diarrhoea at the time of interview. Two children under 8 years of age were hospitalised as a result of their illness. Other than diarrhoea, the most commonly described symptoms for cases were abdominal pain (88.2%), lethargy (82.4%) and nausea (76.5%). The age group and sex distribution of cases and controls are included in Table 1. The median age of cases was 20 years (range: 2–44 years), with 47% of cases aged 12 years or younger. Among adults, cases were predominantly aged 30–39 years. Approximately two-thirds (62%) of cases and controls were female.

Figure: Primary outbreak cases of *Cryptosporidium* infection by date of diarrhoea onset (n=30)



Analytical epidemiology

Of the risk factors for *Cryptosporidium* infection that were explored, illness was significantly associated with recreational water exposure, specifically among those who reported swimming or paddling at a waterpark, and also with prior contact with a household member with a (non-specified) gastrointestinal illness (Table 2). Recreational water exposure in a public or private spa was also significantly associated with illness, although only 13% (4/30) of cases reported this exposure. There was no association between illness and recreational water exposure at public or private swimming pools, paddling pools or natural bodies of water. Consumption of bottled water was also identified as a risk factor for illness.

None of the other food based or animal contact risk factors examined were significantly associated with illness.

In multivariable analysis exposure at a waterpark or a public or private spa remained associated with illness, after adjusting for bottled water consumption and ill household contacts (Table 2). Bottled water consumption remained associated with illness in multivariable models, as did person-to-person transmission through household contacts with prior diarrhoeal illness.

Environmental investigation

Under DHHS criteria, 2 Victorian outdoor waterparks were implicated as exposure sites for 9 of the 11 cases in the current study who indicated they swam at a waterpark. Park A was located within the region (4 linked cases) and Park B in an adjacent region (5 linked cases). An additional 5 cases were identified as having attended Park A in the same period as included cases; however, these were excluded from the study analysis as they were either unable to recall the onset date (n=3) or resided outside of the region of interest (n=2). All cases reported waterpark attendance in their exposure period. More specifically, attendance was focused during a single weekend period in February 2015 at each of the 2 facilities: either 14–15 February (Park A) or 21–22 February (Park B).

Table 1: Age group and sex distribution of cases and controls in a study of cryptosporidiosis in a metropolitan region of Melbourne, February and March 2015

	Age group (years)							Sex
	5 or under	6 to 12	13 to 19	20 to 29	30 to 39	40 to 49	Total	(% female)
Cases	6	8	1	6	6	3	30	62
Controls	14	20	2	9	14	7	66	64

Table 2: Age-adjusted univariable analysis and multivariable logistic regression for significant risk factors associated with *cryptosporidiosis* infection in one metropolitan region of Melbourne, February and March 2015

Risk factor (excluding known secondary cases)	Cases (total = 30)			Control (total = 66)			Univariable			Multivariable		
	Exposed	Total	%	Exposed	Total	%	Age-aOR*	95% CI	*P* value	aOR†	95% CI	*P* value
Other household member with diarrhoeal illness	8	30	27	4	63	6	5.16	1.42–18.7	0.007	12.6	2.13–75.1	0.005
Recreational water exposure (any)	24	30	80	37	66	56	3.18	1.15–8.79	0.023	–	–	–
Waterpark (any)	11	30	37	1	64	2	73.5	6.74–802	<0.0001	–	–	–
Waterpark (Victoria)	10	30	33	1	66	2	45.6	5.20–399	<0.0001	36.9	3.12–435	0.004
Spa (public or private)	4	30	13	1	66	2	9.10	1.01–81.8	0.018	26.4	1.47–472	0.026
Public pool	11	30	37	23	65	35	1.07	0.438–2.56	0.890	–	–	–
Private pool	7	30	23	9	65	14	1.96	0.645–5.95	0.225	–	–	–
Natural bodies of water‡	5	30	17	11	66	17	1.00	0.308–3.25	1.000	–	–	–
Paddling pool	0	30	0	8	64	13	Undefined		0.05	–	–	–
Drank bottled water	24	28	86	35	65	54	5.36	1.60–17.9	0.004	6.31	1.39–28.7	0.017

OR: odds ratio; 95% CI: 95% confidence interval.

* Mantel-Haenszel OR adjusted for matched age-group.

† OR adjusted for all other variables in model, including age-group.

‡ Natural bodies of water include river, lake, dam, bore water or beach.

Local government environmental health staff liaised with the facility manager at Park A and identified deficient filtration systems in several pools, including a shallow play area with water fountains, splash slides and spray areas. Hyperchlorination[11] of all pools at the Park A was conducted to inactivate any *Cryptosporidium* oocysts present in the water, during which time the park was closed to patrons. The local government environment health unit in the region where Park B was located had been advised by members of the public and other council staff of anecdotal cases of gastrointestinal illness in large groups following attendance over a single weekend. As a result, Park B undertook voluntary hyperchlorination prior to DHHS identifying that notified cases were linked to this facility. In addition, DHHS also provided information to staff at both facilities for distribution to patrons about preventative healthy swimming practices, including diarrhoea exclusion periods, personal swim hygiene, use of designated nappy change areas, and avoiding swallowing pool water.[13]

Discussion

The increase in cryptosporidiosis notifications observed in an urban region of Melbourne, Victoria, in February and March 2015 was strongly associated with recreational water exposure, specifically at waterparks. In addition, cases were temporally linked with attendance at 2 waterpark facilities, one of which had evidence of deficiencies in water quality maintenance. This investigation resulted in the execution of immediate control activities at these sites. Internationally, outbreaks of cryptosporidiosis associated with water or splash parks have been reported in the United States of America,[14–17] Canada[18] and the United Kingdom.[19] Our investigation highlights that waterparks also require specific attention for the prevention of cryptosporidiosis outbreaks in Australia.

Cryptosporidium are well suited to transmission in recreational water, and outbreaks can occur in aquatic facilities even if they are well maintained, as swimmers can be exposed to *Cryptosporidium* oocysts during the time it takes for water to be disinfected or filtered.[20] As such, control of transmission relies on both good swimmer hygiene to prevent the introduction of oocysts into recreational waters, and effective pool engineering controls for water filtration and treatment.

The design and nature of use of waterparks, which can differ from standard pool facilities, raise unique considerations for directing prevention and control activities. Waterparks, including those identified in the current investigation, typically include water play areas such as splash zones, fountains and shallow wading pools: features which have been suggested to contribute to the transmission of infection in other studies. While splash activities may lead to increased swallowing of water,[16] increased use of shallow pools by children wearing nappies,[4] sitting on splash features[15,17] and accidental faecal release incidents[14] are also suggested to contribute to *Cryptosporidium* infection in toddler pools at waterparks. Although pools frequented by toddlers are the most plausible source of contamination, a specific wading pool or splash activity was not implicated at either facility in this study.

Responding to an increased number of waterpark-related outbreaks, the Centers for Disease Control and Prevention (CDC) in the United States of America identified that specific patron messaging needed to be designed for water play and interactive areas, to prevent contamination and reduce potential for transmission.[21] It is recommended that similar messaging be developed for use in Australia in addition to existing healthy swimming advice.[13]

Outdoor shallow toddler pools and splash pools present at waterparks inherit special problems associated with their engineering and maintenance. In addition to contamination challenges in toddler pools outlined above, outdoor pool water quality can be compromised through chlorine degradation by UV light and their potential to be polluted by other foreign matter that blows into them.[10] Waterparks can be popular destinations in peak periods. Evidence from this study and other investigations[18,22] suggests that individuals often travel a greater distance to attend these specialised recreational water facilities. As such, high bather densities can further challenge pool maintenance, especially in shallow toddler pools or splash pools where bather pollution can be high for the amount of water involved.[10]

It has been highlighted elsewhere[15] that waterpark operators cannot solely rely on filtration and chlorine disinfection to protect patrons from *Cryptosporidium*. In alignment with other advice, in order to minimise transmission of *Cryptosporidium* infection, we also recommend that separate maintenance systems should be applied to pools with high infant bather loads.[10,14] These may encompass the use of separate backwash and filtration systems, regular filter maintenance and hyperchlorination procedures, and more frequent pool content circulation.

In this study, almost half of the cases were aged 12 years or younger and another third were adults aged 30–39 years. This pattern is consistent with the bimodal age distribution of *Cryptosporidium*

notification rates in Victoria observed between 2001 and 2009.[23] While children are suggested to be more susceptible to parasitic infections,[20,24] other researchers have noted that this distribution may also reflect notification bias, with medical care more likely to be sought for children.[25] Additionally, increased swallowing of water when swimming or playing in shallow pool areas and splash zones (such as those identified in the implicated waterparks) may mean that children have a greater risk of being infected.[16,20,25]

Interestingly, after adjusting for other confounding factors, consumption of bottled water remained significantly associated with illness. This finding is in contrast to those of a study of a large statewide outbreak of cryptosporidiosis in New South Wales in 1997 to 1998, which found that cases were less likely to report drinking bottled water.[26] It is feasible that bottled water is a potential source of cryptosporidiosis as other international studies have identified *Cryptosporidium* oocysts in commercially bottled water[27,28] and a case-control study conducted in South Australia in 1993 identified consumption of bottled spring water as risk factor for illness.[29]

In the current study, information wasn't specifically collected on brands or amounts of bottled water drunk by cases and controls to enable further examination of this association. Furthermore, the association of illness with bottled water consumption in this study may have limited plausibility given that we know from the department's recent food frequency survey (Marion Easton, personal communication) that around 33% of Victorians surveyed, regardless of the time of year, drank bottled water. It is likely then, that if there was a true association between drinking bottled water with illness it would have been expected that the impact of this would have been observed Victoria-wide, rather than limited to a specific region. It is also possible that this association may be a result of uncontrolled confounding, as several studies have described sociodemographic factors that are associated with bottled water use through their influence on consumer perception of water risk and quality.[30]

To a lesser extent, the odds of illness were also increased among those cases who used public or private spas and person-to-person transmission in a household environment. Although not significantly associated with the rise in cases in the region, interviews with cases also highlighted the diversity of risk factors associated with sporadic *Cryptosporidium* infection, which otherwise contributed to background rates in this region and would have also been captured in the study population.

These findings need to be considered within the limitations of this study. As controls were not contacted until after all cases were interviewed, and were asked about activities over a similar time period as cases, this may have resulted in a systematic difference in recall in controls. Furthermore, case exclusions may have meant that not all risk factors for cryptosporidiosis were fully elucidated.

Outbreaks of cryptosporidiosis can be difficult to identify, as it can often be a mild, self-limiting illness for which medical attention is not sought.[1] Despite strong epidemiological evidence, this study did not include a microbiological investigation for evidence of water contamination at the implicated facilities. It may be that species identification and molecular sub-typing of *Cryptosporidium* may have strengthened links between cases and exposure sites and also provided further evidence for the diversity of sources of infection; however, a number of studies exist that provide a rich body of evidence in this area.[31–34]

While sub-typing may have assisted in the attribution of causality, *Cryptosporidium* species differentiation is not routinely performed in Australian diagnostic laboratories[35] and cannot currently be used for retrospective public health investigations in Victoria. An enhanced environmental investigation at each facility may have served to understand individual and overall pool treatment and backwash systems and identify specific deficits in these, as well as the incidence and management of faecal incidents.

Conclusion

This study has shown that recreational water facilities, including waterparks, remain high risk sites for the acquisition of *Cryptosporidium* infection. This assertion is further strengthened by the identification of cases linked to attendance at 2 separate waterparks during this investigation.

The maintenance of water quality at aquatic facilities requires vigilance by both operators and the public. This study is an important reminder of the need for preventive risk management through healthy swimming practices and ensuring the adequate performance of treatment processes within aquatic facilities. Specific recommendations include the development of healthy splash advice specific for waterpark patrons, and the use of separate backwash and filtration systems for high risk pools and activities.

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Annual report

Influenza viruses received and tested by the Melbourne WHO Collaborating Centre for Reference and Research on Influenza annual report, 2015

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Abstract

As part of its role in the World Health Organization's (WHO) Global Influenza Surveillance and Response System, the WHO Collaborating Centre for Reference and Research on Influenza in Melbourne received a total of 5,557 influenza positive samples during 2015. Viruses were analysed for their antigenic, genetic and antiviral susceptibility properties. In 2015, influenza B viruses predominated over influenza A(H1)pdm09 and A(H3) viruses, accounting for a total of 58% of all viruses analysed. The vast majority of A(H1) pdm09, A(H3) and influenza B viruses analysed at the Centre were found to be antigenically similar to the respective WHO recommended vaccine strains for the Southern Hemisphere in 2015. However, phylogenetic analysis of a selection of viruses indicated that the majority of circulating A(H3) viruses were genetically distinct from the WHO recommended strain for 2015, resulting in an update to the recommended vaccine strain for the Southern Hemisphere for 2016. With an increasing predominance of B/Victoria lineage viruses over B/Yamagata lineage viruses through the course of 2015, WHO also updated the recommended influenza B strain in the trivalent influenza vaccine for 2016. Of more than 3,300 samples tested for resistance to the neuraminidase inhibitors oseltamivir and zanamivir, only 1 A(H1) pdm09 virus showed highly reduced inhibition by oseltamivir. The Centre undertook primary isolation of candidate vaccine viruses directly into eggs, and in 2015 a total of 45 viruses were successfully isolated in eggs. *Commun Dis Intell* 2017;41(2):E150–E160.

Keywords: annual report; influenza

Introduction

The WHO Collaborating Centre for Reference and Research on Influenza in Melbourne is part of the World Health Organization Global Influenza Surveillance and Response System (WHO GISRS). GISRS is a worldwide network of laboratories that was established in 1952 to monitor changes in influenza viruses circulating in the human population with the aim of reducing its impact through the use of vaccines and antiviral medications. The Centre in Melbourne is one of 5 such Collaborating Centres (the others being in Atlanta, Beijing, London and Tokyo) that monitor the antigenic and genetic changes in circulating human influenza viruses, and makes biannual recommendations on which influenza strains should be included in the influenza vaccine for the upcoming winter season in either the Northern or Southern Hemisphere. This report summarises the results of virological surveillance activities undertaken at the Centre in Melbourne during 2015.

Two types of influenza cause significant disease in humans: types A and B. Influenza A viruses are further classified into subtypes, based on their haemagglutinin (H) surface protein. Globally, there are currently 2 subtypes circulating in human populations: A(H1N1)pdm09 and A(H3N2). Here the N refers to the type of neuraminidase surface protein, of which there are also several types. Influenza B viruses are not classified into subtypes; however, there are 2 lineages, which currently co-circulate globally, B/Victoria/2/87 (B/Victoria) and B/Yamagata/16/88 (B/Yamagata). In addition, each year influenza C viruses are detected from humans, but these viruses tend not to cause severe disease and are not a major focus of surveillance.

Methods

Virus isolation

All A(H1)pdm09 and all influenza B viral isolates received at the Centre were re-passaged in cell culture (Madin-Darby Canine Kidney (MDCK) cells), whilst all A(H3) viral isolates were re-passaged in MDCK-SIAT1 cells.[1] Virus isolation was also attempted from a selection of original clinical specimens received. In addition, influenza positive original clinical samples were directly inoculated into eggs as potential candidate vaccine viruses.

Antigenic analysis

The antigenic properties of influenza viral isolates were analysed using the haemagglutination inhibition (HI) assay as previously described.[2] 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 2015, results were reported by reference to the A/California/7/2009 (H1N1pdm09)-like, A/Switzerland/9715293/2013 (H3N2)-like, B/Phuket/3073/2013-like (Yamagata lineage), and B/Brisbane/60/2008-like (Victoria lineage) viruses that were recommended for the 2015 influenza vaccine. In recent years, including 2015, HI assays involving A(H3) viruses have been performed in the presence of oseltamivir carboxylate in order to reduce non-specific binding of the N protein.[3]

Genetic analysis

A subset of all influenza viruses analysed at the Centre underwent genetic analysis by sequencing of viral RNA genes; usually the H and N genes as well as the matrix gene for influenza A viruses and non-structural protein genes for influenza B viruses. In addition, the full genomes (all 8 gene segments) of a smaller subset of viruses were sequenced.

For sequencing, RNA was extracted from isolates or original clinical specimens using either manual QIAGEN QIAamp spin columns method or the automated QIAGEN QIAXtractor robot, followed by reverse transcription polymerase chain reaction (PCR) using the BIOLINE MyTaq one step reverse transcription PCR kit according to the manufacturer's recommendations with gene specific primers (primer sequences available on request). Conventional Sanger sequencing was carried out on PCR product using an Applied Biosystems 3500 XL sequencer. Sequence assembly was performed using the Seqman Pro Module of DNASTAR Lasergene version 13 software (DNASTAR, Madison, WI, USA). Next generation sequencing (NGS) was performed on a selection of viruses using an Applied Biosystems Ion Torrent Personal Genome Machine System according to the manufacturer's recommendations. These sequences were analysed using a proprietary FluAnalysis pipeline. Phylogenetic analysis was performed using Geneious 9.0.4 software (Biomatters Ltd, Auckland, New Zealand) and FigTree v1.3.1.

Antiviral drug resistance testing

As there is potential for influenza viruses to develop resistance to antiviral drugs, 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 with values obtained with sensitive reference viruses of the same subtype or lineage. NAI assays were performed as previously described[4] with the incorporation of a robotic platform by TECAN EVO200 and Infinite 200Pro for liquid handling and plate reading (Tecan Australia). 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 neuraminidase inhibition 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 NAIs. 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 neuraminidase mutation in B viruses.[5]

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,[6] except for the following modifications. First, the viruses were initially inoculated into the amniotic cavity only, and once growth was established the isolates were then further passaged in the allantoic cavity. This was followed by harvest only from either the amniotic or allantoic cavity, depending on site of inoculation. Egg incubation conditions also differed slightly with A(H1)pdm09 and A(H3) viruses incubated at 35°C for 3 days, and influenza B viruses incubated

at 33°C for three days. These isolates were then analysed by haemagglutination assay, HI assay, real time (RT)-PCR and genetic sequencing.

Results

During 2015, the Centre received 5,557 clinical specimens and/or virus isolates from 34 laboratories in 14 countries (Figure 1). As in previous years, most samples were submitted by laboratories in the Asia–Pacific region, including Australian laboratories[7] and were received during the Southern Hemisphere influenza season. Figure 2 shows the temporal distribution of samples sent to the Centre by type or subtype and lineage.

Isolation was attempted for 4,911 (88%) of the samples received and was successful in 3,360 cases (68%). Of these, 2,815 were characterised by HI assay in comparison with the 2015 vaccine strains (Table). In addition, 229 samples were characterised by RT-PCR to determine their type, subtype or lineage. Sanger sequencing and NGS techniques were used to sequence the haemagglutinin genes of 957 viruses. The full genomes of 39 viruses were sequenced using either Sanger sequencing or NGS. Of the samples for which results could be obtained via antigenic or genetic analysis, (n=3,503), influenza B viruses predominated, comprising 58% of viruses analysed (30% B/Yamagata and 28% B/Victoria). The remaining portion of viruses were mostly A(H3) (31% of total number of viruses), followed by A(H1)pdm09 (11%). There was 1 sample with mixed A(H3)/B viruses.

Figure 2: Number of samples received at the WHO Collaborating Centre for Reference and Research on Influenza, 2015, by week of sample collection

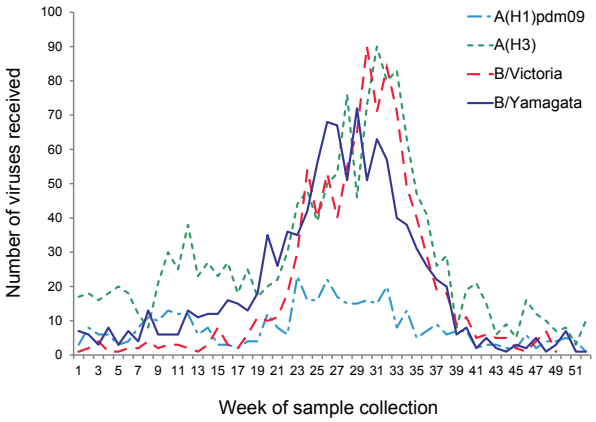
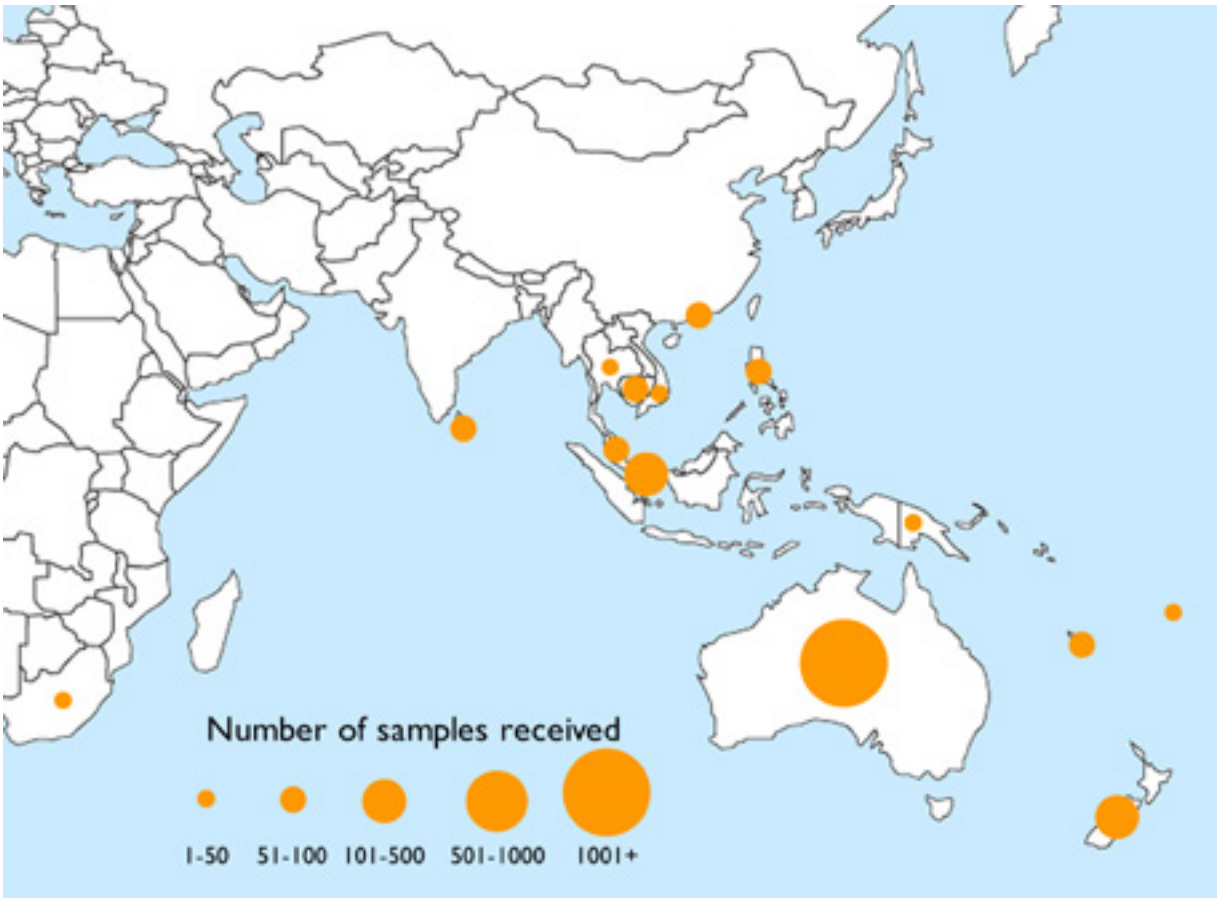


Figure 1: Geographic spread of influenza laboratories sending viruses to the WHO Collaborating Centre for Reference and Research on Influenza during 2015

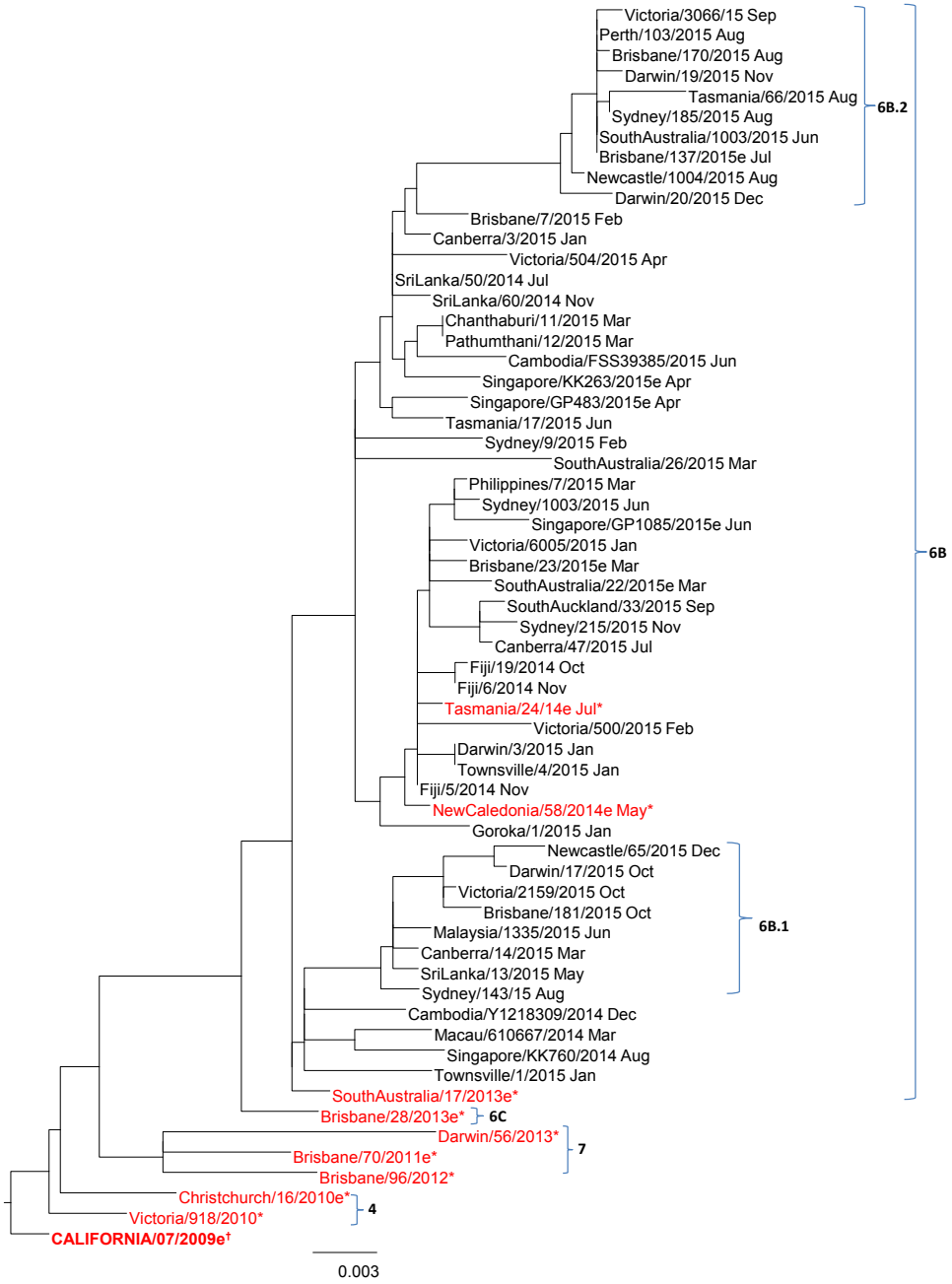


A(H1N1)pdm09

Of the 395 A(H1)pdm09 isolates analysed by HI assay using ferret antisera in 2015, the majority (98.7%) were antigenically similar to the vaccine reference strain A/California/7/2009 (Table).

Sequencing and phylogenetic analysis of haemagglutinin genes from 67 viruses showed that A(H1)pdm09 viruses sent to the Centre during 2015 fell mainly into the 6B clade with a smaller number of viruses in subclades 6B.1 and 6B.2 (Figure 3). No antigenic differences were detectable between viruses from the 6B, 6B.1 or 6B.2 genetic clades in ferret antisera HI assays and the majority of viruses reacted in a similar manner to the reference and 2015 vaccine virus A/California/7/2009.

Figure 3: Phylogenetic tree of representative haemagglutinin genes of A(H1)pdm09 viruses received by the WHO Collaborating Centre for Reference and Research on Influenza, 2015



* Reference virus is indicated in red text.

† 2015 Southern Hemisphere vaccine strain is presented in capital letters.

e: egg isolate

Scale bar represents 0.3% nucleotide sequence difference between viruses.

} Braces indicate clades

Twenty-three viruses were inoculated into eggs for vaccine candidate strain isolation. Of these, 9 (39%) were successfully isolated, including viruses from each of the 2 emerging subclades (6B.1 and 6B.2).

Of 380 A(H1)pdm09 viruses tested 1 exhibited highly reduced inhibition by oseltamivir. This virus was from Australia and had the H275Y mutation in the N gene, which is known to reduce inhibition by oseltamivir. No A(H1)pdm09 viruses received during 2015 showed highly reduced inhibition to zanamivir.

A(H3N2)

Antigenic analysis of 651 A(H3) subtype isolates showed that only 1.7% were low reactors to the cell-propagated reference strain A/Switzerland/9715293/2013 (Table). However, 47% of viruses were low reactors to the egg-propagated strain A/Switzerland/9715293/2013 (data not shown). An additional 232 A(H3) viruses were inoculated and/or isolated by cell culture but did not reach sufficient titres for antigenic analysis, whilst a further 503 were successfully isolated but did not reach sufficient titres when tested by HI assay in the presence of oseltamivir.

A total of 673 haemagglutinin genes from A(H3) viruses were sequenced. Phylogenetic analysis indicated that the majority of circulating viruses fell into clade 3C.2a, which is a genetically different clade to the 2015 vaccine strain A/Switzerland/9715293/2013 (clade 3C.3a) and is represented by the A/Hong Kong/4801/2014 virus (Figure 4). A small number of H3 viruses fell into the 3C.3b clade.

Fifty-four viruses were inoculated into eggs, of which 21 (39%) grew successfully. These viruses included at least 17 viruses from clade 3C.2a and 4 viruses from clade 3C.3b.

None of the 1,339 A(H3) viruses tested had highly reduced inhibition by either oseltamivir or zanamivir.

Influenza B

Similar proportions of viruses from both B lineages (B/Victoria and B/Yamagata) were received at the Centre during 2015. A total of 1,769 influenza B viruses were characterised by HI assay. All but 3 of the 837 B/Victoria viruses received and analysed were similar to B/Brisbane/60/2008 (Table). Only 4 of the 932 B/Yamagata lineage viruses analysed were low reactors to the B component of the 2015 vaccine, B/Phuket/3073/2013 vaccine strain (these comparisons were made to ferret antisera generated to the MDCK-isolated virus induced infections).

Sequencing was performed on 217 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, which was represented by the 2015 vaccine strain B/Phuket/3073/2013 as well as B/Wisconsin/1/2010 (the 2013 Southern Hemisphere influenza vaccine strain) (Figure 6).

Egg isolation was attempted for 26 B/Victoria and 15 B/Yamagata viruses, with 8 (31%) B/Victoria viruses and 7 (47%) B/Yamagata viruses successfully isolated in eggs. At least 1 representative from the major clades of both B lineages was among the successful isolates.

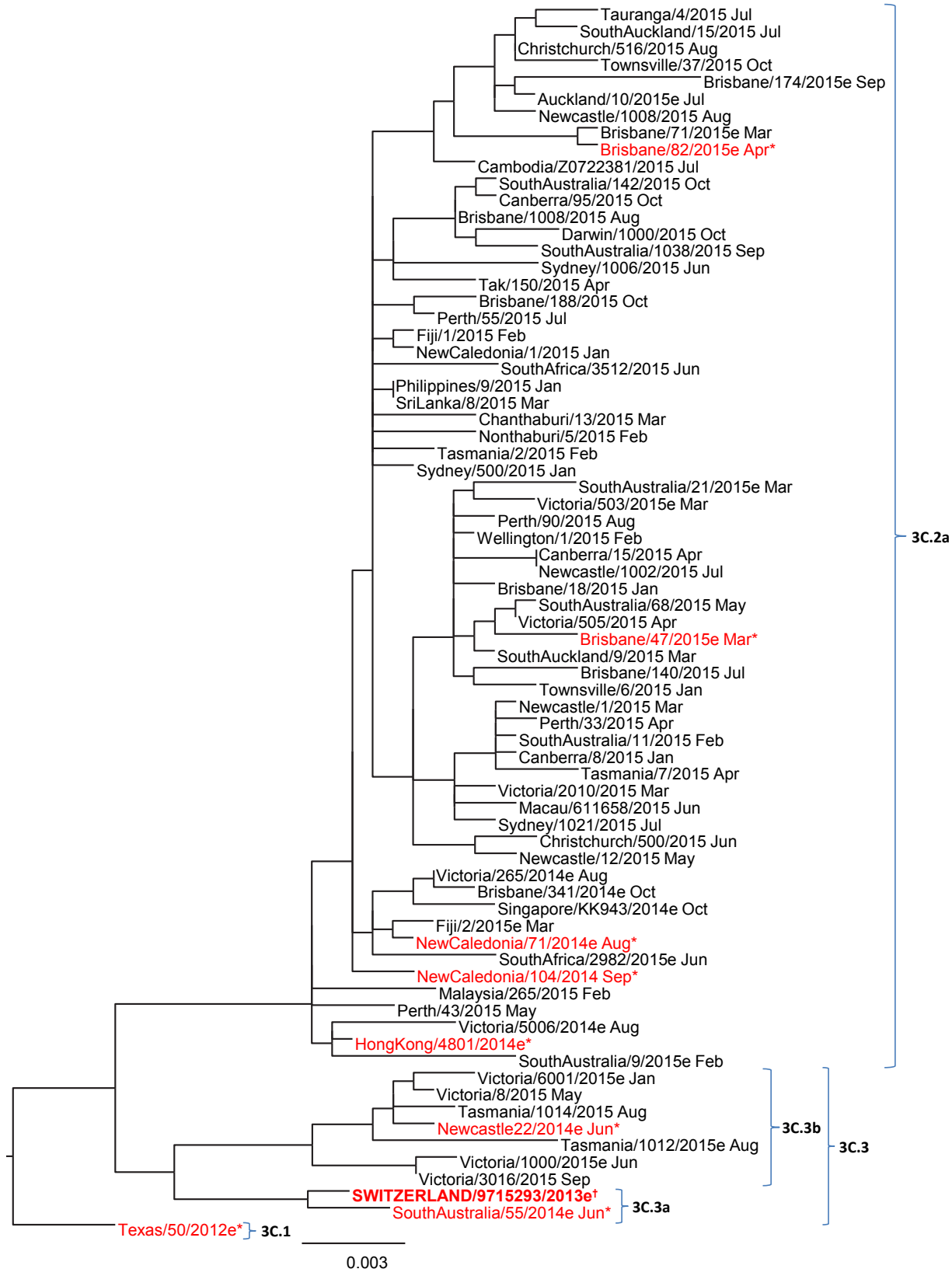
Of 780 B/Victoria and 825 B/Yamagata viruses tested, none displayed highly reduced inhibition by either oseltamivir or zanamivir.

Table: Antigenic analysis of viruses received by the WHO Collaborating Centre for Reference and Research on Influenza, 2015, by country of origin

Region	A(H1N1)pdm09 reference strain: A/California/7/2009 (cell)		A(H3N2)* reference strain: A/Switzerland/975193/2013 (cell)		B/Victoria reference strain: B/Brisbane/60/2008 (cell)		B/Yamagata reference strain: B/Phuket/3073/2013 (cell)	
	Like	Low reactor (%)	Like	Low reactor (%)	Like	Low reactor (%)	Like	Low reactor (%)
Australasia	224	4	459	9	809	3	766	2
Pacific	18	0	7	0	12	0	51	0
South East Asia	102	1	133	2	13	0	87	2
East Asia	14	0	25	0	0	0	18	0
South Asia	25	0	12	0	0	0	2	0
Africa	7	0	4	0	0	0	4	0
Total	390	5 (1.3%)	640	11 (1.7%)	834	3 (0.4%)	928	4 (0.4%)

* Note that many A(H3N2) virus isolates that were obtained could not be analysed by haemagglutination inhibition assay due to low haemagglutination assay titre in the presence of oseltamivir.

Figure 4: Phylogenetic tree of representative haemagglutinin genes of A(H3) viruses received by the Centre during 2015



* Reference virus is indicated in red text.

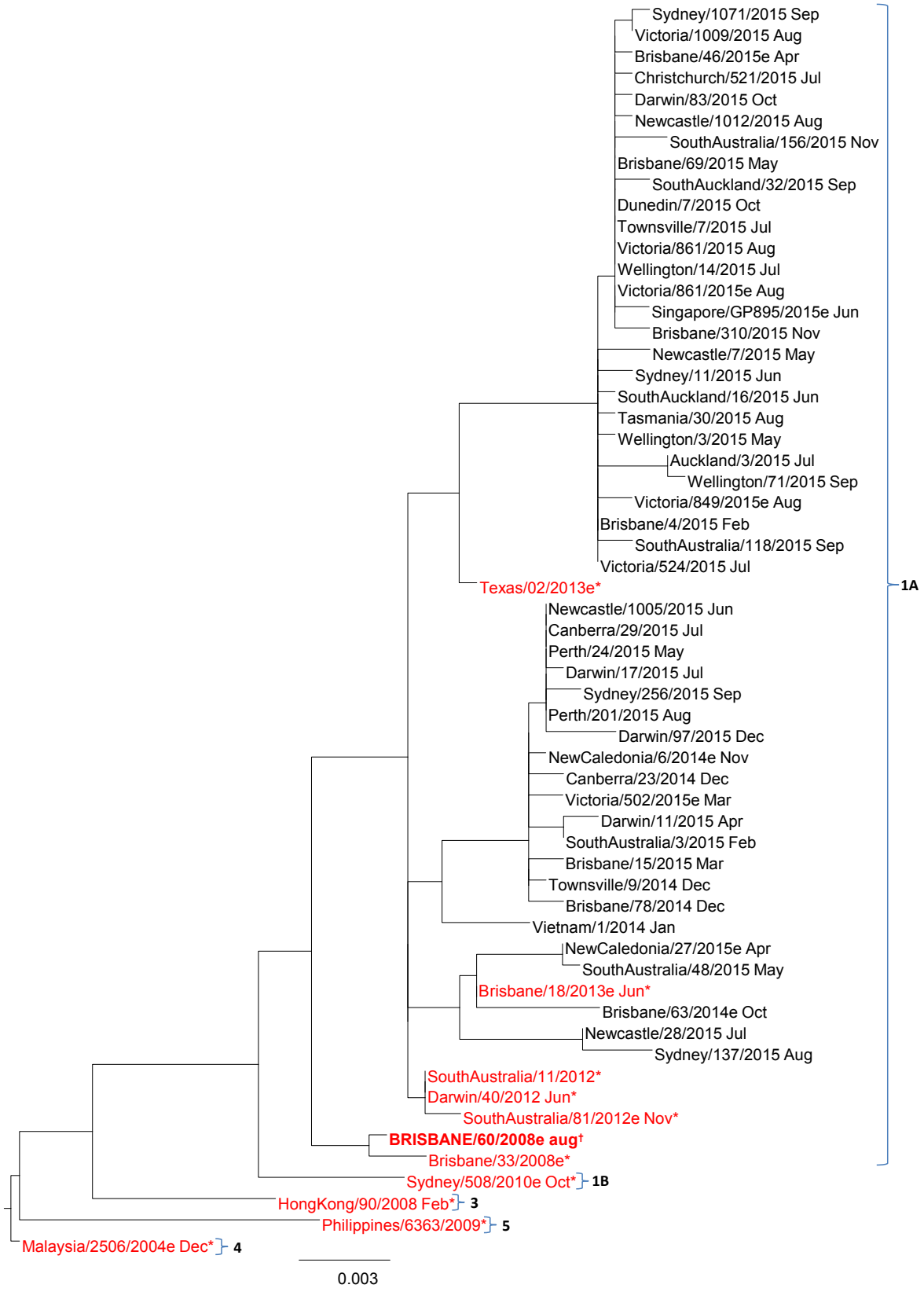
† 2015 Southern Hemisphere vaccine strain is presented in capital letters.

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 received by the WHO Collaborating Centre for Reference and Research on Influenza, 2015



* Reference virus is indicated in red text.

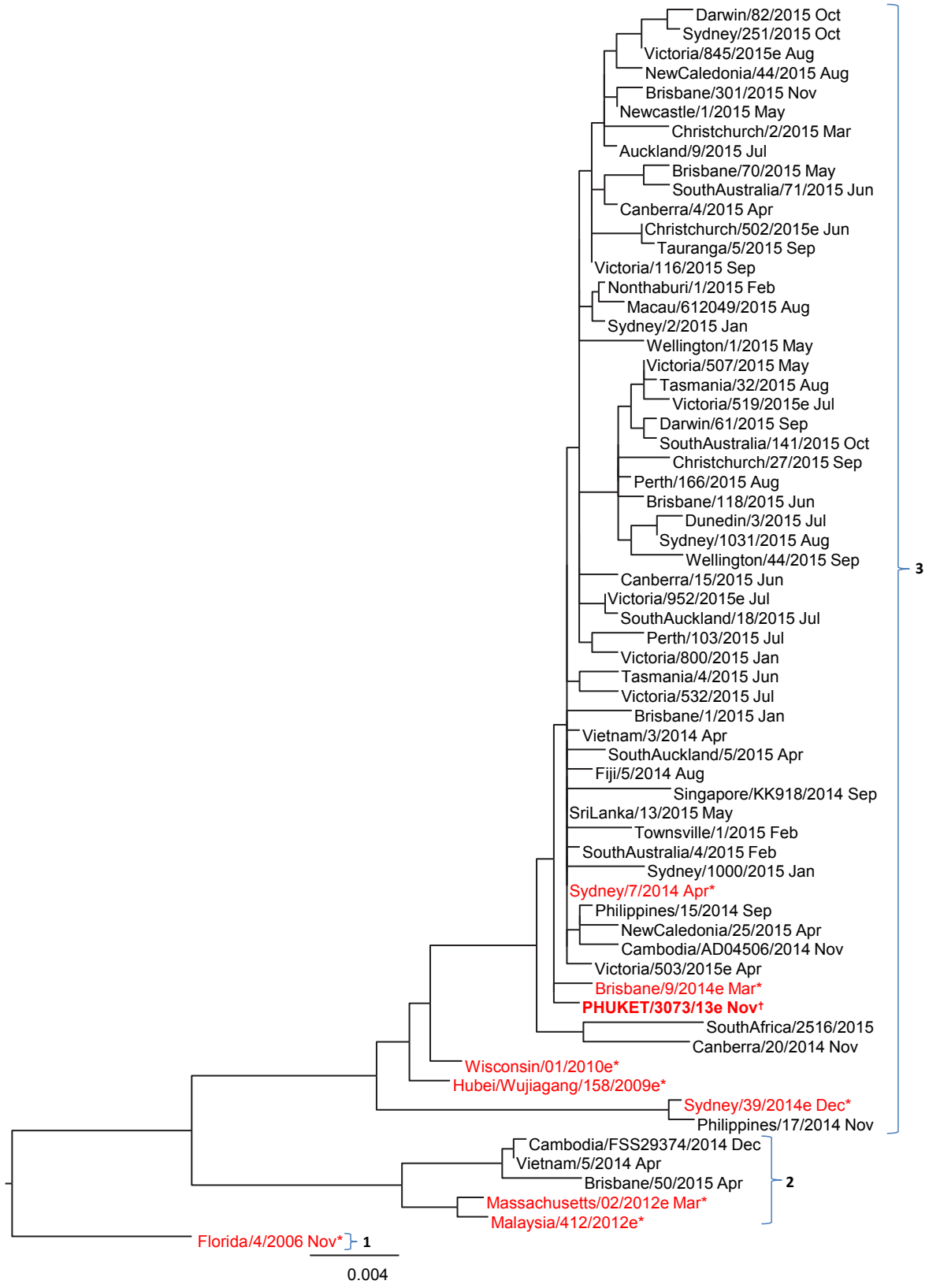
† 2015 Southern Hemisphere vaccine strain is presented in capital letters.

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 received by the WHO Collaborating Centre for Reference and Research on Influenza, 2015



* Reference virus is indicated in red text.

† 2015 Southern Hemisphere vaccine strain is presented in capital letters.

e: egg isolate

Scale bar represents 0.4% nucleotide sequence difference between viruses

} Braces indicate clades

Discussion

As in previous years,[7] the vast majority of samples received at the Centre in 2015 were from Australia. Data from the National Notifiable Diseases Surveillance System indicated that in 2015, Australia reported the highest number of notifications of laboratory-confirmed cases (n=100,584)[8] on record, which was at least partly due to increased influenza testing.[9] This was also reflected in the large quantity of samples received at the Centre in 2015, the greatest annual number since 2009, the year of the A(H1N1)pdm09 pandemic, when 6,435 samples were received. In addition, a total of 45 candidate vaccine viruses were isolated in eggs during 2015, the largest number and highest rate of successful isolations since 2010.

Data from national influenza centres indicated that influenza B predominated over influenza A viruses in Australia during 2015,[8] which was also reflected in the viruses analysed by the Centre. When considering only samples received from Australia and for which the lineage could be confirmed, there were slightly more B/Victoria viruses than B/Yamagata viruses (52%–48%). However, when samples submitted to the Centre from all countries were included, there were a larger number of B/Yamagata viruses (53%) compared with B/Victoria viruses (47%). This can be attributed to more B/Yamagata viruses being received from all non-Australian countries, especially New Caledonia, New Zealand and Singapore; this is also reflected in a larger number of B/Yamagata infections than B/Victoria infections recorded by the New Zealand surveillance system during 2015.[10]

In Australia, there was a rapid rise in the number of B/Victoria lineage viruses detected from May onwards.[11] The predominance of influenza B over influenza A viruses in any given year occurs relatively infrequently at a rate of approximately once a decade.[12] The last year in which this occurred in Australia was 2008, and a similar switch in predominance from B/Yamagata to B/Victoria viruses was also observed in that year.[13] The reasons for this change between the 2 lineages within the season are not clear, but may be due to subtle phylogenetic differences between the 2 lineages, differential population susceptibility or a combination of these factors.[13] The change in the relative predominance of B lineage viruses from B/Yamagata to B/Victoria during 2015 was also reflected in the change in recommended B strain for the 2016 Southern Hemisphere trivalent influenza vaccine (TIV) to the B/Victoria-lineage reference virus B/Brisbane/60/2008.

Almost all B/Yamagata lineage viruses tested were antigenically similar to the 2015 vaccine virus B/Phuket/3073/2013 and this was reflected in the overall vaccine effectiveness (VE) estimates for Australia (VE 71%, 95% CI: 57–80).[14] Similarly, the majority of B/Victoria lineage viruses tested were antigenically similar to the reference virus B/Brisbane/60/2008. Lower VE estimates were reported for B/Victoria lineage viruses (VE 42%, 95% CI: 13–61) in Australia in 2015,[14] however this is unsurprising as these estimates were calculated for the trivalent influenza vaccine (TIV), which included only a B/Yamagata lineage virus. Vaccine effectiveness against both influenza B lineages may be improved through the use of the quadrivalent influenza vaccine (QIV) which was introduced in Australia in 2015, but not widely used as it was not included in the National Immunisation Program and only available for private purchase.

Despite the predominance of influenza B during 2015, influenza A(H3N2) predominated in Tasmania, and co-circulation of A(H3N2) and influenza B was observed in Western Australia.[11] The majority of circulating A(H3) viruses were antigenically similar to the cell derived A/Switzerland/9715293/2013 reference strain in the vaccine. However, more than half of the viruses analysed at the Centre were low reactors to the egg derived A/Switzerland/9715293/2013 reference strain. The difficulties in detecting antigenic changes in A(H3) subtype viruses using the HI assay reported in recent years[7,15] continue to present an ongoing challenge for surveillance. This factor and poor viral growth reduced the number of A(H3) viruses received during 2015 that could be analysed by the HI assay by 44%. The Centre is continuing to work on the development of other assays (e.g. virus neutralisation) to characterise the antigenicity of recent A(H3) viruses, however at this time such assays remain time- and labour-intensive and would most likely complement rather than replace the HI assay. In light of this, genetic analysis remains an important tool for detecting both minor and major changes in A(H3) viruses.

Genetic data from the Centre indicated that many of the A(H3) viruses in 2015 fell into clade 3C.2a. Viruses from this clade have been circulating in Australia since 2014 and are somewhat antigenically distinct to the A/Switzerland/9715293/2013 reference strain in the 2015 Southern Hemisphere vaccine, which lies in clade 3C.3a. Phylogenetic data from the Northern Hemisphere suggested that the A(H3) component of the 2015 TIV may not be ideally matched and VE estimates for Australia were indeed low to moderate for A(H3N2) (VE 44%; 95% CI: 21-60).[14] In the WHO recommended vaccine for the southern hemisphere TIV/QIV in 2016, the A(H3) vaccine virus was updated from A/Switzerland/9715293/2013 to A/Hong Kong/4801/2014, which lies in clade 3C.2a.

Finally, antigenic and genetic data for A(H1)pdm09 viruses analysed at the Centre in 2015 indicated an overall good match with the recommended vaccine strain A/California/7/2009. Whilst phylogenetic analysis indicated the emergence of 2 distinct subclades. These changes did not affect the antigenic behaviour of the viruses in HI assays using ferret antisera. As such, A/California/7/2009 was recommended for inclusion again in the TIV/QIV for 2016. This was also reflected in the Australian vaccine effectiveness estimates for A(H1)pdm09 viruses in 2015 (VE 79%, 95% CI: 33–93),[14] as well as interim vaccine effectiveness estimates from the northern hemisphere for the 2015–2016 season that indicated that vaccines containing A/California/7/2009 were also effective against viruses in the emerging 6B.1 clade (VE 64% (95% CI: 44%–77%)).[16,17]

With continual change and evolution in influenza viruses and the absence of a universal vaccine, there remains a need for ongoing influenza surveillance for the foreseeable future. In light of this, the work performed by the Centre in Melbourne is crucial to the efforts of the global surveillance community to ensure that viruses recommended for the influenza vaccine remain updated and as closely matched to circulating viruses as possible.

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Australian National Enterovirus Reference Laboratory annual report, 2014

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Abstract

Following the World Health Organization (WHO) recommendation, Australia conducts surveillance for cases of acute flaccid paralysis (AFP) in children less than 15 years of age as the main method to monitor its polio-free status. Cases of AFP in children are notified to the Australian Paediatric Surveillance Unit or the Paediatric Active Enhanced Disease Surveillance System and faecal specimens are referred for virological investigation to the National Enterovirus Reference Laboratory. In 2014, no cases of poliomyelitis were reported from clinical surveillance and Australia reported 1.4 non-polio AFP cases per 100,000 children, meeting the WHO performance criterion for a sensitive surveillance system. Non-polio enteroviruses can also be associated with AFP and enterovirus A71 and echovirus types 6 and 7 were identified from clinical specimens from cases of AFP. Globally, 359 cases of polio were reported in 2014, with the 3 endemic countries, Afghanistan, Nigeria and Pakistan, accounting for 95% of the cases. In May 2014, the WHO declared the international spread of wild poliovirus to be a public health emergency of international concern and has since maintained recommendations for polio vaccination of travellers from countries reporting cases of wild polio. *Commun Dis Intell* 2017;41(2):E161–E169.

Keywords: poliovirus, acute flaccid paralysis, surveillance, enterovirus, poliomyelitis, eradication, vaccination

Introduction

Australia has established clinical and virological surveillance schemes to monitor its polio-free status. The clinical surveillance follows the World Health Organization (WHO) recommendation of investigating cases of acute flaccid paralysis (AFP) in children less than 15 years of age as an age group at high risk of poliovirus infection. AFP cases are ascertained either by clinicians notifying the Australian Paediatric Surveillance Unit (APSU) via a monthly report card or through the Paediatric Active Enhanced Disease Surveillance System (PAEDS) at 5 sentinel tertiary paediatric hospitals.[1–3] The WHO recommends that 2 faecal specimens be collected at least 24 hours apart and within 14 days of the onset of paralysis from cases of AFP for virological investigation to exclude poliovirus as the causative agent. It is a requirement of the WHO polio eradication program that the specimens are tested in a WHO accredited laboratory, which for Australia is the National Enterovirus Reference Laboratory (NERL) at the Victorian Infectious Diseases Reference Laboratory (VIDRL). The clinical and laboratory data from AFP cases in children are reviewed by the Polio Expert Panel (PEP) and reported to the WHO as evidence of Australia's continued polio-free status.

Clinical specimen and environmental surveillance programs were established as virological surveillance for poliovirus, to complement the clinical surveillance program focussed on AFP cases in children. Enteroviruses other than poliovirus have been associated with AFP and poliovirus infection may manifest clinically without paralysis, such as meningitis. The Enterovirus Reference Laboratory Network of Australia (ERLNA) involves public diagnostic virology laboratories reporting enterovirus typing results from clinical specimens to exclude poliovirus involvement and to establish the epidemiology of non-polio enteroviruses (NPEVs) in Australia. Most poliovirus infections are asymptomatic with the virus shed for weeks in the faeces of infected persons. WHO supports the testing of environmental or raw sewage samples as a means of detecting the presence of wild poliovirus in polio-free countries. In 2014, the testing of environmental samples commenced at a sentinel site in metropolitan Melbourne.

The number of wild polio cases worldwide decreased from 416 in 2013 to 359 in 2014.[4] Pakistan reported 306 of the cases and has become the major source of wild poliovirus transmission, this also being the source of cases in neighbouring Afghanistan. Nigeria is the 3rd remaining endemic country with only 6 wild polio cases reported there in 2014, the lowest number since the global polio eradication program started in 1988. Only wild poliovirus serotype 1 was detected in 2014, with the last report of wild poliovirus type 3 being in Nigeria in November 2012 and of wild poliovirus type 2 being in India in 1999.[4,5] This latter achievement led to the removal of the Sabin 2 serotype from oral polio vaccine (a live vaccine) along with laboratory containment of this serotype, involving restricted access at a limited number of facilities worldwide in 2016.[6,7] All 3 serotypes will still be incorporated in the inactivated polio vaccine. In May 2014, the WHO Director-General declared

the international spread of wild poliovirus in the Northern Hemisphere low season to be a Public Health Emergency of International Concern. The situation has been assessed every 3 months since then and countries known to be exporting wild poliovirus must ensure all residents and long-term visitors are vaccinated between 4 weeks and 12 months prior to international travel.[8,9] The Australian Government updated the Poliomyelitis Outbreak Response Plan in December 2014. The plan will be activated in the event of a polio importation.[10]

This report summarises the polio surveillance program in Australia for 2014; encompassing clinical surveillance for AFP cases in children and virological surveillance for poliovirus.

Methods

Acute flaccid paralysis surveillance

Paediatricians reviewing a patient less than 15 years of age presenting with AFP, or clinicians reviewing a patient of any age with suspected poliomyelitis, are requested to notify the NERL (telephone 03-9342 9607, email enterovirus@mh.org.au). Paediatricians also notify the AFP case to the APSU (http://www.apsu.org.au/) via a monthly report card. Upon receipt of the notification, the AFP National Surveillance Co-ordinator based at VIDRL forwards a clinical questionnaire for the clinician to complete. Alternatively, AFP cases are ascertained by PAEDS nursing staff from medical records and are enrolled in the surveillance program with parental or guardian consent.

According to the WHO surveillance criterion, to be classified as adequate, 2 faecal specimens must be collected more than 24 hours apart due to intermittent virus shedding, and within 14 days of the onset of paralysis, while the virus titre remains high. The faecal specimens are tested free of charge by the NERL.

The PEP, a subcommittee of the Communicable Diseases Network Australia, reviews the clinical and laboratory data for all notified cases of AFP, irrespective of whether they are an eligible or ineligible case. An eligible case is an Australian child less than 15 years of age with AFP (including Guillain-Barré syndrome and transverse myelitis) or an Australian of any age with suspected polio.

The PEP classifies cases of AFP as:

- poliomyelitis due to wild poliovirus, vaccine-derived poliovirus (VDPV) or vaccine associated paralytic poliomyelitis;
- polio compatible if there is insufficient evidence to exclude poliomyelitis;
- non-polio AFP or;
- non-AFP.

A follow-up questionnaire is sent to notifying clinicians if the PEP requires more information regarding the AFP case before a final classification can be made. After each PEP meeting, the Australian AFP case classifications are forwarded to WHO for inclusion in the global AFP surveillance data published in the Weekly Epidemiological Record (http://www.who.int/wer/en/). Ineligible cases are not reported to the WHO.

The WHO AFP surveillance performance indicator for a polio non-endemic country is 1 case of non-polio AFP per 100,000 children aged less than 15 years. For Australia in 2014, this equated to 44 cases per year, based on the Australian Bureau of Statistics data released in December 2013. An AFP surveillance scheme that satisfies the WHO surveillance performance indicator is deemed sufficiently sensitive to detect a wild poliovirus importation in children of that country. The WHO surveillance performance indicator for laboratory testing is that at least 80% of notified AFP cases have adequate faecal specimens collected and tested in a WHO accredited laboratory.

At the end of each calendar year, a number of AFP notifications remain pending as there is insufficient clinical and laboratory data for the PEP to report a final classification. The PEP classifies such notifications as 'polio compatible-zero evidence' if a final review reveals no proof of clustering among the cases. The WHO considers these cases to be non-polio AFP when determining Australia's case rate.

Virus culture

Upon receipt at the NERL, faecal specimens are treated with minimum essential medium containing Hank's salts, chloroform (9.1% v/v) and foetal bovine serum (2%). The suspension is clarified and the supernatant inoculated onto a series of mammalian cell lines. Two WHO recommended cell lines are used for the isolation of poliovirus, L20B (a transgenic mouse epithelial cell line expressing the human poliovirus receptor, CD155) and RD-A (human rhabdomyosarcoma).[11,12] Diagnostic laboratories in Australia are encouraged to refer enteroviruses of unknown serotype to the NERL for further characterisation.

Two WHO real time reverse transcription polymerase chain reaction (RT-PCR) tests are used to determine whether a poliovirus is a wild strain,

oral poliomyelitis vaccine (OPV) strain (Sabin-like) or a VDPV, in a process known as intratypic differentiation (ITD).[13] The NERL sequences the complete poliovirus viral protein 1 (VP1) genomic region, which contains a major neutralising antibody binding site. The VP1 genomic sequence provides valuable biological information, including the number of mutations within a significant region of the OPV virus strain and it enables phylogenetic analysis of wild poliovirus to rapidly determine the likely source of the virus, as utilised in the 2007 wild poliovirus importation.[14]

Enterovirus surveillance

The ERLNA was established primarily as a means of detecting imported poliovirus among untyped enteroviruses from clinical specimens. The network consists of 11 public sector diagnostic virology laboratories in the Australian Capital Territory (Canberra Hospital), New South Wales (Royal Prince Alfred Hospital and the Institute of Clinical Pathology and Medical Research), Queensland (Queensland Health and Scientific Services), South Australia (Flinders Medical Centre and the Institute of Medical and Veterinary Science), Tasmania (Royal Hobart Hospital), Victoria (Royal Children's Hospital and VIDRL) and Western Australia (Queen Elizabeth II Medical Centre and the Princess Margaret Hospital for Children).

The NERL encourages members of the ERLNA to perform their own enterovirus typing and report the results to the NERL for inclusion in the national enterovirus database. It has advised members of ERLNA on enterovirus detection, supplied laboratory and computer analysis protocols and performed tests in parallel with other laboratories for quality assurance purposes. The NERL receives untyped enteroviruses from 4 laboratories for typing on a regular basis.

The NERL screens clinical specimens for enterovirus using a semi-nested RT-PCR directed to highly conserved sequence in the 5' non-translated region.[15] Enterovirus typing is primarily performed by amplifying a fragment of the VP1 genomic region according to a published method,[16] but the complete nucleotide sequence of VP1 is required to type some enteroviruses. The enterovirus typing RT-PCR is directed to a region of sequence divergence that allows differentiation between enterovirus genomes. As a consequence, the enterovirus sequence based typing assay is not as sensitive as the pan-enterovirus detection assay. This can result in an enterovirus being detected by pan-enterovirus RT-PCR in a clinical specimen without subsequent identification by the VP1 enterovirus typing assay.

Environmental surveillance

The laboratory cell culture protocol implemented by the NERL for environmental surveillance is based on a two-phase separation procedure published by WHO[17] and further advice was obtained from the Enterovirus Laboratory at the National Public Health Institute, Finland, a Global Specialised Laboratory in the WHO Polio Laboratory Network. In brief, 800 ml of sewage is collected as a grab sample prior to any biological or chemical treatment and referred to the NERL within 24 hours. At the laboratory, 500 mL of the sample is vigorously shaken at 4°C with dextran, polyethylene glycol and sodium chloride. The mixture is incubated overnight at 4°C in a separating funnel and the lower organic phase collected the next day and clarified with chloroform as for a faecal specimen. Aliquots of the sewage concentrate are inoculated onto the L20B and RD-A cell lines and observed microscopically for cytopathic effect. The sewage extracts are tested in parallel by cell culture and a pan-enterovirus RT-PCR. The pan-enterovirus RT-PCR is a validated in-house test utilised to confirm the cell culture results, as not all human enteroviruses infect the RD-A cell line. All enterovirus isolates from cell culture and positive detections by RT-PCR are investigated to determine the virus type by nucleic acid sequencing.

Results

Classification of acute flaccid paralysis cases

A total of 80 notifications of AFP cases involving children less than 15 years of age were received in 2014 (Table 1). The PEP classified 58 cases as non-polio AFP, a rate of 1.4 cases per 100,000 children less than 15 years of age, which exceeds the WHO AFP surveillance performance criterion for a polio-free country of 1 case of non-polio AFP per 100,000 children (Table 2, Figure 1). Twelve cases were notified by more than 1 source, whether by 2 or more clinicians or a clinician and the PAEDS system. Seven notifications were deemed to be ineligible due to the patient's age being greater than 14 years or where the clinical presentation was subsequently determined not to be AFP. Specimens were received from 2 notifications of AFP cases that were later classified by the PEP as polio compatible-zero evidence due to insufficient clinical information. The WHO accepts such cases as non-polio AFP when reviewing Australia's surveillance data due to the quality and breadth of the national polio surveillance program.

In 2014, an Australian adult was hospitalised upon returning from the Horn of Africa. The patient had experienced fever and headache and

Table 1: Notification of acute flaccid paralysis cases, Australia, 2014, by state or territory

State or territory	Estimated population aged <15 years*	Expected number of reported AFP cases in 2014	Total number of notifications	Ineligible notifications	Duplicate notifications	Polio compatible-zero evidence	Eligible cases with final classification by the Polio Expert Panel	Non-polio AFP rate per 100,000 children
ACT	70,739	1.0	0	0	0	0	0	0.0
NSW	1,398,648	14.0	17	3	1	0	13	0.9
NT	53,589	0.5	1	0	0	1	0	2.0
Qld	924,703	9.0	16	1	3	0	12	1.3
SA	295,303	3.0	3	1	0	0	2	0.7
Tas.	94,959	1.0	0	0	0	0	0	0.0
Vic.	1,051,114	10.5	35	2	7	1	25	2.5
WA	482,308	5.0	7	0	1	0	6	1.2
Australia	4,371,363	44.0	79	7	12	2	58	1.4

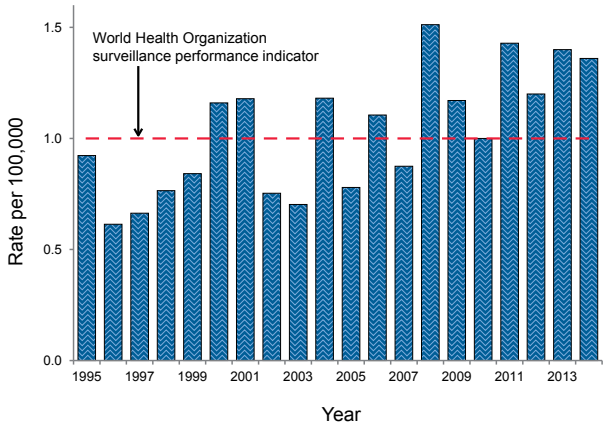
* Australian Bureau of Statistics, estimated population at 30 June 2013. Available from the Australian Bureau of Statistics web site (www.abs.gov.au).

AFP Acute flaccid paralysis

Table 2: Australia's surveillance for cases of acute flaccid paralysis, 2014, compared with the main World Health Organization performance indicators

WHO surveillance performance indicator for acute flaccid paralysis cases in children <15 years	Performance of Australia's acute flaccid paralysis surveillance	
≥1.0 non-polio acute flaccid paralysis (AFP) case per 100,000 children (44 cases for Australia in 2014)	60 cases classified as non-polio AFP	1.36 (60 / 44) non-polio AFP cases per 100,000 children <15 years
≥80% of classified AFP cases with adequate specimens (2 faecal specimens collected at least 24 hours apart and within 14 days of onset of paralysis	30 AFP cases with adequate specimens collected	50% (30 / 60) classified non-polio AFP cases with adequate specimens

Figure 1: Non-polio acute flaccid paralysis rate for children less than 15 years of age, Australia, 1995 to 2014*



* The World Health Organization acute flaccid paralysis surveillance performance indicator for a polio non-endemic country is one case per 100,000 children less than 15 years of age, which is highlighted by the dotted line.

subsequently developed a flaccid paralysis of an upper limb while overseas. The attending clinicians in Australia notified the case as suspected poliomyelitis but enterovirus was not detected in the specimens referred to the NERL for testing. The case was initially reported to WHO as polio compatible pending further clinical and laboratory investigation. The differential diagnosis included acute myelitis due to viral infection but the risk to persons in contact with the index case while in transit and within Australia was considered very low based on enterovirus not being detected in the patient's specimens. Polio serology was performed when acute and convalescent sera became available and no evidence of an increasing titre (that would be consistent with recent poliovirus infection) was observed. The case was subsequently classified by the PEP as non-polio AFP and diagnosed as West Nile virus infection by the attending clinicians based on serial serum sampling.[18]

Notification of acute flaccid paralysis cases by state and territory

In 2014, AFP cases were notified from all jurisdictions in Australia except the Australian Capital Territory and Tasmania; it is estimated that these 2 jurisdictions will report 1 case per year based on the population less than 15 years of age so this result may not be surprising (Table 1). The non-polio AFP rates for eligible cases per jurisdiction exceeded the WHO AFP surveillance performance indicator of 1 case per 100,000 children in the Northern Territory, Queensland, Victoria and Western Australia, accounting for 73% of the national cases. The most populous state, New South Wales, did not achieve the expected rate of reporting of non-polio AFP cases for the first time since 2007.

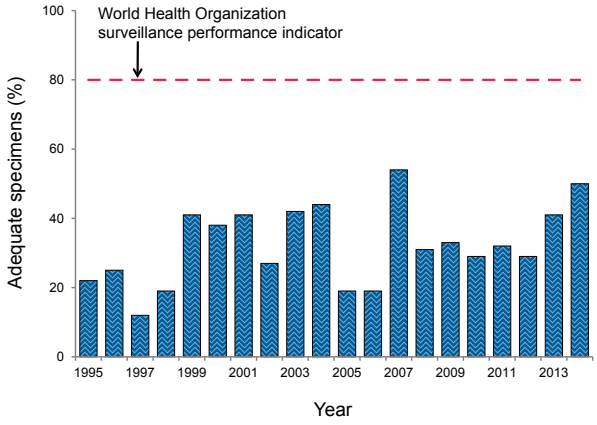
Faecal collection from acute flaccid paralysis cases

A total of 95 faecal specimens from 47 of the 60 eligible cases were tested at the NERL in 2014, but only 30 AFP cases met the WHO criteria for adequate specimen collection with 2 specimens collected within 14 days of the onset of paralysis and more than 24 hours apart (Figure 2, Tables 2 and 3). The proportion of cases with at least 1 specimen collected within 14 days of the onset of paralysis was 73%, while 78% of cases had a specimen collected any time after the onset of paralysis. No poliovirus was detected in any of the specimens. The NPEVs echovirus types 6 and 7 were individually detected from faecal specimens of 2 AFP cases. Enterovirus A71 was detected from 1 AFP case with onset in December 2014, which was in contrast to the outbreak reported in 2013, when this type of NPEV was reported from 9 AFP cases between February and September.

Table 3: Specimens referred to the National Enterovirus Reference Laboratory, Australia, 2014

Result	Specimens from acute flaccid paralysis cases involving children <15 years of age	Specimens from acute flaccid paralysis cases involving patients ≥15 years of age	Enterovirus surveillance	Environmental surveillance	Total
Non-polio enterovirus	3	0	62	3	68
Rhinovirus	0	0	2	0	2
No enterovirus identified	92	5	31	0	128
Total	95	5	95	3	198

Figure 2: Adequate faecal specimen rate, Australia, 1995 to 2014*



* The main World Health Organization criteria for adequate specimen collection is 2 faecal specimens collected more than 24 hours apart and within 14 days of the onset of paralysis from 80% of the cases classified as non-polio acute flaccid paralysis.

Enterovirus surveillance

Poliovirus was not detected by any member of the ERLNA in 2014, with a total of 506 NPEVs typed by members of the network from clinical specimens (Table 4). The most common genotypes identified in order of decreasing frequency were coxsackievirus A6, echovirus 6, coxsackievirus B5 and coxsackievirus B4 accounting for two-thirds of the total, while only sporadic detections of enterovirus A71 were reported.

Environmental surveillance

Environmental surveillance for poliovirus was established at a sentinel site in metropolitan Melbourne from December 2014. No poliovirus was identified from the 3 grab samples tested but each was positive for NPEVs that act as an indicator organism for the collection, transport and test procedures. It is planned to continue the collections at the Melbourne site on a weekly basis through 2015 and then reassess the choice of site based on the results.

Poliovirus regional reference laboratory activities

In 2014, as part of its role as a Polio Regional Reference Laboratory, the NERL received specimens from AFP cases referred from Brunei Darussalam (2 cases), Pacific Island countries (12 cases) and Papua New Guinea (10 cases). The Sabin poliovirus serotype 3 vaccine strain was isolated from both specimens of 1 AFP case in Papua New Guinea, along with an NPEV. NPEVs were reported from another 7 cases in Papua New Guinea and 4 AFP cases from the Pacific Islands.

Quality assurance programs

In 2014, the NERL was accredited as a WHO Polio Regional Reference Laboratory through participation in the annual WHO polio quality assurance panels for RT-PCR for ITD and vaccine derived poliovirus and poliovirus VP1 sequencing. The laboratory also successfully participated in the Royal College of Pathologists of Australasia quality assurance panel for enterovirus detection by RT-PCR.

Discussion

In 2014, Australia reached the WHO surveillance target of ≥1 non-polio AFP case per 100,000 children under 15 years of age, for the 7th year in a row. The combination of clinicians notifying AFP cases via the APSU monthly report card and nurses ascertaining cases through the PAEDS system provided Australia with a polio surveillance system that meets the international standard to detect an imported case of polio in children less than 15 years of age through these well-established schemes.[1–3] Australia has never met the strict WHO surveillance target for adequate stool collection from 80% of the non-polio AFP cases, with 47% of cases having 2 specimens tested in 2014, and 65% of cases with at least 1 specimen.

Three different types of NPEV-echovirus types 6 and 7 and enterovirus A71 were detected from 3 distinct AFP cases in 2014. Since most enterovi-

Table 4: Enterovirus test results from the National Enterovirus Reference Laboratory, Australia, 1995 to 2014

Year	Poliovirus		Non-polio enterovirus	No enterovirus detected	EVID results referred*	Total samples reviewed
	Sabin-like	Non-Sabin-like				
1995	190	0	200	13	0	403
1996	224	0	198	9	0	431
1997	124	0	76	0	0	200
1998	52	0	15	4	0	71
1999†	60	1	9	9	0	79
2000	45	0	44	47	0	136
2001†	46	5	33	75	0	159
2002	36	0	21	49	0	106
2003	9	0	15	47	0	71
2004	6	0	26	61	0	93
2005	18	0	10	39	0	67
2006	2	0	6	71	29	108
2007‡	0	2	32	115	107	256
2008	0	0	20	92	77	189
2009§	1	0	63	78	113	255
2010	0	0	170	39	108	317
2011	0	0	174	61	205	440
2012	0	0	155	97	123	375
2013¶	1	0	242	198	230	671
2014	0	0	68	128	506	702

* Enterovirus Identification (EVID) results include retrospective data made available via the Enterovirus Reference Laboratory Network of Australia.

† Untyped enterovirus or uncharacterised poliovirus isolates were referred for further testing after completion of a laboratory inventory. The 6 isolates tested as non-Sabin-like and were subsequently identified as wild type poliovirus prototype strains and were destroyed.

‡ Wild poliovirus type 1 was imported from Pakistan.

§ A Sabin-like poliovirus type 1 was identified from an unimmunised infant.

¶ A Sabin-like poliovirus type 2 was identified from an infant who was immunised overseas with oral polio vaccine and hospitalised with diarrhoea upon return to Australia.

rus infections are asymptomatic, and the viruses were detected from a non-sterile site (faeces), the results could be an incidental finding; however, all three viruses have been associated with AFP worldwide. Even though enterovirus A71 was reported from only one AFP case in 2014, public health officials continue to monitor accounts of this enterovirus type after the outbreak in 2013, when it was detected from 9 AFP cases in New South Wales and Victoria.

Enterovirus D68 became of public health interest in 2014, with reports of geographical and temporal associations with AFP in the United States of America (USA).[19] Enterovirus D68 was first isolated in 1962 and its detection in clinical specimens worldwide was sporadic until 2008, when acute respiratory illness including fatalities were reported in the Philippines (2008–2009), United Kingdom (2009–2010), Japan (2010), Netherlands (2010), New Zealand (2010), Canada (2014) and the USA (2014).[19,20] While enterovirus D68 was not detected in cerebrospinal fluid during the USA outbreak, there have been 2 such reports: a young adult with AFP and a fatal meningomyeloencephalitis in a 5-year-old in 2005 and 2008, respectively.[21,22] The extensive clinical, virological and epidemiological evidence from the enterovirus D68 outbreak during 2014 in the USA strengthens the evidence for a causal association with AFP but further investigation of the virus lineage to elucidate the genetic elements concerned with neurological disease is warranted. Enterovirus D68 was detected once from an AFP case in Australia in 2010[23] and heightened activity was noted in Western Australia in 2011 and 2013.[24] The NERL will continue to screen the ERLNA typing results for any apparent variation in enterovirus transmission of this and other enterovirus types.

Environmental surveillance by testing grab samples of raw sewage has proved to be a sensitive means of detecting poliovirus in the absence of clinical cases overseas, such as occurred in Israel with 150 positive samples in 2013 to 2014[25] and in Quetta, Rawalpindi and Lahore in Pakistan in 2014.[26] WHO estimates the environmental test protocol to be capable of detecting 1 person shedding poliovirus among 10,000 uninfected persons.[17] Australia implemented sentinel environmental surveillance for poliovirus at 4 rural and regional sites from 2009. In 2012, the Polio Surveillance Systems Review recommended environmental surveillance be trialled at a major metropolitan site[27] and this led to testing commencing in Melbourne in 2014. The much larger population catchment of a metropolitan site necessitated more frequent testing to compensate for reduced sample sensitivity compared with the quarterly testing at the rural and regional sites, so collections in Melbourne will continue on a weekly basis in 2015.

It is extremely important that all countries employ sensitive surveillance for AFP in children and poliovirus even as the global case count and number of endemic countries reduces. It will require 3 years of sensitive surveillance to certify global polio eradication due to most infections being asymptomatic and to account for potential gaps in immunisation and surveillance.

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Australian Paediatric Surveillance Unit annual report, 2015

Marie Deverell, Amy Phu, Yvonne A Zurynski, Elizabeth J Elliott, and all chief investigators of Australian Paediatric Surveillance Unit surveillance studies

Context

This report provides an update on the surveillance conducted by the Australian Paediatric Surveillance Unit (APSU) during the period January to December 2015.

Introduction

The APSU was established in 1993 to facilitate national active surveillance of uncommon diseases of childhood including selected communicable diseases. This report includes data on the following conditions: acute flaccid paralysis (AFP), a surrogate condition for poliovirus infection; congenital cytomegalovirus; congenital rubella; perinatal exposure to HIV and paediatric HIV infection; neonatal and infant herpes simplex virus (HSV); congenital varicella; neonatal varicella; and juvenile-onset recurrent respiratory papillomatosis (JoRRP). Surveillance of severe complications of influenza was undertaken during the influenza season (1 July to 30 September 2015).

Methods

Australian Paediatric Surveillance Unit

APSU study protocols and case definitions are developed with collaborating study investigators who provide specialised clinical expertise for each condition studied. These conditions are listed in the Table. Each month approximately 1,500 paediatricians and other child health clinicians nationally are sent the APSU report card. Over 90% of clinicians report via email; they respond each month whether or not they have a case to report for any of the conditions listed on the report card.[1] The APSU collects de-identified clinical and/or laboratory data via a case report form completed by the doctor looking after the child. Completed case report forms are then forwarded on to study investigators. All study protocols and case report forms are available for download from the APSU website (www.apsu.org.au). The response rate to the monthly report card was 90% in 2015.

For surveillance of AFP, the APSU collaborates with the Paediatric Active Enhanced Disease Surveillance (PAEDS) system. PAEDS is a hospital-based surveillance system reliant on active case ascertainment by specialist surveillance nurses and operates in 5 tertiary hospitals around Australia.[2] For data on AFP collected through PAEDS please refer to the PAEDS annual report for 2015 published on the *Communicable Diseases Intelligence* website.[3]

All reported rates are based on child population estimates published by the Australian Bureau of Statistics.[4]

Results

Acute flaccid paralysis

Paediatricians are instructed to report all cases of AFP immediately after they are identified to the APSU and the National Polio Reference Laboratory. Data from the APSU are submitted regularly to the Polio Expert Panel. In 2015, there were a total of 25 confirmed cases of AFP notified to the APSU. Of the 25 confirmed cases, 14 were reported from Victoria, 4 from New South Wales, 3 from Tasmania, 3 from Western Australia and 1 from South Australia. All cases were reviewed by the Polio Expert Panel and classified as non-polio AFP. The main diagnoses associated with AFP cases were Guillain-Barré syndrome (40%), transverse myelitis (14%), acute disseminated encephalomyelitis (14%), botulism (8%) and acute axonal neuropathy (8%). APSU contributes to the national AFP surveillance efforts to reach the World Health Organization surveillance target of 1 case per 100,000 children aged less than 15 years per annum.[5]

Congenital cytomegalovirus

In 2015, 18 confirmed cases of congenital cytomegalovirus were reported to the APSU, with 291 confirmed cases reported during the entire study period 1999 to 2015. Of the 18 confirmed cases, 6 were from Queensland, 5 were from New South Wales, 3 from Victoria, 2 from Western Australia and 2 from the Northern Territory. All of the 18 children were born in Australia, none identified as Aboriginal or Torres Strait Islander.

Table: Confirmed cases identified Australian children aged < 16 years in 2015 and for the total study period, and reported rates per 100,000 of the relevant child population, by condition

Condition	Date study commenced	Questionnaire returned (%)	Number of confirmed cases 2015	Reported rate for 2015 (per 100,000)	Number of confirmed cases for total study period	Reported rate for total study period (per 100,000 per annum)
Acute flaccid paralysis	Mar 1995	100	25*	0.56†	892	1.04†
Congenital cytomegalovirus	Jan 1999	81	18	5.93‡	291	6.58‡
Congenital rubella (with defects)	May 1993	100	1	0.02§	54	0.06‖
Perinatal exposure to HIV	May 1993	100	38	12.68‡	664	11.15‡
HIV Infection	May 1993		Nil	Nil	87	0.09§
Neonatal – herpes simplex virus infection	Jan 1997	87	16	5.34‡	180	6.35‡
Infant – herpes simplex virus infection	Jan 2012		3	0.99¶	15	0.98¶
Congenital varicella	May 2006	No notifications	Nil	Nil	2	0.00§
Neonatal varicella	May 2006	100	4	0.09‡	26	0.08‡
Juvenile onset recurrent respiratory papillomatosis (JoRRP)**	Oct 2011	100	1	0.02†	13	0.07†
Severe complications of influenza††	Influenza season each year since 2008	99	84	1.88†	488	1.42†

* Includes all cases of acute flaccid paralysis reported via the Australian Paediatric Surveillance Unit. All cases have been classified by the Polio Expert Panel as 'non-polio acute flaccid paralysis according to World Health Organization criteria. The number of confirmed cases for the total study period includes both the Australian Paediatric Surveillance Unit and Paediatric Active Enhanced Disease Surveillance data.

† Based on population of children aged less than 15 years.

§ Based on number of births.

‖ Based on population of children aged less than 16 years.

¶ Based on population aged less than 12 months.

** Includes both confirmed (visualisation via endoscopy and histology report) and probable cases (visualisation via endoscopy but no histology report).

†† Influenza surveillance was conducted each year since 2008 during the influenza season, 1 July to 30 September except in the pandemic year (2009) when surveillance occurred from 1 June to 31 October.

Congenital rubella

There were 2 notifications of congenital rubella reported to the APSU during 2015. There was 1 confirmed case from Queensland and the other case from South Australia did not meet criteria to be classified as congenital rubella syndrome. This means that there have been 3 cases of congenital rubella detected in the last 3 years including 2 confirmed cases in 2013. All children were born in Australia to mothers who had been born overseas (Thailand and Indonesia); 2 of the mothers reported that they were unvaccinated and 1 did not know her vaccination status. No cases had been reported in the 5 years 2009 to 2014. During the entire study period 1993 to 2015 there have been 59 cases of congenital rubella (54 confirmed and 5 probable) reported to the APSU. It is mainly due to the National Immunisation Program that Australia has seen a reduction in congenital rubella infection. However, reports of imported and locally acquired cases among immigrant unvaccinated women during previous years reinforce the need for continued surveillance and vaccination.[6]

Perinatal exposure to HIV and HIV infection

There were 38 confirmed cases of perinatal exposure to HIV reported to the APSU in 2015, but no cases of HIV infection in children. Of the 38 confirmed cases, 20 were from Victoria, 15 were from New South Wales, 2 from Western Australia and 1 from the Australian Capital Territory. Only 1 child with perinatal exposure to HIV was of Aboriginal or Torres Strait Islander descent.

The majority of mothers of these children were receiving antiretroviral therapy (n=30, 79%). Women most frequently gave birth by vaginal delivery (n=20, 53%) or by elective caesarean section (n=10, 26%) and 3 had an emergency caesarean section. Most mothers (n=32, 84%) reported avoiding breastfeeding their children.

Neonatal and infant herpes simplex virus

Of 26 notifications, there were 19 confirmed cases of neonatal or infant HSV reported to the APSU in 2015. There were 16 neonatal cases aged less than 1 month, and 3 were infant onset cases aged between 1 month and 1 year. Of the 16 neonatal cases, 5 were reported from New South Wales, 4 from Victoria, 2 from Western Australia, 2 from Australian Capital Territory, 2 from Queensland and 1 from South Australia.

Eleven cases had HSV-1 and 5 had HSV-2. Seven cases had skin, eye, mouth (SEM) disease, 6 had HSV central nervous system disease and 3 had disseminated disease. Of the 3 infant onset cases, 2 were reported from New South Wales and 1 from Victoria. All 3 cases had HSV-1 and all 3 had SEM disease. There were 3 deaths in 2015 and all 3 were neonatal cases. One of these had SEM disease but died from causes other than HSV, 1 had HSV encephalitis and 1 had disseminated HSV with central nervous system symptoms.

Congenital and neonatal varicella

There were no cases of congenital varicella reported during 2015. The last case of congenital varicella reported to the APSU was in 2007. Four cases of neonatal varicella were reported to the APSU in 2015. Of these, 2 were from Queensland, 1 from New South Wales and 1 from Western Australia. All 4 infants were exposed to varicella after birth, however the details of the infective contacts were not known. All infants required hospitalisation due to varicella infection (length of stay 3–10 days), and all were treated with Aciclovir.

Juvenile onset recurrent respiratory papillomatosis

There was 1 confirmed case of JoRRP in 2015. The case from New South Wales was confirmed by visualisation of lesions on endoscopy and histology results. The child was Caucasian and 9 months of age. During the total study period (2011–2015) there have been 18 notifications, with detailed clinical data available for 17 (94%) cases. Of the 17 completed case reports there were 3 duplicate reports and 1 error. Of the remaining 13 notifications there were 10 confirmed and 3 probable cases: 6 confirmed and 1 probable case in 2012; 2 confirmed and 1 probable in 2013; and 1 confirmed and 1 probable case in 2014. These data suggest a declining trend in JoRRP since surveillance commenced in 2011. This may be a result of the successful HPV Vaccination Program introduced to Australia in 2007.

Severe complications of influenza

A total of 84 children admitted to hospital with serious complications of influenza were reported to the APSU from 1 July to 30 September 2015. Of the 84 children, 41 were from Queensland, 16 from New South Wales, 14 from South Australia, 8 from Victoria, 3 from Tasmania and 2 from Western Australia. None of the children were Aboriginal or Torres Strait Islander.

There has been an increase in the number of notifications reported to the APSU compared to previous years (n=22 in 2013 and n=87 in 2014). However, this increase could be due to the introduction of online reporting of cases. The most commonly reported strain in 2015 was Influenza B (n=59).

Twenty-five children had Influenza A. Serious complications included pneumonia (n=41), seizures (n=24) and encephalitis (n=11).

In 2015, 28 (33%) children required an intensive care unit admission and 3 (4%) children died. Of the 84 children, 53 were previously healthy, while 30 had chronic predisposing conditions including asthma, cerebral palsy, chronic lung disease, and congenital heart disease.

Only 4 of the 84 children were vaccinated for influenza within the last 12 months and all of them had chronic predisposing conditions. Children with chronic predisposing conditions are recommended and funded for annual influenza vaccination under the National Immunisation Program, however only 4 (13%) of the 30 of eligible children were vaccinated.

Conclusions and future directions

For over 20 years the APSU has been facilitating the active surveillance of uncommon rare childhood diseases, complications of common diseases or adverse effects of treatment. This year the APSU introduced surveillance of microcephaly in children less than 12 months of age. Microcephaly is defined as an occipito-frontal head circumference more than 2 standard deviations below the mean for age, gender and gestation. This rare condition is often associated with symptoms of neurological impairment including seizures and may also be associated with developmental delay, intellectual impairment, problems with vision, hearing and feeding. There are many causes of microcephaly, including congenital infections such as cytomegalovirus, rubella and rarely, herpes simplex virus, syphilis and varicella zoster virus, and very rarely, HIV. Microcephaly is of current interest due to the proven relationship between maternal Zika virus infection during pregnancy. For more information on current APSU surveillance of microcephaly please visit the APSU website (www.apsu.org.au).

The APSU continues to lead the way in rare disease research and provides valuable data on clinical, treatment and outcome data on infectious and vaccine preventable conditions in Australian children. The data collected through the APSU contribute significantly to the national surveillance effort, providing valuable information for clinicians, policy makers and the community.

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Chief Investigators of APSU surveillance studies:

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Quarterly report

OzFoodNet quarterly report, 1 January to 31 March 2015

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. In addition, 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 2015.

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 2015 (1 January to 31 March), OzFoodNet sites reported 399 outbreaks of enteric illness, including those transmitted by contaminated food or water. Outbreaks of gastroenteritis are often not reported to health authorities, which results in current figures underrepresenting the true burden of enteric disease outbreaks within Australia. There were 5,899 people affected in these outbreaks and 253 hospitalisations. There were 20 deaths reported during these outbreaks. This represents a decrease in the number of people affected compared with the 5-year average from 2010 to 2014 for the 1st quarter (6,626). The majority of reported outbreaks of gastrointestinal illness in Australia are due to person-to-person transmission. In this quarter, 74% (296/399) of outbreaks were transmitted via this route (Table 1). This percentage was slightly higher than the same quarter in 2014 (72%, 335/465) but the total number is lower than the 5-year mean (1st quarter, 2010–2014) of 302 outbreaks transmitted person-to-person. Of the person-to-person outbreaks in the 1st quarter of 2015, 52% (153/296) occurred in child care facilities and 39% (114/296) occurred in aged care facilities.

Foodborne and suspected foodborne disease outbreaks

There were 47 outbreaks during this quarter where consumption of contaminated food was suspected or confirmed as being the primary mode of transmission (Appendix). These outbreaks affected 891 people, of which 602 were laboratory-confirmed cases, and resulted in 101 hospitalisations. There were 2 deaths reported during these outbreaks.

This was a decrease on the number of foodborne outbreaks that were reported in the 4th quarter of 2014 (54) and an increase on the 5-year mean for the 1st quarter between 2010 and 2014 (45). The data within this report, provided by OzFoodNet sites, has associated limitations, including the potential variation in categorisation of features of outbreaks, depending on varied circumstances and investigator interpretation. Changes in the

Table 1: Outbreaks and clusters of gastrointestinal illness and number ill reported by OzFoodNet, Australia, 1 January to 31 March 2015, by mode of transmission

Transmission mode	Number of outbreaks and clusters	Per cent of total*	Number ill
Person-to-person	296	74	4,361
Foodborne and suspected foodborne	47	12	891
Unknown	43	11	361
Unknown (*Salmonella* cluster)	11	3	265
Suspected waterborne	2	1	21
Total	399	100	5,899

* May not add up to 100% due to rounding.

number of foodborne disease outbreaks should be interpreted with caution due to the small number each quarter.

Salmonella Typhimurium was identified as the aetiological agent in 74% (35/47) of foodborne or suspected foodborne outbreaks during this quarter (Appendix); a higher proportion than for the same quarter in 2014 (67%, 33/49). The aetiological agents for the remaining outbreaks included: norovirus in 3 outbreaks; ciguatoxin in 2 outbreaks; and *Clostridium perfringens;* *S.* Bovismorbificans; *Salmonella* (untyped); *S.* Virchow; and histamine poisoning for 1 outbreak each. For 2 outbreaks the aetiological agent was unknown.

Twenty-seven outbreaks (57% of all foodborne or suspected foodborne outbreaks) reported in this quarter were associated with food prepared in restaurants (Table 2). This was higher than the average number of restaurant associated foodborne or suspected foodborne outbreaks in the 1st quarter from 2010 to 2014 (18).

To investigate these outbreaks, sites conducted 2 cohort studies, 5 case control studies and collected descriptive case series data for 27 investigations. For 13 outbreaks, no individual patient data were collected. The evidence used to implicate food vehicles included analytical evidence in 4 outbreaks, microbiological evidence in 8 outbreaks, both analytical and microbiological evidence in 4 outbreaks, and descriptive evidence in 31 outbreaks.

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

Australian Capital Territory

There were 2 outbreaks of foodborne or suspected foodborne illness reported in the Australian Capital Territory in this quarter. For reporting purposes, both of these outbreaks were considered New South Wales events, one of which is described in the New South Wales key outbreaks.

New South Wales

There were 15 outbreaks of foodborne or suspected foodborne illness reported in New South Wales in this quarter. The aetiological agents identified were *S.* Typhimurium (for 9 outbreaks) and *S.* Bovismorbificans, *S.* Virchow, *Salmonella* (untyped) and histamine poisoning (for 1 outbreak each). Two outbreaks were of unknown aetiology.

Description of key outbreaks

An outbreak was investigated after salmonellosis affected 33 residents across 10 aged care facilities (ACFs) in New South Wales and the Australian Capital Territory. Facility-based attack rates ranged from 0.6% to 7.5%. The ACF's were managed by the same organisation and shared common food suppliers. All 33 cases tested positive for *S.* Bovismorbificans, 30 were further characterised as phage type (PT) 14 and the remaining 3 were not phage typed. The environmental investigation

Table 2: Outbreaks of foodborne or suspected foodborne disease and number ill reported by OzFoodNet, Australia, 1 January to 31 March 2015, by food preparation setting

Food preparation setting	Number of outbreaks	Per cent of foodborne outbreaks*	Number ill	Number laboratory confirmed
Restaurant	27	57	375	258
Takeaway	5	11	51	14
Aged care	4	9	48	42
Private residence	2	4	26	12
Primary production	2	4	8	0
Other (Conference centre)	2	4	170	151
Commercial caterer	1	2	7	1
National franchised fast food restaurants	1	2	48	46
Institution – not otherwise specified	1	2	69	31
Picnic	1	2	4	3
Unknown	1	2	85	44
Total	47	100	891	602

* May not add up to 100% due to rounding.

identified *S.* Bovismorbificans PT 14 at the premises of a baked dessert supplier to the ACFs and in food samples.

An outbreak was investigated in February after 4 separate cases of suspected histamine fish poisoning. Cases presented with red face, headache, tingling, sweating, vomiting and palpitations. An investigation identified a total of 7 suspected cases who had consumed tuna salad from the same local food outlet. Onset of symptoms was within 10 to 15 minutes of tuna consumption. The NSW Food Authority initiated an investigation resulting in a trade level recall of an imported canned tuna product.

Northern Territory

There were no outbreaks of foodborne or suspected foodborne illness reported in the Northern Territory in this quarter.

Queensland

There were 16 outbreaks of foodborne or suspected foodborne illness reported in Queensland in this quarter. This represents 34% of the total number of foodborne or suspected foodborne outbreaks for the quarter (16/47) nationally, but 66% of the total number of people affected (584/891). The aetiological agents identified were *S.* Typhimurium (for 13 outbreaks), ciguatoxin (for 2 outbreaks) and norovirus (for 1 outbreak).

Description of key outbreaks

An outbreak was investigated in January after Queensland Health was notified of gastrointestinal illness by several hospital emergency departments and members of the public who had consumed meals at the same restaurant over several days. In total, 138 people from multiple groups reported symptoms including diarrhoea and/or vomiting and/or stomach cramps. *S.* Typhimurium with the multi-locus variable number tandem repeat analysis (MLVA) pattern 03-12-11-12-523* was identified in 95 cases. A case-control study identified multiple food items significantly associated with illness including deep fried ice-cream (adjusted odds ratio [aOR] 122.7, 95% confidence interval [CI] 31.0–485.4, $P = 0.001$), lemon chicken (aOR 14.5, 95% CI 2.1–98.5, $P = 0.006$) and sweet and sour pork (aOR 5.2, 95% CI 1.3–21.1, $P = 0.02$). Environmental health officers identified multiple food hygiene issues and widespread bacterial contamination throughout the premises. *S.* Typhimurium MLVA 03-12-11-12-523 was detected on surfaces and in multiple food items from the restaurant kitchen, as well as in drag swabs and (spent) chicken feed from the egg farm that supplied the restaurant. Regulatory action was subsequently taken against the restaurant.

An outbreak was investigated in March after multiple cases of illness were associated with meals consumed at the same café. In total, 44 people reported either diarrhoea, vomiting or stomach cramps within 3 days after consuming a meal at the café, or had a stool sample that was culture positive for *S.* Typhimurium MLVA 03-17-09-11-523. Thirty-one cases were laboratory confirmed. A case-control study identified a significant association between an eggs Benedict meal and illness (OR 124.4, 95% CI 14.2–3693.7, $P < 0.001$). Environmental health officers identified hygiene and temperature control issues at the café and the license was temporarily suspended. *S.* Typhimurium MLVA 03-17-09-11-523 was isolated from guacamole, tea towels, a cleaning cloth and swabs from the sink area at the café. These findings were indicative of cross contamination within the kitchen. The same pathogen was also found in drag swabs and chicken faeces samples from the supplying egg farm.

An outbreak involving at least 140 cases of salmonellosis was investigated in association with a conference held in late February. Nine cases were hospitalised. A case control study identified 2 items significantly associated with illness: rum and raisin bread cake (aOR 4.0, 95% CI 1.7–9.3, $P = 0.001$) and custard (aOR 11.3, 95% CI 4.8–26.5, $P < 0.001$). *S.* Typhimurium MLVA 03-12-12-09-523 was isolated from the faecal specimens collected from 58 attendees. Isolates with the same MLVA type were identified on 3 swabs taken from a stick blender used to prepare the implicated food, and in environmental samples from the farm of one of the egg suppliers. Following this investigation, the facility removed stick blenders from the kitchen, introduced the use of pasteurised egg and 'high risk' menu items were removed. It also adopted an ongoing microbial monitoring regime as part of its food safety program.

An outbreak was investigated in January and February after 48 cases of *S.* Typhimurium PT U307, MLVA 03-12-11-12-523 were linked to over 20 outlets of the same franchised food chain. Fourteen hospitalisations were reported. Food histories collected from the cases identified a chocolate mousse product that was consumed by 84% of cases. Environmental health investigations identified that the chocolate mousse was supplied to a central kitchen by another manufacturer.

* In December 2014, the Queensland enteric reference laboratory Queensland Health Forensic and Scientific Services agreed to harmonise reporting of the MLVA STTR3 allele '524' as '523' in line with other states and territories.

Chocolate mousse samples collected from retail and multiple food samples collected from the manufacturer (including chocolate mousse and a selection of cheesecakes and chocolate cakes) were positive for *S.* Typhimurium 03-12-11-12-523. These products were all prepared using a raw egg mixture with no subsequent cooking step. Investigations identified that the chocolate mousse product had also been supplied to 2 venues where outbreaks of the same *S.* Typhimurium MLVA had occurred in the 4th quarter of 2014. All food businesses supplied with these raw egg-based products were requested to withdraw the products from sale and destroy them. The manufacturer ceased making the products and switched to the use of pasteurised egg.

An outbreak in January affected at least 85 people who reported consuming Korean style 'kimbap' sushi packs. Kimbap is a potentially hazardous food because it is usually kept at room temperature to avoid retrogradation of the rice starch.[1] It also is not acidified with rice vinegar. The kimbap packs were from multiple outlets and contained combinations of egg, tuna, vegetables, ham or seafood extender. Environmental health investigations were able to trace the production of the kimbap packs to a single unlicensed manufacturer, although the kitchen where the kimbap was prepared was not located. Forty-four faecal specimens tested positive for *S.* Typhimurium MLVA 03-12-11-12-523, as did 2 retail samples of kimbap. A source of contamination was unable to be confirmed during this outbreak, though eggs were suspected based on findings from multiple concurrent outbreaks occurring within Queensland with the same MLVA profile.

South Australia

There were 4 outbreaks of foodborne and suspected foodborne illness reported in South Australia in this quarter. The aetiological agent identified for all 4 outbreaks was *S.* Typhimurium.

Description of key outbreaks

An outbreak was investigated after initial interviewing identified 3 *S.* Typhimurium PT 9 cases that had consumed meals at the same hotel. Further investigation linked a total of 7 cases to the same hotel. Two cases were hospitalised. *S.* Typhimurium PT 9, MLVA 03-24-13-10-523 was subsequently isolated from environmental swabs of the internal components of the stick blender used to prepare raw egg aioli, soups and dressings at the hotel. All 7 human cases had the same MLVA profile as the environmental sample from the stick blender.

Tasmania

There were no outbreaks of foodborne or suspected foodborne illness reported in Tasmania in this quarter.

Victoria

There were 6 outbreaks of foodborne or suspected foodborne illness reported in Victoria in this quarter. The aetiological agents identified were *S.* Typhimurium (for 5 outbreaks) and *C. perfringens*.

Description of key outbreaks

An outbreak investigation commenced in February after 4 cases of *S.* Typhimurium PT 9, MLVA 03-24-16/15-12-525 were identified to be clustered geographically by residence. Investigations revealed that the cases had eaten at one of 2 cafés owned and operated by the same proprietors. Further case finding identified an additional 14 cases who had eaten at either of these cafés. Of the 18 ill, 13 (including 4 staff members), were confirmed with *S.* Typhimurium PT 9 with the outbreak MLVA pattern. Five cases were hospitalised. Sixteen cases had eaten sandwiches (14 chicken and mayonnaise and 2 tuna and mayonnaise), including 2 employees of the café. A sample of the chicken and mayonnaise mixture was positive for *S.* Typhimurium PT 9, with the outbreak MLVA pattern. The implicated mayonnaise was prepared onsite with raw eggs. The source of *S.* Typhimurium PT 9 in the chicken and mayonnaise mixture was unable to be confirmed.

An outbreak was investigated in February following notification of 2 cases of *S.* Typhimurium PT 135a, MLVA 03-11-11-16-525 in residents of the same ACF. The cases resided in the same wing, required fully assisted feeding and only consumed vitamised meals. An inspection revealed that the blender used to vitamise meals was also used for blending raw eggs. Faecal specimens were taken from a further 12 residents who were asymptomatic but consumed vitamised meals; 5 were positive for *S.* Typhimurium PT 135a, MLVA 03-11-11-16-525. Food and environmental samples including swabs of the blender used to vitamise meals were negative for *Salmonella*. This outbreak was suspected to have been caused by cross contamination from eggs.

Western Australia

There were 4 outbreaks of foodborne or suspected foodborne illness reported in Western Australia in this quarter. The aetiological agents identified were norovirus and *S.* Typhimurium (for 2 outbreaks each).

Description of key outbreaks

An outbreak was investigated in January following a report of gastroenteritis in 32 prisoners and 10 staff at a correctional facility. *S.* Typhimurium pulsed-field gel electrophoresis (PFGE) 3 was isolated from 8 faecal specimens. One suspect meal was identified, which included a potato salad made with a raw egg mayonnaise. The eggs were sourced from a prison farm. In February, the same farm reported a gastroenteritis outbreak involving 13 prisoners, with *S.* Typhimurium PFGE 3 isolated from 2 faecal specimens. In this outbreak, prisoners reported drinking raw egg milkshakes. In March, a 2nd outbreak occurred at a regional prison, involving 6 prisoners and 1 staff member. *S.* Typhimurium PFGE 3 was isolated from 2 faecal specimens. Prisoners again reported drinking raw egg milkshakes. A further 7 apparently sporadic cases of *S.* Typhimurium PFGE 3 were notified from 6 prisons. An inspection of the egg production facilities at the implicated farm found a basic operation, with no candling or routine cleaning of eggs. A chicken feed sample was positive for *S.* Typhimurium PFGE 3 but eggs sampled, and subsequent feed samples, were negative for *Salmonella*.

Multi-jurisdictional investigations

Between October 2014 and May 2015, an outbreak of hepatitis A associated with the consumption of a frozen mixed berry product occurred across multiple jurisdictions. This required a multi-jurisdictional response involving state health departments, OzFoodNet, public health reference laboratories, agriculture and food agencies. Although the majority of cases occurred in this quarter, the report of this investigation will be included in the 1 April to 30 June 2015 (2nd quarter) OzFoodNet national quarterly report.

Cluster investigations

During this quarter, OzFoodNet sites conducted investigations into 54 outbreaks and clusters for which no common food vehicle or source of infection could be identified. Aetiological agents that were able to be identified during the investigations included *S.* Typhimurium (for 7 clusters), *Shigella* spp. (for 2 clusters), and *S.* Agona, *S.* Kiambu, *S.* Newport, *S.* Saintpaul and rotavirus (for 1 cluster each).

Comments

During this quarter, 891 people were affected by foodborne or suspected foodborne outbreaks. This is a 24% increase on the number affected in the 1st quarter of 2014 (721) and a 65% increase on the 5-year mean (2010–2014) for this quarter (539). Outbreaks in Queensland accounted for 66% (584) of the total number of people affected by foodborne outbreaks; 96% (558) of these were affected in 13 outbreaks of *S.* Typhimurium. The 2 largest of these outbreaks, which affected 140 people attending a conference and 138 people at a restaurant respectively, are discussed under Queensland's description of key outbreaks.

In this quarter, *S.* Typhimurium was the aetiological agent for 81% (13/16) of the foodborne outbreaks for which an analytical and/or microbiological link to a food vehicle was established. Seventy-seven per cent (10/13) of the *S.* Typhimurium outbreaks were associated with the consumption of raw or minimally cooked egg dishes. These outbreaks affected 516 people, which was 94% (516/550) of the total affected by *S.* Typhimurium outbreaks in the quarter.

Cross contamination between raw eggs or raw egg products and ready-to-eat food items can occur when equipment, such as stick or other blenders, are not properly cleaned and sanitised between processing of these food types. In this quarter, blending equipment was associated with 4 outbreaks of *S.* Typhimurium (2 in Queensland, 1 in South Australia and 1 in Victoria), affecting 2,518 people. Cross contamination is a risk when using stick mixers or blenders to process raw foods such as eggs, due to the potential contamination of internal components and the difficulty in being able to thoroughly clean them. Blenders and stick mixers should be regularly dismantled, cleaned and sanitised.[2] In addition, one piece of mixing equipment should be allocated for raw high risk foods (such as cakes/pancake batter and raw egg deserts) and another separate piece of mixing equipment for ready-to-eat foods that will not be cooked after the blending step (such as whipped cream).

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Appendix: Outbreaks of foodborne or suspected foodborne disease reported by OzFoodNet sites,* 1 January to 31 March 2015 (n=47)

State or territory	Month†	Setting prepared	Agent responsible	Number affected‡	Number lab confirmed	Number hospitalised‡	Evidence	Responsible vehicles
NSW	Jan	Restaurant	*Salmonella* Typhimurium MLVA 03-09-08-12-523	3	1	0	D	Tiramisu
NSW	Jan	Restaurant	Unknown	3	0	0	D	Unknown
NSW	Jan	Aged care facility	*S.* Bovismorbificans	33	33	12	AM	Baked dessert items
NSW	Jan	Restaurant	*S.* Typhimurium MLVA 03-12-11-14-523	12	11	0	D	Menu items containing undercooked egg
NSW	Jan	Restaurant	*S.* Typhimurium MLVA 03-12-11-14-523	2	2	0	D	Suspected raw egg sauces
NSW	Jan	Restaurant	*S.* Typhimurium MLVA 03-12-13-09-523	13	13	3	D	Unknown
NSW	Feb	Restaurant	*S.* Typhimurium MLVA 03-09-07-12-523 and 03-12-12-09-523	5	3	0	D	Unknown
NSW	Feb	Takeaway	Unknown	30	0	Unknown	D	Vietnamese style chicken and salad rolls
NSW	Feb	Takeaway	Histamine fish poisoning	7	0	0	M	Canned tuna
NSW	Feb	Restaurant	*S.* Virchow	3	2	1	D	Menu items containing undercooked egg
NSW	Feb	Restaurant	*S.* Typhimurium MLVA 03-10-08-12-523	9	3	0	D	Unknown
NSW	Mar	Community event	*S.* Typhimurium PT 12A	4	3	3	D	Pancake batter containing eggs
NSW	Mar	Restaurant	*S.* Typhimurium MLVA 03-24-14-10-523	7	7	0	D	Unknown
NSW	Mar	Restaurant	*Salmonella* spp. (PCR only)	5	1	1	D	Tiramisu
NSW	Mar	Restaurant	*S.* Typhimurium MLVA 03-09-07-13-523	4	4	0	D	Menu items containing undercooked egg
Qld	Jan	Restaurant	*S.* Typhimurium MLVA 03-12-11-12-523	138	95	Unknown	AM	Deep fried ice-cream
Qld	Jan	Restaurant	*S.* Typhimurium MLVA 03-12-12-12-523	16	11	0	AM	Deep fried ice-cream
Qld	Jan	Unknown	*S.* Typhimurium MLVA 03-12-11-12-523	85	44	Unknown	M	Kimbap style sushi
Qld	Jan	Aged care facility	*S.* Typhimurium MLVA 03-12-11-12-523	4	2	1	M	Suspected egg dish
Qld	Jan	National franchised fast food restaurant	*S.* Typhimurium PT U307, MLVA 03-12-11-12-523	48	46	14	M	Chocolate mousse
Qld	Jan	Primary produce	Ciguatera fish poisoning	2	0	0	D	Cod
Qld	Feb	Restaurant	*S.* Typhimurium MLVA 03-12-13-09-523	17	9	2	A	Roast duck
Qld	Feb	Restaurant	*S.* Typhimurium MLVA 03-12-13-09-523	3	3	0	D	Lamb tartare with raw egg
Qld	Feb	Private residence	*S.* Typhimurium MLVA 03-12-10-12-523	6	6	1	D	Sushi
Qld	Feb	Restaurant	Norovirus GII	18	11	0	D	Unknown
Qld	Feb	Restaurant	*S.* Typhimurium MLVA 03-12-12-09-523	7	7	1	D	Eggs Benedict
Qld	Feb	Other	*S.* Typhimurium MLVA 03-12-12-09-523	30	11	3	D	Unknown
Qld	Feb	Other	*S.* Typhimurium MLVA 03-12-12-09-523	140	140	9	AM	Rum and raisin bread cake with custard
Qld	Mar	Restaurant	*S.* Typhimurium MLVA 03-17-09-11-523	44	31	23	A	Eggs Benedict
Qld	Mar	Primary produce	Ciguatera fish poisoning	6	0	1	D	Spanish mackerel
Qld	Mar	Private residence	*S.* Typhimurium MLVA 03-12-10-11-523	20	6	4	M	Chicken long soup (with egg and chicken)

Appendix *continued*: Outbreaks of foodborne or suspected foodborne disease reported by OzFoodNet sites,* 1 January to 31 March 2015 (n=47)

State or territory	Month†	Setting prepared	Agent responsible	Number affected‡	Number lab confirmed	Number hospitalised‡	Evidence	Responsible vehicles
SA	Feb	Restaurant	*S.* Typhimurium PT 135, MLVA 03-12-09-11-523	7	7	1	D	Raw egg sun-dried tomato aioli
SA	Feb	Restaurant	*S.* Typhimurium PT 135a, MLVA 03-12-12-09-523	3	2	0	D	Raw egg aioli
SA	Feb	Restaurant	*S.* Typhimurium PT 135a, MLVA 03-13-10-10-523	2	2	0	D	Unknown
SA	Mar	Restaurant	*S.* Typhimurium PT 9, MLVA 03-24-13-10-523	7	7	2	M	Contaminated stick blender
SA	Mar	Restaurant	*S.* Typhimurium PT 9, MLVA 03-15-06-11-550 and MLVA 03-24-13-10-523	7	7	2	D	Eggs; poached, scrambled or omelette
SA	Mar	Takeaway	*S.* Typhimurium PT 9, MLVA 03-14-08-11-550	4	4	1	D	Unknown
Vic.	Jan	Aged care facility	*Clostridium perfringens*	4	1	0	D	Unknown
Vic.	Feb	Restaurant	*S.* Typhimurium PT 9	18	13	5	M	Chicken and raw egg mayonnaise mixture
Vic.	Feb	Aged care facility	*S.* Typhimurium PT 135a, MLVA 03-11-11-16-525	7	7	0	D	Vitamised food suspected
Vic.	Mar	Restaurant	*S.* Typhimurium PT 135a	3	2	0	M	Raw egg mayonnaise
Vic.	Mar	Restaurant	*S.* Typhimurium PT 135a	10	3	1	A	Pork dish
Vic.	Mar	Takeaway	*S.* Typhimurium PT 135a	6	6	6	D	Chicken suspected
WA	Jan	Restaurant	Norovirus	9	1	0	A	Multiple salads
WA	Jan	Institution – not otherwise specified	*S.* Typhimurium PFGE 3, MLVA 03-11-15-10-523	69	31	4	D	Raw egg mayonnaise and raw egg milkshakes
WA	Mar	Commercial caterer	Norovirus	7	1	0	D	Unknown
WA	Mar	Takeaway	*S.* Typhimurium PFGE 13, MLVA 05-04-14/15-11-490	4	4	0	D	Premade sandwiches
Total				891	602	101		

* No foodborne outbreaks were reported in the Australian Capital Territory, the Northern Territory or Tasmania, during the quarter.

† Month of outbreak is the month of onset of the first case or month of notification of the first case or month the investigation of the outbreak commenced.

‡ 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. The number of people affected does not necessarily equal the number of laboratory-confirmed cases.

A Analytical epidemiological association between illness and one 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.

MLVA Multi-locus variable number tandem repeat analysis.

PFGE Pulsed-field gel electrophoresis.

PT Phage type.

National Notifiable Diseases Surveillance System, 1 January to 31 March 2017

A summary of diseases currently being reported by each jurisdiction is provided in Table 1. There were 74,389 notifications to the National Notifiable Diseases Surveillance System (NNDSS) between 1 January and 31 March 2017 (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
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
Paratyphoid	All jurisdictions
Shiga toxin/verotoxin-producing *Escherichia coli*	All jurisdictions
Salmonellosis	All jurisdictions
Shigellosis	All jurisdictions
Typhoid fever	All jurisdictions
Quarantinable diseases	
Avian influenza in humans	All jurisdictions
Cholera	All jurisdictions
Middle East respiratory syndrome coronavirus	All jurisdictions
Plague	All jurisdictions
Rabies	All jurisdictions
Severe acute respiratory syndrome	All jurisdictions
Smallpox	All jurisdictions
Viral haemorrhagic fever	All jurisdictions
Yellow fever	All jurisdictions
Sexually transmissible infections	
Chlamydial infection	All jurisdictions
Donovanosis	All jurisdictions
Gonococcal infection	All jurisdictions
Syphilis < 2 years duration	All jurisdictions
Syphilis > 2 years or unspecified duration	All jurisdictions
Syphilis - congenital	All jurisdictions

Table 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
Rotavirus	All jurisdictions except ACT and Victoria
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 except Australian Capital Territory
Dengue virus infection	All jurisdictions
Flavivirus infection (unspecified)	All jurisdictions
Japanese encephalitis virus infection	All jurisdictions
Malaria	All jurisdictions
Murray Valley encephalitis virus infection	All jurisdictions
Ross River virus infection	All jurisdictions
West Nile/Kunjin virus infection	All jurisdictions
Zoonoses	
Anthrax	All jurisdictions
Australian bat lyssavirus infection	All jurisdictions
Brucellosis	All jurisdictions
Leptospirosis	All jurisdictions
Lyssavirus infection (NEC)	All jurisdictions
Ornithosis	All jurisdictions
Q fever	All jurisdictions
Tularaemia	All jurisdictions
Other bacterial infections	
Legionellosis	All jurisdictions
Leprosy	All jurisdictions
Meningococcal infection – invasive	All jurisdictions
Tuberculosis	All jurisdictions

NEC Not elsewhere classified.

Table 2: Notifications of diseases received by state and territory health authorities, 1 January to 31 March 2017, by date of diagnosis*

Disease	State or territory								Total 1st quarter 2017	Total 4th quarter 2016	Total 1st quarter 2016	Last 5 years mean 4th quarter	Ratio	Year to date 2017	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	3	2	13	3	0	18	2	41	36	43	47.4	0.9	41	47.4
Hepatitis B (unspecified)‡	23	597	18	257	48	9	429	147	1,528	1,406	1,563	1,578.6	1.0	1,528	1,578.6
Hepatitis C (newly acquired)†	1	8	0	64	11	2	24	36	146	176	201	161.2	0.9	146	161.2
Hepatitis C (unspecified)‡	36	1,046	53	548	103	50	471	290	2,597	2,806	2,886	2,506.2	1.0	2,597	2,506.2
Hepatitis D	0	3	0	2	3	0	2	3	13	15	17	13.0	1.0	13	13.0
Gastrointestinal diseases															
Botulism	0	0	0	0	0	0	0	0	0	0	0	0.6	0.0	0	0.6
Campylobacteriosis	157	NN	91	2,132	941	217	2,078	893	6,509	6,992	5,931	4,909.2	1.3	6,509	4,909.2
Cryptosporidiosis	50	738	30	715	126	5	348	242	2,254	944	2,318	1,571.6	1.4	2,254	1,571.6
Haemolytic uraemic syndrome	0	1	0	1	0	0	2	2	6	5	4	5.6	1.1	6	5.6
Hepatitis A	1	7	0	10	1	0	26	6	51	41	55	68.2	0.7	51	68.2
Hepatitis E	0	6	0	2	1	1	6	1	17	13	15	12.6	1.3	17	12.6
Listeriosis	2	4	0	5	0	1	9	1	22	21	27	24.0	0.9	22	24.0
Paratyphoid	1	7	0	4	0	0	9	3	24	17	34	29.4	0.8	24	29.4
STEC§	0	12	0	8	94	3	10	18	145	144	44	38.6	3.8	145	38.6
Salmonellosis	206	1,497	148	1,777	514	108	1,115	918	6,283	3,919	6,545	5,228.6	1.2	6,283	5,228.6
Shigellosis	0	56	52	91	6	5	157	43	410	358	367	263.8	1.6	410	263.8
Typhoid fever	0	24	1	9	3	0	20	1	58	25	37	49.8	1.2	58	49.8
Quarantinable diseases															
Avian influenza in humans	0	0	0	0	0	0	0	0	0	0	0	0.0	0.0	0	0.0
Cholera	0	0	0	0	0	0	0	0	0	1	0	0.2	0.0	0	0.2
Middle East respiratory syndrome coronavirus	0	0	0	0	0	0	0	0	0	0	0	0.0	0.0	0	0.0
Plague	0	0	0	0	0	0	0	0	0	0	0	0.0	0.0	0	0.0
Rabies	0	0	0	0	0	0	0	0	0	0	0	0.0	0.0	0	0.0
Severe acute respiratory syndrome	0	0	0	0	0	0	0	0	0	0	0	0.0	0.0	0	0.0
Smallpox	0	0	0	0	0	0	0	0	0	0	0	0.0	0.0	0	0.0
Viral haemorrhagic fever	0	0	0	0	0	0	0	0	0	0	0	0.0	0.0	0	0.0
Yellow fever	0	0	0	0	0	0	0	0	0	0	0	0.0	0.0	0	0.0

Table 2 *continued:* Notifications of diseases received by state and territory health authorities, 1 January to 31 March 2017, by date of diagnosis*

Disease	State or territory								Total 1st quarter 2017	Total 4th quarter 2016	Total 1st quarter 2016	Last 5 years mean 4th quarter	Ratio	Year to date 2017	Last 5 years YTD mean
	ACT	NSW	NT	Qld	SA	Tas.	Vic.	WA							
Sexually transmissible infections															
Chlamydial infection[‖,¶]	384	7,962	641	6,233	1,575	144	832	3,257	21,028	17,506	18,667	21,639.4	1.0	21,028	21,639.4
Donovanosis	0	0	0	0	0	0	0	0	0	0	0	0.2	0.0	0	0.2
Gonococcal infection[¶]	71	2,657	420	1,440	293	40	2,072	1,037	8,030	6,181	5,640	4,410.0	1.8	8,030	4,410.0
Syphilis <2 years duration[¶]	13	207	65	271	29	2	205	95	887	779	815	536.2	1.7	887	536.2
Syphilis >2 years or unspecified duration[‡,¶]	2	104	19	71	18	0	389	33	636	484	506	441.0	1.4	636	441.0
Syphilis – congenital	0	0	0	0	1	0	0	0	1	0	0	0.2	5.0	1	0.2
Vaccine preventable diseases															
Diphtheria	0	0	0	0	0	0	0	0	0	2	1	0.6	0.0	0	0.6
Haemophilus influenzae type b	0	2	0	1	0	0	0	0	3	4	4	3.0	1.0	3	3.0
Influenza (laboratory confirmed)	56	2,312	476	2,949	911	108	1,027	410	8,249	14,524	5,809	3,555.4	2.3	8,249	3,555.4
Measles	0	18	2	7	0	0	3	11	41	29	37	51.6	0.8	41	51.6
Mumps	0	34	22	69	27	0	16	8	176	106	338	114.0	1.5	176	114.0
Pertussis	119	1,603	41	300	474	6	533	279	3,355	5,471	5,892	4,634.0	0.7	3,355	4,634.0
Pneumococcal disease – invasive	2	79	5	41	27	7	58	27	246	385	183	208.8	1.2	246	208.8
Poliovirus infection	0	0	0	0	0	0	0	0	0	0	0	0.0	0.0	0	0.0
Rotavirus	NN	153	15	298	77	6	NN	113	662	838	795	662.0	668.8	669	1.0
Rubella	0	0	0	3	0	0	0	2	5	2	3	5.6	0.9	5	5.6
Rubella – congenital	0	0	0	0	0	0	0	0	0	0	0	0.0	0.0	0	0.0
Tetanus	0	1	0	0	0	0	0	0	1	1	3	1.6	0.6	1	1.6
Varicella zoster (chickenpox)	17	NN	24	48	100	12	17	167	385	728	591	480.8	0.8	385	480.8
Varicella zoster (shingles)	70	NN	95	19	567	95	11	461	1,318	1,704	1,794	1,421.0	0.9	1,318	1,421.0
Varicella zoster (unspecified)	43	NN	0	1,959	217	47	1,484	455	4,205	4,292	3,788	2,938.8	1.4	4,205	2,938.8
Vectorborne diseases															
Barmah Forest virus infection	0	17	7	64	1	0	10	11	110	68	127	520.4	0.2	110	520.4
Chikungunya virus infection	0	3	0	2	1	1	6	4	17	43	18	26.2	0.6	17	26.2
Dengue virus infection	11	95	12	124	21	2	60	68	393	317	747	664.0	0.6	393	664.0
Flavivirus infection (unspecified)	0	2	0	4	0	0	1	1	8	14	53	14.8	0.5	8	14.8
Japanese encephalitis virus infection	0	0	0	0	0	0	0	0	0	0	0	0.6	0.0	0	0.6
Malaria	1	21	7	43	2	0	25	22	121	74	71	88.2	1.4	121	88.2
Murray Valley encephalitis virus infection	0	0	0	0	0	0	0	0	0	0	0	0.4	0.0	0	0.4
Ross River virus infection	11	840	81	517	355	24	1,422	477	3,727	1,135	1,232	2,436.2	1.5	3,727	2,436.2
West Nile/Kunjin virus infection	0	0	0	0	0	0	0	0	0	0	0	0.0	0.0	0	0.0

Table 2 *continued*: Notifications of diseases received by state and territory health authorities, 1 January to 31 March 2017, by date of diagnosis

Disease	State or territory								Total 1st quarter 2017	Total 4th quarter 2016	Total 1st quarter 2016	Last 5 years mean 4th quarter	Ratio	Year to date 2017	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	0.0
Australian bat lyssavirus infection	0	0	0	0	0	0	0	0	0	0	0	0.2	0.0	0	0.2
Brucellosis	0	0	0	1	0	0	0	0	1	6	2	4.8	0.2	1	4.8
Leptospirosis	0	5	9	40	0	0	1	0	55	24	53	32.2	1.7	55	32.2
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	1	0	0	1	2	12	1	6.8	0.3	2	6.8
Q fever	0	48	0	65	3	0	2	4	122	145	148	130.8	0.9	122	130.8
Tularaemia	0	0	0	0	0	0	0	0	0	0	0	0.0	0.0	0	0.0
Other bacterial infections															
Legionellosis	0	27	1	5	14	2	21	9	79	92	83	91.4	0.9	79	91.4
Leprosy	0	0	0	2	0	0	0	1	3	6	4	2.2	1.4	3	2.2
Meningococcal infection – invasive**	0	16	2	22	5	4	16	8	73	81	45	33.8	2.2	73	33.8
Tuberculosis	6	115	7	81	10	0	97	30	346	388	323	313.4	1.1	346	313.4
Total	1,283	20,330	2,346	20,317	6,583	901	13,032	9,597	74,389	72,360	67,860			67,846	

* 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 began reporting hepatitis C newly acquired from 1 September 2016. Previous notifications are reported 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.

¶ 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.

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 January to 31 March 2017, 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	3.4	1.1	0.7	0.0	1.2	0.3	0.7
Hepatitis B (unspecified)§	24.1	32.1	30.2	22.0	11.6	7.1	29.6	23.3	26.3
Hepatitis C (newly acquired)‡	1.0	0.4	0.0	5.5	2.7	1.6	1.7	5.7	2.5
Hepatitis C (unspecified)§	37.7	56.3	88.9	47.0	24.9	39.7	32.5	45.9	44.8
Hepatitis D	0.0	0.2	0.0	0.2	0.7	0.0	0.1	0.5	0.2
Gastrointestinal diseases									
Botulism	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
Campylobacteriosis	164.6	NN	152.6	182.8	227.1	172.2	143.4	141.3	165.1
Cryptosporidiosis	52.4	39.7	50.3	61.3	30.4	4.0	24.0	38.3	38.9
Haemolytic uraemic syndrome	0.0	0.1	0.0	0.1	0.0	0.0	0.1	0.3	0.1
Hepatitis A	1.0	0.4	0.0	0.9	0.2	0.0	1.8	0.9	0.9
Hepatitis E	0.0	0.3	0.0	0.2	0.2	0.8	0.4	0.2	0.3
Listeriosis	2.1	0.2	0.0	0.4	0.0	0.8	0.6	0.2	0.4
Paratyphoid	1.0	0.4	0.0	0.3	0.0	0.0	0.6	0.5	0.4
STEC‖	0.0	0.6	0.0	0.7	22.7	2.4	0.7	2.8	2.5
Salmonellosis	216.0	80.6	248.2	152.4	124.0	85.7	76.9	145.3	108.3
Shigellosis	0.0	3.0	87.2	7.8	1.4	4.0	10.8	6.8	7.1
Typhoid fever	0.0	1.3	1.7	0.8	0.7	0.0	1.4	0.2	1.0
Quarantinable diseases									
Avian influenza in humans	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
Cholera	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
Middle East respiratory syndrome coronavirus	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
Plague	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
Rabies	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
Severe acute respiratory syndrome	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
Smallpox	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
Viral haemorrhagic fever	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
Yellow fever	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
Sexually transmitted infections									
Chlamydial infection¶,**	402.6	428.4	1,074.9	534.6	380.1	114.3	57.4	515.5	362.5
Donovanosis	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
Gonococcal infection**	74.4	143.0	704.3	123.5	70.7	31.7	143.0	164.1	138.4
Syphilis < 2 years duration**	13.6	11.1	109.0	23.2	7.0	1.6	14.1	15.0	15.3
Syphilis > 2 years or unspecified duration§,**	2.1	5.6	31.9	6.1	4.3	0.0	26.8	5.2	11.0
Syphilis – congenital	0.0	0.0	0.0	0.0	0.2	0.0	0.0	0.0	0.0
Vaccine preventable diseases									
Diphtheria	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
Haemophilus influenzae type b	0.0	0.1	0.0	0.1	0.0	0.0	0.0	0.0	0.1
Influenza (laboratory confirmed)	58.7	124.4	798.2	252.9	219.8	85.7	70.9	64.9	142.2
Measles	0.0	1.0	3.4	0.6	0.0	0.0	0.2	1.7	0.7
Mumps	0.0	1.8	36.9	5.9	6.5	0.0	1.1	1.3	3.0
Pertussis	124.8	86.3	68.8	25.7	114.4	4.8	36.8	44.2	57.8
Pneumococcal disease – invasive	2.1	4.3	8.4	3.5	6.5	5.6	4.0	4.3	4.2
Poliovirus infection	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
Rotavirus	NN	8.2	25.2	25.6	18.6	4.8	NN	17.9	11.4

Table 3 *continued*: Notification rates of diseases, 1 January to 31 March 2017, 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*									
Rubella	0.0	0.0	0.0	0.3	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	0.1	0.0	0.0	0.0	0.0	0.0	0.0	0.0
Varicella zoster (chickenpox)	17.8	NN	40.2	4.1	24.1	9.5	1.2	26.4	9.8
Varicella zoster (shingles)	73.4	NN	159.3	1.6	136.8	75.4	0.8	73.0	33.4
Varicella zoster (unspecified)	45.1	NN	0.0	168.0	52.4	37.3	102.4	72.0	106.6
Vectorborne diseases									
Barmah Forest virus infection	0.0	0.9	11.7	5.5	0.2	0.0	0.7	1.7	1.9
Chikungunya virus infection	0.0	0.2	0.0	0.2	0.2	0.8	0.4	0.6	0.3
Dengue virus infection	11.5	5.1	20.1	10.6	5.1	1.6	4.1	10.8	6.8
Flavivirus infection (unspecified)	0.0	0.1	0.0	0.3	0.0	0.0	0.1	0.2	0.1
Japanese encephalitis virus infection	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
Malaria	1.0	1.1	11.7	3.7	0.5	0.0	1.7	3.5	2.1
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	11.5	45.2	135.8	44.3	85.7	19.0	98.1	75.5	64.2
West Nile/Kunjin virus infection	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
Zoonoses									
Anthrax	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
Australia bat lyssavirus infection	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
Brucellosis	0.0	0.0	0.0	0.1	0.0	0.0	0.0	0.0	0.0
Leptospirosis	0.0	0.3	15.1	3.4	0.0	0.0	0.1	0.0	0.9
Lyssavirus infection (NEC)	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
Ornithosis	0.0	0.0	0.0	0.0	0.2	0.0	0.0	0.2	0.0
Q fever	0.0	2.6	0.0	5.6	0.7	0.0	0.1	0.6	2.1
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	1.5	1.7	0.4	3.4	1.6	1.4	1.4	1.4
Leprosy	0.0	0.0	0.0	0.2	0.0	0.0	0.0	0.2	0.1
Meningococcal infection – invasive††	0.0	0.9	3.4	1.9	1.2	3.2	1.1	1.3	1.3
Tuberculosis	6.3	6.2	11.7	6.9	2.4	0.0	6.7	4.7	6.0

* 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 began reporting hepatitis C newly acquired from 1 September 2016. Previous notifications are reported 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.

** 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 Meningococcal Surveillance Programme, 1 January to 31 March 2017

Monica M Lahra, Rodney P Enriquez for the for the National Neisseria Network, Australia

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*.

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Table: Number of laboratory confirmed cases of invasive meningococcal disease, Australia, 1 January to 31 March 2017, by serogroup and state or territory

State or territory	Year	Serogroup A Q1	A YTD	B Q1	B YTD	C Q1	C YTD	Y Q1	Y YTD	W Q1	W YTD	ND/other Q1	ND/other YTD	All Q1	All YTD
Australian Capital Territory	2017	0	0	0	0	0	0	0	0	0	0	0	0	0	0
	2016	0	0	0	0	0	0	0	0	0	0	0	0	0	0
New South Wales	2017	0	0	9	9	2	2	2	2	3	3	0	0	16	16
	2016	0	0	5	5	1	1	0	0	4	4	3	3	13	13
Northern Territory	2017	0	0	0	0	0	0	1	1	1	1	0	0	2	2
	2016	0	0	0	0	0	0	0	0	0	0	0	0	0	0
Queensland	2017	0	0	12	12	0	0	7	7	3	3	0	0	22	22
	2016	0	0	4	4	0	0	4	4	2	2	0	0	10	10
South Australia	2017	0	0	1	1	0	0	2	2	2	2	0	0	5	5
	2016	0	0	6	6	0	0	0	0	0	0	0	0	6	6
Tasmania	2017	0	0	1	1	0	0	0	0	4	4	0	0	5	5
	2016	0	0	0	0	0	0	0	0	2	2	0	0	2	2
Victoria	2017	0	0	4	4	0	0	4	4	7	7	1	1	16	16
	2016	0	0	3	3	1	1	0	0	5	5	0	0	9	9
Western Australia	2017	0	0	3	3	1	1	0	0	4	4	1	1	9	9
	2016	0	0	1	1	0	0	0	0	2	2	0	0	3	3
Total	2017	0	0	30	30	3	3	16	16	24	24	2	2	75	75
	2016	0	0	19	19	2	2	4	4	15	15	3	3	43	43