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Editorial

The importance of enteric infections in Australia

Martyn D Kirk

This issue of *Communicable Diseases Intelligence* contains several reports highlighting the importance of enteric infections. Infections spread via the faecal-oral route result in significant social and economic costs, regardless of a country's level of industrialisation.[1] In Australia, the burden of disease transmitted by contaminated food was estimated to cost AUD$1.2 billion annually.[2] Data from the OzFoodNet network (www.ozfoodnet.gov.au) was critical to estimating these costs, which are largely driven by lost productivity due to people taking time off work as a result of their own illness or to care for someone else who was ill. OzFoodNet is a national network of epidemiologists, which has dramatically improved public health action for enteric infections.[3] A simple example of how OzFoodNet has improved the surveillance of enteric infections is shown in the number of outbreaks of gastroenteritis and foodborne disease reported over time. In 2002, OzFoodNet reported 513 foodborne and gastrointestinal outbreaks compared with 1,640 outbreaks in 2010, indicating a much-matured surveillance system.[4,5]

Rotavirus is a common cause of gastroenteritis and one of the most significant enteric pathogens globally due to the resulting high mortality in young children, particularly in low-income countries.[6] Various studies have highlighted the impact that the introduction of rotavirus vaccines into the Australia immunisation schedule has had on public health, which is reinforced in the article by David and Kirk in this issue.[7–9] Also in this issue is a report of molecular surveillance of rotavirus in Australia that shows a dynamic pattern of circulating wild-type strains, highlighting the importance of prospective surveillance to monitor the epidemiology of rotavirus in a post-vaccine era.[10]

Enteric infections often manifest as gastroenteritis consisting of vomiting and diarrhoea, but may also result in more serious outcomes, such as hepatitis, meningitis, or bacteraemia. Determining the mode of transmission of enteric agents is often challenging, as there are usually multiple means of transmitting illness.[11] For example, norovirus is one of the most common causes of infectious gastroenteritis globally and is highly-infectious.[12] The virus may be transmitted by contaminated food, water, or from contact with another infected person or contaminated fomites.[13] The two reports of outbreaks of norovirus associated with oysters in northern New South Wales and Tasmania respectively, highlight the risks that occur when filter-feeding molluscs are grown in water contaminated by human sewage.[14,15] In Australia, oyster related outbreaks are rare due to the safeguards instituted by industry and government, although they can still occur where there are breakdowns in sanitation, which occurred in these two outbreaks. Data from the OzFoodNet network illustrate that most outbreaks of norovirus are spread from one infected person to another, particularly in institutionalised settings.[5,16] It is likely that vaccines against noroviruses will be produced in the future due to the potential economic and public health benefits.[17]

The investigation of hepatitis A infections transmitted by sharing Kava on page E26 demonstrates the highly infectious nature of the virus.[18] In recent years, the incidence of hepatitis A infections in Australia has declined significantly.[19] Many cases of hepatitis A reported to health departments in Australia are in travellers returning from overseas, including Pacific Island countries and territories (http://www.health.nsw.gov.au/Infectious/alerts/Documents/Hepatitis_A_Alert_8March2013.pdf).[20,21] The three additional cases (along with an additional tertiary case) of hepatitis A that occurred in this cluster after sharing kava with the primary case while they were infectious provides some clues as to how people acquire infection when travelling to Pacific Island countries and territories and illustrates the need for vaccination for travellers to the region.[22] While the incidence of locally-acquired cases in Australia is low, the outbreak due to imported semi-dried tomatoes in 2008–09 highlights that Australia is vulnerable to large and serious foodborne outbreaks of hepatitis A.[23]

Finally, this issue also includes two articles highlighting the public health response to clusters of two important bacterial infections due to *Salmonella* Typhi in a family and *Campylobacter* associated with duck livers.[24,25] In particular, the outbreak of campylobacteriosis is very similar to other previously reported outbreaks where food

premises have served undercooked poultry livers in Australia and overseas.[26–28] The short incubation period demonstrates that the cooking process was inadequate and that affected persons were likely to have received large doses of *Campylobacter* from the contaminated dish.

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Original article

Rotavirus gastroenteritis hospitalisations following introduction of vaccination, Canberra

Rosemary L David, Martyn D Kirk

Abstract

Objectives: To determine the effect of rotavirus vaccination on rotavirus hospitalisations in children under 5 years of age at The Canberra Hospital, Australian Capital Territory.

Methods: Rotavirus hospitalisations in children under 5 years of age at the Canberra Hospital were identified through a retrospective clinical audit of electronic medical hospitalisations in the pre-vaccine (2004–2006) and post-vaccine (2008–2012) periods. Records and confirmation with rotavirus pathology results were compared using MS Excel and Stata.

Results: Laboratory confirmed rotavirus infections resulted in 289 children being admitted to the Canberra Hospital between January 2004 and December 2012. Hospitalisation for rotavirus gastroenteritis decreased by 76% in the 5 years following vaccine introduction compared with pre-vaccine periods. Seasonal patterns of hospitalisation were prominent in pre-vaccine periods but were attenuated post-vaccine. The greatest decreases in hospitalisation between pre– and post-vaccine periods were observed in the 12–23 (80%) and 24–35 (88%) month age categories. Decreases in hospitalisation were reported for patients unlikely to have received vaccine cover at that time, indicating an indirect protective effect of rotavirus vaccine.

Conclusions: This study reports significant reductions in rotavirus hospitalisation of children under 5 years of age at The Canberra Hospital following vaccine introduction, mid-2007. These findings support rotavirus vaccination as an effective measure to reduce hospitalisation in children under 5 years of age. *Commun Dis Intell* 2014;38(1):E3–E8.

Introduction

Rotavirus is the most common causative agent of severe acute gastroenteritis in childhood worldwide.[1] Each year over half a million deaths occur globally due to rotavirus gastroenteritis, with the majority occurring in developing nations.[1,2] While most infections in Australia do not result in death there is still substantial morbidity.[2] Before the introduction of vaccination an estimated 10,000 hospitalisations, 22,000 emergency department visits and 115,000 general practitioner visits due to rotavirus occurred annually in children under 5 years of age.[3] These amounted to a total estimated annual cost of 30 million dollars in Australia.[3]

In July 2007 rotavirus vaccination was introduced to the National Immunisation Program (NIP).[4,5] Two oral, live-attenuated vaccines covering prevalent rotavirus genotypes were registered: Rotarix™ (GlaxoSmithKline) and RotaTeq™ (CSL Limited/Merck and Co inc).[5] Rotarix™ is a monovalent (G1P1A[8] strain) vaccine strain given in 2 doses at 2 and 4 months, and has demonstrated effectiveness against a wide range of other strains.[5] RotaTeq™ is a pentavalent vaccine containing reassortants of G1, G2, G3, G4 and P1A[8] human strains and bovine strains G6 and P7, and is given in 3 doses at 2, 4 and 6 months.[5] Both vaccines have upper age limits: the Rotarix™ course must be completed by 25 weeks of age and RotaTeq™ by 33 weeks.[5] The type of vaccine used in publicly funded programs varies by state, with Rotarix™ being used in the Australian Capital Territory.[5] The Australian Capital Territory typically records some of the highest vaccination coverage rates in Australia.[6] In the Australian Capital Territory in 2010, completed course coverage for rotavirus vaccine at 12 months of age was 88%.[4]

Several studies report significant declines in the burden of rotavirus gastroenteritis following the introduction of vaccination.[7–11] Hospitalisations for rotavirus gastroenteritis at a tertiary paediatric centre in Westmead, Sydney decreased by 75% in the 2 seasons following vaccination.[11] In South Australia, an 83% reduction in rotavirus gastroenteritis coded admissions was seen in a 2 year period following vaccine implementation.[8] Similar declines in rotavirus hospitalisation were also observed at the Royal Children's Hospital, Melbourne and in state-wide analysis of hospitalisations in Queensland.[7,10] Analysis of pre– and post-vaccine periods, found a 71% decline in rotavirus-coded hospitalisations of children under 5 years of age in New South Wales, Victoria, Queensland, Western Australia, and South Australia.[9] Studies

have indicated that herd immunity was likely to have led to declines in rotavirus cases being seen in children who were ineligible for vaccination due to the upper age limits.[7–11]

Prior to the introduction of the vaccine implementation, clinical trials reported that 85%–100% of hospitalisations due to rotavirus gastroenteritis could be prevented in the 1st year following infant vaccination.[12,13] The cost-effectiveness of vaccination relies on large declines in cases of rotavirus gastroenteritis, particularly in severe cases requiring hospitalisation.[14,15] The current evidence confirms large decreases in morbidity associated with rotavirus infection. It is important to continue surveillance for rotavirus infection to monitor the epidemiology of infections and ensure that new strains do not emerge.

The aim of this study was to determine the effect of rotavirus vaccination on rotavirus gastroenteritis hospitalisations in children under 5 years of age at The Canberra Hospital (TCH), a major tertiary hospital servicing the Canberra region.

Methods

A retrospective clinical audit of electronic patient records and pathology data was conducted at TCH. Records were requested for hospital admissions occurring between January 2004 and December 2012 in children under 5 years of age that were coded as ‘rotavirus enteritis’, A08.0 according to the International Classification of Diseases, 10th Revision, Australian Modification (ICD-10-AM).

Simultaneously, the TCH pathology department was asked for the records of all children under 5 years of age who tested positive for rotavirus from 2004–2012. The following information for each patient with positive rotavirus pathology: patient name, hospital identification number, place of specimen collection, date of specimen collection and date of positive test result was requested.

From the TCH patient records, the following information: a unique patient identifier, hospital identification codes, gender, date of birth, date of onset of illness, date of admission, date of rotavirus detection, date of discharge, immunisation status and primary diagnosis at discharge was recorded. Length of stay and age at admission were calculated to compare pre– and post-vaccine periods. Patients admitted and discharged on the same day were allocated a length of stay of 1 day (http://meteor.aihw.gov.au/content/index.phtml/itemId/269422).

A rotavirus case-patient was defined as someone with both a hospital record coded for rotavirus and laboratory evidence of a positive test result. Only cases where the specimen collection data for a pathology result was between the date of admission and discharge was included, to ensure the positive result identified was related specifically to the rotavirus coded admission. Patients with multiple positive rotavirus pathology results corresponding to a single admission were counted only once.

As the Rotarix™ vaccine was introduced to the vaccine schedule in mid-2007 the pre-vaccine period was defined as 2004–2006 and the post-vaccine period as 2008–2012. The pre-vaccine period was used as a baseline for comparison of different characteristics of case patients, including: ages of patients, male to female ratio and days of hospitalisation. Means and medians were compared for continuous data and proportions for categorical data. $P<0.05$ was considered as statistically significant. Trends in the number of hospitalisations for all children under 5 years, number of hospitalisations for specified age categories and seasonality of admissions were examined.

The ACT Health Human Research Ethics Committee (ACTH-HREC) approved this study. Data were analysed and stored using Microsoft Excel 2010, and Stata 12.1.

Results

Two hundred and ninety-nine patient records with rotavirus coded admissions were received from the TCH medical records department. Following pathology data matching, 10 patients for whom there was no rotavirus positive pathology test associated with admission were excluded.

There were 289 admissions in children under 5 years of age from January 2004 until December 2012 that met the study criteria (Table). Of these, 52% (151/289) of case patients were male.

There was a substantial drop in the number of rotavirus hospitalisations each year at TCH in children under 5 years following the introduction of vaccination in mid-2007 (Figure 1). Pre-vaccine, there was a mean of 62 rotavirus hospitalisations per year in children under 5 years at TCH. In 2007 the vaccine was introduced mid-year. Thereafter hospitalisations declined 53% compared with the mean hospitalisations for the pre-vaccine period. For the post-vaccine period, there was an average decrease in the number of hospitalisations of 76% compared with the pre-vaccine period. In 2012, only 7 hospitalisations were recorded, representing an 89% decrease from the mean hospitalisation in the pre-vaccine period.

The total number of days of hospitalisation per year declined significantly from pre– to post-vaccine

Table: Rotavirus hospitalisations in children less than 5 years of age, Canberra Hospital, 2004 to 2012

Year	Rotavirus hospitalisations	Days of hospitalisation	Median days of hospital stay (range)	Proportion male (%)	Mean age at admission (months)
2004	40	102	2 (1–14)	61	20.8
2005	56	139	2 (1–15)	43	20.8
2006	90	224	2 (1–23)	46	20.7
2007	29	124	2 (1–23)	62	19.8
2008	15	95	2 (1–44)	40	17.9
2009	18	36	2 (1–4)	56	28.6
2010	17	47	2 (1–12)	50	16.8
2011	17	75	3 (1–16)	39	23.9
2012	7	37	5 (1–16)	57	11.6
Total	289	879	2 (1–44)	52	20.7

Figure 1: Rotavirus hospitalisations in children less than 5 years of age, Canberra Hospital, 2004 to 2012, by year

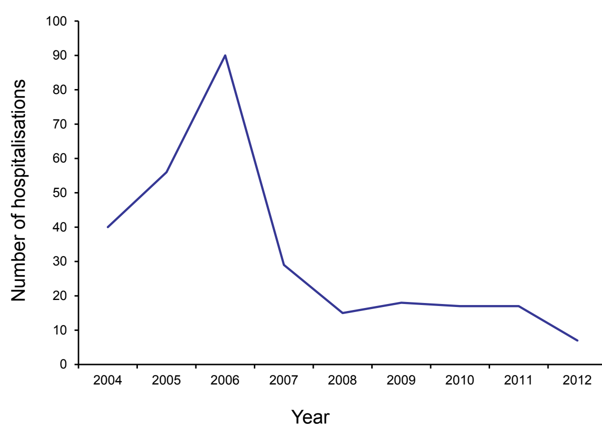
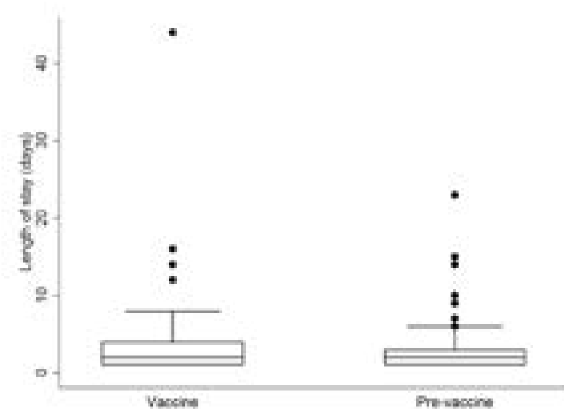


Figure 2: Length of stay for rotavirus hospitalisations in pre-vaccine (2004–2006) and post-vaccine (2008–2012) periods at the Canberra Hospital, Canberra



periods. In the pre-vaccine period, rotavirus resulted in a mean of 155 days of hospitalisation per year, compared with a mean of 58 days of hospitalisations per year in the post-vaccine period.

The median length of stay for rotavirus gastroenteritis in the pre-vaccine period was 2 days (range 1–23), compared with 2 days (range 1–44) for the post vaccination period (Figure 2). In the pre-vaccine period 93% (172/185) of cases had rotavirus recorded as the primary diagnosis compared with 72% (51/57) post-vaccine ($P<0.001$).

In the pre-vaccine period there was a clear pattern of seasonality related to rotavirus hospitalisations, with 39% of admissions in winter and 46% in spring. In the post-vaccine period, the winter–spring predominance was maintained with 32% and 36% of hospitalisations in these seasons respectively. However, the seasonal pattern of hospitalisations was weaker in the post-vaccine period due to small numbers (Figure 3). The proportion of hospitalisations occurring in summer increased from 4% to 19% and the number of hospitalisations did not decline between pre– and post-vaccine periods.

The mean age at admission pre-vaccine was 20.8 months. This was not found to be significantly different from the mean age post-vaccine of 19.7 months ($P=0.87$). In both periods, the highest proportion of hospitalisations occurred in children who were 12–23 months of age, with 42% and 35% in the pre– and post-vaccination periods respectively.

Declines in the number of hospitalisations in the post-vaccine period in all age categories were observed, particularly in children aged 12–23 (81%) and 24–35 (88%) months (Figure 4). In children younger than 12 months of age, there was an annual mean of 13.7 hospitalisations in

Figure 3: Mean annual hospitalisations for rotavirus pre-vaccine (2004–2006) and post-vaccine (2008–2012) periods at the Canberra Hospital, Canberra, by season

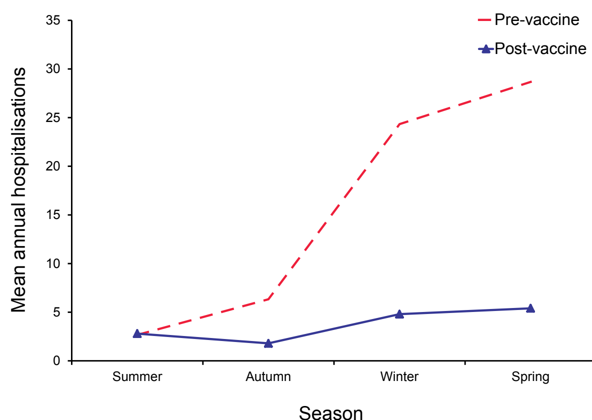
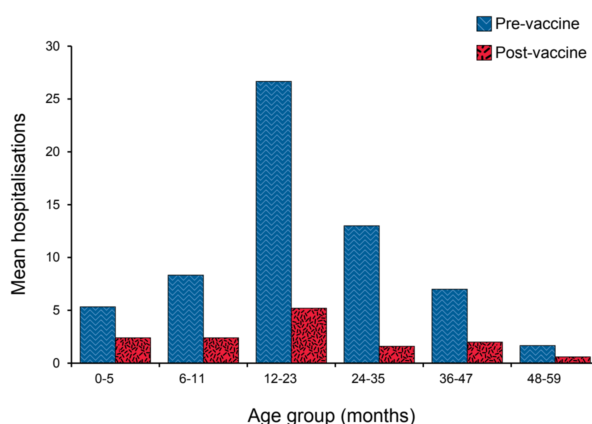


Figure 4: Mean number of hospitalisations for rotavirus pre-vaccine (2004–2006) and post-vaccine (2008–2012) periods, the Canberra Hospital, Canberra, by age at admission



the pre-vaccine period, compared with 4.8 hospitalisations in the post-vaccine period. In 2007, the number of hospitalisations of children aged 24–35 and 36–47 months decreased from the mean pre-vaccine number of hospitalisations by 85% and 43% respectively, despite children not being eligible for vaccination at that time.

Discussion

In this study examining the effect of rotavirus vaccination on rotavirus gastroenteritis hospitalisations in children in the Australian Capital Territory, a 76% reduction in rotavirus-coded hospitalisations was observed between pre-vaccine (2004–2006) and post-vaccine (2008–2012) periods at The Canberra Hospital. Despite natural fluctuations in disease activity, decreases were consistently above 71% throughout the 5 year period post-vaccination. The total number of days of hospitalisation declined significantly from 155 days pre-vaccine to 58 days post-vaccine. Decreases in the number of children hospitalisations in all age groups was observed, with the largest declines seen in the 12–23 and 24–35 month age categories. The declines observed were consistent with high rotavirus vaccine coverage in the Australian Capital Territory.

The magnitude of decrease in hospitalisation following rotavirus vaccination in the Australian Capital Territory is consistent with that reported for other Australian states. Studies have consistently shown decreases in rotavirus hospitalisation of over 70% from pre– to post-vaccine periods.[7–11] One study analysed a national database of hospitalisations and showed 71% decreases in rotavirus hospitalisation post-vaccine in Queensland, South Australia, New South Wales, Victoria and Western Australia.[9]

Prior to the addition of the rotavirus vaccine to the publicly funded NIP, detailed cost-effectiveness analysis was carried out.[14,15] There were an estimated 10,000 rotavirus hospitalisations each year in Australia between July 1998 and June 2003, which represented a significant economic and social burden.[3] Justification for funding the vaccine relied on significant declines in hospitalisations, contributing substantial healthcare savings. This study strengthens the existing evidence for the cost-effectiveness of rotavirus vaccination through large declines in rotavirus hospitalisation.

In this study, hospitalisations occurring in 2007 were analysed separately as vaccine was introduced mid-2007. A 53% decrease in hospitalisation between the pre-vaccine period and 2007 was observed, despite the likelihood that only a small proportion of children would have been immunised in that year. Vaccine was introduced mid-year and the upper age limits for Rotarix™ vaccine excluded most children. The proportion of children who received the vaccine in 2007 was unable to be determined.

A 65% reduction in the number of hospitalisations of children under 12 months of age for rotavirus was observed. This is important as rotavirus infection is more likely to be severe or complicated, requiring intensive treatment, in this younger age category. Closer examination of hospitalisations in the 24–35 and 36–47 month categories show declines in hospitalisation as early as 2007. These children were unlikely to have been eligible for vaccination in 2007, even accounting for early vaccine availability in the private market. These findings support the plausibility of rotavirus vaccination having an indirect protective effect for those

not receiving vaccine.[16] This effect was observed in several other states including Queensland, New South Wales, South Australia and Victoria and early evidence shows protection extending to individuals older than 5 years.[7–11] The size of the indirect protective effect of vaccine was unable to be evaluated without accurate recording of rotavirus specific vaccination status on hospital records.

Prominent seasonal patterns of rotavirus admissions in the pre-vaccine period consistent with patterns previously documented in the Australian Capital Territory were observed.[17] In the post-vaccine period, the seasonal pattern of hospitalisations was weakened, although spring–winter predominance was maintained. These findings are consistent with attenuated winter and spring hospitalisations observed in New South Wales post-vaccine.[11] Alterations to patterns of seasonality may have important consequences for health service planning including resource allocation. Interestingly, no decrease in hospitalisations occurring in summer months post-vaccine compared with pre-vaccine were seen. It is possible that patient cases occurring in summer months are fundamentally different from those in the spring–winter months. Summer infection is perhaps occurring in individuals with impaired immunity or may be related to a strain that is not covered by the current vaccine.

It was found that rotavirus coded hospital records were a good predictor of true rotavirus status and only 3% of rotavirus coded records could not be verified with positive pathology. It is unlikely that this analysis included all rotavirus hospitalisations at TCH as a proportion would not receive rotavirus coding due to inaccurate coding, false negative results or the absence of rotavirus stool tests being carried out. Several studies have documented declines in acute gastroenteritis coded hospitalisations between pre– and post-vaccine periods, suggesting a proportion is likely to be due to rotavirus. Decreases in rotavirus-coded hospitalisations likely underestimate true declines in hospitalisations due to rotavirus.

Several recent Australian studies have reported decreased nosocomial transmission of rotavirus following vaccine introduction. The study aimed to determine whether a case was likely to be nosocomial using the primary and secondary diagnosis on the patient discharge summary. It was found that recording of primary and secondary diagnoses varied in accuracy, which prevented meaningful analysis of potential nosocomial transmission. In both pre– and post-vaccine periods there were a number of hospitalisations with long lengths of stay, which were potentially nosocomial, however, other cases with shorter lengths of stay could not be as easily identified.[11]

There may have been changes in the hospital catchment area or total numbers of children under 5 years being serviced by TCH during our study, although it is unlikely that this would have altered significantly from 2004 to 2012. Regardless, the large decline in hospitalisations at TCH still represents a significant economic and resource saving for the hospital. It was also unclear whether changes occurred in the testing patterns for rotavirus following presentation with diarrhoea. This may have changed the proportion of all rotavirus cases being coded as rotavirus between 2004 and 2012. It is likely that any background changes in testing would have had only small impacts on the declines observed.

This study has shown large decreases in rotavirus hospitalisation of children under 5 years of age at TCH following introduction of rotavirus vaccine. Evidence of an indirect protective effect of vaccine extending to those who did not receive vaccine cover was reported. The findings of this study support childhood vaccination as an effective strategy to reduce economic and social burden associated with rotavirus infection. Further research could include investigation of hospitalisation for acute gastroenteritis pre– and post-vaccine to further illustrate vaccine efficacy. It would be useful to investigate the effect of vaccine introduction on nosocomial transmission in the hospital setting. The emergence of new strains of rotavirus is an ongoing possibility and it is important to continue observing paediatric hospitalisations over longer periods of time.

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An outbreak of norovirus genogroup II associated with New South Wales oysters

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Abstract

Introduction: Currently available antigen tests for norovirus (NoV) have excellent specificity but negative results do not always rule out infection. Real-time reverse transcription polymerase chain reaction (RT-PCR) is a useful method for detecting and genotyping NoV in humans and oysters. An outbreak of NoV associated with oyster consumption in northern New South Wales confirmed the value of real-time RT-PCR where immunochromatography (ICT) tests were negative.

Methods: Eight cases of gastrointestinal illness in northern NSW, clinically suggestive of NoV infection, were associated with consumption of oysters. A joint environmental investigation was conducted by the New South Wales Food Authority and local council. One human sample was collected and tested for NoV using ICT and real-time RT-PCR. Oyster samples were tested for NoV utilising real-time RT-PCR.

Results: The patient with a stool sample had NoV genogroup II (GII) confirmed by real-time RT-PCR after testing negative by ICT. Illness in all cases was consistent with NoV with median incubation and duration of 36 and 50.5 hours respectively. All cases consumed oysters that were harvested from the same area. Three oyster samples from the harvest area were also positive for NoV GII. A nearby leaking sewer line was identified as the likely source of the contamination with hydrological studies confirming its potential to contaminate implicated oyster leases.

Conclusion: This investigation confirmed the value of real-time RT-PCR testing of human specimens where ICT tests are negative and clinical illness is suggestive of NoV infection. NoV real-time RT-PCR and epidemiological evidence effectively linked human infection with oyster contamination to motivate a thorough environmental investigation and appropriate action to mitigate further public health risk. *Commun Dis Intell* 2014;38(1):E9–E15.

Keywords: oyster, outbreak, norovirus, RT-PCR, genotyping, epidemiology

Introduction

Norovirus (NoV) is a highly infectious pathogen that causes acute gastroenteritis in humans.[1] It is the most frequently identified cause of gastroenteritis in the community and institutional settings in Australia.[2,3] NoV is robust and may survive in marine environments in high concentrations if sewage is released, thus posing a contamination threat to shellfish.[1] Oysters have previously been identified as a transmission vehicle in NoV outbreaks.[4–6]

During an outbreak investigation, the detection of the same pathogen in human cases and epidemiologically implicated food assists investigators to implement appropriate public health action. This is particularly useful when the pathogen is indistinguishable in food and clinical samples using a discriminating sub-typing method. Australia has only recently developed the capacity for NoV detection and sub-typing in oyster tissue. Previously, oyster samples were processed in New Zealand laboratories with resultant delays in withdrawal of product contaminated with norovirus.[6]

The methods available for the detection of NoV in human faeces include electron microscopy, real-time reverse transcription-polymerase chain reaction (RT-PCR), enzyme-linked immunosorbent assays (ELISA) and immunochromatographic tests (ICT).[7] ELISA and ICT methods are relatively quick and inexpensive to use but the sensitivity of ELISA varies from 36%–90% while that of ICT varies from 57%–90%.[7,8] They are useful when screening large numbers of faecal samples where a single false negative is less critical. Real-time RT-PCR is resource intensive but is more sensitive (91%–98%)[9], and is useful for assessing critical samples that are clinically suspicious but negative using ELISA or ICT. In oysters, real-time RT-PCR is the preferred method for detecting NoV, as viral concentrations are generally much lower than those found in clinical specimens.[1]

There are currently 6 recognised NoV genogroups (G), three of which cause human infection (GI, GII and GIV).[10] The NoV GII currently accounts for most (>80%) human infections.[1] Both NoV GI and GII have previously been implicated in outbreaks associated with oyster consumption. It is not uncommon to find both genogroups in oyster samples collected during outbreak investigations.[5,6]

We report a NoV outbreak associated with New South Wales oysters where the value of real-time RT-PCR and genotyping was proven; real-time RT-PCR allowed confirmation of human infection where ICT tests were negative, and genotyping of both the human and oyster samples supported the epidemiological link between NoV infection and oyster consumption.

Methods

Ethical approval for this investigation was not required under the *NSW Public Health Act, 2010.*

Epidemiological investigation

On 30 October 2012, the Hunter New England (HNE) OzFoodNet site was notified by the New South Wales Food Authority (NSWFA) of gastrointestinal illness in 6 people from a cohort of 30 that had attended a social event at a caravan park between 22 and 26 October 2012. The HNE OzFoodNet site is part of the national OzFoodNet network that is responsible for the investigation and management of foodborne illness in Australia. An additional 2 cases with similar symptoms, but with no links with the social event, were also notified to HNE OzFoodNet on 1 November 2012, by the NSWFA. All 8 cases were interviewed using a standardised questionnaire for suspected foodborne illness. The interviewers were trained in the use of the questionnaire. Details on demographics, symptom profile, onset, duration, contact with ill persons, social activities, accommodation, travel and a 3 day food history were collected. Stool specimens were requested.

A suspected case was defined as a person from the region of interest who reported vomiting and/or diarrhoea plus one or more of headache, fever, abdominal cramping, lethargy or joint/muscle pain with onset between 25 and 27 October 2012. A confirmed case was a person who met the suspected case definition, and who had NoV GII detected by real-time RT-PCR in a stool sample.

The HNE OzFoodNet team initiated active case finding utilising the Public Health Real-Time Emergency Department Surveillance System (PHREDSS) to identify gastroenteritis presentations at emergency departments in this health district and two adjoining health districts.[11]

Laboratory investigation

Human sample

Only 1 stool sample was collected. The sample was initially tested for NoV by ICT (SD-Bioline) at a local laboratory. The sample was then sent to the University of New South Wales (UNSW) school of Biotechnology and Biological Sciences (BABS) for real-time RT-PCR testing and genotyping as UNSW is the only place in New South Wales that has the capacity and the validated methods to perform NoV genotyping.[12] RNA was extracted directly from stool samples using QIAamp Stool Mini Kit (Qiagen, Hilden, Germany) according to the manufacturer's instructions and suspended in 200 μL of elution buffer. RNA (10 μL) was converted to cDNA using the High Capacity Reverse Transcription Kit with RNase inhibitors (Applied Biosystem, Foster City, CA) according to the manufacturer's instructions. NoV GI or GII RNA was detected by real-time RT-PCR, and the products were purified, sequenced and genotyped as described previously.[12]

Oyster samples

Eleven samples collected on 5 November 2012 were analysed for the presence of NoV (GI and GII RNA), and for *Escherichia coli* as a recognised indicator of faecal contamination.[13] A further 8 samples collected on 14 January 2013 were analysed for NoV GI and GII RNA only. Samples collected were representative of the harvest area where the implicated lease was located. Not all leases in the harvest area could be sampled due to operational and resource constraints. Leases that were not sampled were either empty or contained immature stock. More samples were collected from leases with large volumes of saleable stock.

Samples were labelled and cold stored at 4°C for shipping to the laboratory. Once at the laboratory, the samples were assigned a laboratory number and stored at 4°C until processed.

The protocol used for NoV testing of the oysters was based on the International Standard horizontal method for detection of hepatitis A virus and norovirus in food matrices (European Committee for Standardization in collaboration with ISO, committee 34 – ISO/TC34).[14–16]

Briefly, the method consists of 5 steps: sample preparation, virus extraction, nucleic acid extraction, molecular detection using real-time RT-PCR, and result analysis. Samples were prepared by dissecting and finely chopping the digestive glands from the oysters. Proteinase K was then used to extract the virus from 2 g of the chopped digestive gland and the virus control (murine NoV) was added to determine the efficiency of the extraction. The Minimag® System (NucliSENS®) was used for RNA extraction using guanidine thiocyanate to disrupt the virus capsid and silica particles to adsorb RNA and to assist purification.[14–16]

Real-time RT-PCR was carried out in a 1 step process with a different set of primers/probes for NoV GI and GII (as specified in the ISO standard method). Each sample was tested in triplicate, and 2 serial dilutions were included. Positive and negative controls for each step of the method were included as per the ISO technical specification.

Environmental investigation

On 1 November 2012, the harvest area (Figure) was closed and a joint environmental investigation by the NSWFA and local council officers into the source of the contamination was initiated. All oyster farmers in the harvest area were contacted and advised to hold oyster sales at this time. A trade level product retrieval was commenced.

Amongst the 11 oyster samples collected for NoV analysis on 5 November 2012, sample size varied from 15 to 30 oysters, with 250 individual oysters collected in total.

Two samples were obtained from embargoed product held in an oyster farmer's cool-room on 2 November 2012 and 9 samples were obtained directly from the implicated oyster harvest area on 5 November 2012. The samples from the cool-room were harvested from the implicated area on 29 October 2012 and had been depurated for 36 hours.

Inspections of potential pollution sources were undertaken on 6 and 7 November 2012. All sewage pumping stations, manholes and on-site sewage management systems in the towns surrounding the implicated oyster leases were visually inspected for signs of recent discharge. A number of stormwater drains were sampled for faecal coliforms.

A local caravan park's reticulation system was targeted for further investigation due to the size of the system and its close proximity to the harvest area. On 27 November 2012 the NSWFA, in conjunction with local council officers, undertook dye testing of the system to check for leaks in the system.

On 20 December 2012, NSWFA officers conducted a basic hydrological study in relation to a subsequently identified sewage spill site. The study used marked oranges to provide a visual indication of river flow. The marked oranges were deployed at 8 am on 20 December (approximately 10 minutes after the turn of the tide) and observed for 6 hours.

On 14 January 2013, 8 samples of oysters were collected from leases located throughout the oyster harvest area for follow-up testing including 3 samples from the implicated leases. Each sample was analysed by real-time RT-PCR for NoV GI and GII RNA.

Results

Epidemiological investigation

No additional cases were detected through the PHREDSS review. A total of 8 cases were interviewed, with a median age of 69.5 years (range 64 to 77 years) and four were female. The median incubation period from oyster consumption to illness onset was 36 hours, with median illness duration of 50.5 hours. Symptoms included vomiting (6/8) and diarrhoea (8/8).

Case summaries

The 1st cases were a married couple and another adult who had stayed in separate self-contained motorhomes in the caravan park where the social event was hosted. Their only shared meal was 2 dozen oysters consumed at 2 pm on 24 October 2012. These oysters had been purchased directly from a local oyster supplier 2 hours earlier on the same day. Although the group had other social contact in the 4 days prior to onset, none had symptoms of gastroenteritis prior to, or during the shared meal. The onset of illness for this group was between 3 am and 9 am on 26 October 2012.

A further affected individual purchased and consumed 1 dozen oysters on 25 October 2012 at midday. These oysters were purchased from the same supplier as the first sub-group. He had not previously been ill and had no recollection of contact with ill persons prior to consuming the oysters. Although he was a participant in the social event, the only common link between this case and the first sub-group was consumption of oysters from the same supplier. His onset of illness was 3 am on 27 October 2012.

Two cases consumed oysters at a restaurant located in close proximity to the caravan park on 25 October 2012 at the same dinner service but at separate tables. The dinner service was at 7 pm. These 2 people had resided in two separate accommodation sites in the same caravan park during the social event. They had not had prior contact with each other nor with other cases attending the social event. This was the only meal that was common to both these people. The onset of illness in this group was between 7 am and midday on 27 October 2012.

Two further cases were a married couple who did not reside at the caravan park and were not associated with the social event at the caravan park. They purchased and consumed oysters from the same oyster supplier as the first 3 sub-groups on 25 October 2012 at 7.30 pm but also shared a number of additional food exposures prior to illness

onset. Their illness onset was identical at midnight on 26 October 2012. They did not report any contact with ill persons prior to their illness.

All cases reported oyster consumption with six of them purchasing oysters directly from the same supplier, while the trace back investigation identified that the oysters that the remaining 2 cases had purchased from a local restaurant were also sourced from that supplier. One stool sample was collected from a case in the first sub-group, who had recovered.

Laboratory investigation

Human specimen

The single human sample was negative for NoV by ICT, however, tested positive for NoV GII RNA by real-time RT-PCR. Sequencing and phylogenetics revealed the NoV was a GII.4 New Orleans 2009 variant.

Oyster specimens

A total of 19 samples of oysters were analysed for NoV GI and GII RNA. The results of the NoV analysis for 11 samples collected on 5 November are shown in the Table. None were positive for NoV GI by real-time RT-PCR. Three samples, collected from the western upstream section of the oyster harvest area were positive for NoV GII. It was not possible to genotype these samples due to the low levels of viral genetic material. *E. coli* was detected at 70 most probable number (MPN)/100 gram or less for the samples positive for NoV GII. There was no correlation between *E. coli* and NoV positive results. All samples collected on 14 January 2013 were negative for NoV GI and GII.

Environmental investigation

Of the 3 oyster samples that were positive for NoV GII, one was from the oyster farmer's coolroom (harvested on 29 October 2012, from the upstream section of the western side of the river) and the other 2 positive samples were collected on 5 November 2012 with one taken from each side of the river (Figure).

Follow up investigations revealed that the NoV positive oyster sample from the eastern side of the river had only recently been moved there from the western side of the river. All 3 positives samples were traced back to the western upstream section of the oyster harvest area.

On 6 and 7 November 2012, all sewerage pumping stations and on-site sewerage management systems in the proximity of the harvest area were found to be well maintained, with no signs of overflow, leakage or discharge to the environment. The results of the stormwater sampling were generally low (in the range of < 2–130 coliforms/100 ml) with one sample result of >200 faecal coliforms/100 ml from a stagnant pond attributed to contamination by duck faeces. None were suggestive of sewage ingress into the storm water system. The dye study conducted on 27 November 2012 indicated that there were no leaks in the caravan park reticulation system.

On 2 December 2012, a sewage leak was detected in a main sewerage line immediately east of the road bridge after flow from the leak caused subsidence to a section of road adjacent to the bridge (Figure). The leak was just upstream of where the caravan park line enters the system preventing its detection in the dye tracing study. The leaking sewerage line was promptly repaired by council on the same day.

The marked oranges released near the discharge point of the leaking sewerage line on the eastern side of the bridge drifted towards the centre of the river and proceeded down the main channel (Figure).The oranges by-passed the leases on the eastern side of the river, keeping to the main channel in the centre of the river. About 1.8 km downstream of the bridge the oranges started to drift apart. By 2 pm oranges were observed on both sides of the river in line with the leases implicated in the initial illness outbreak. The prevailing wind during the study was generally westerly at about 7 km per hour, which would have influenced the drift of the oranges in an easterly direction.

Applying a precautionary approach, all leases in the implicated area were initially closed with product that had been released to the market recalled on

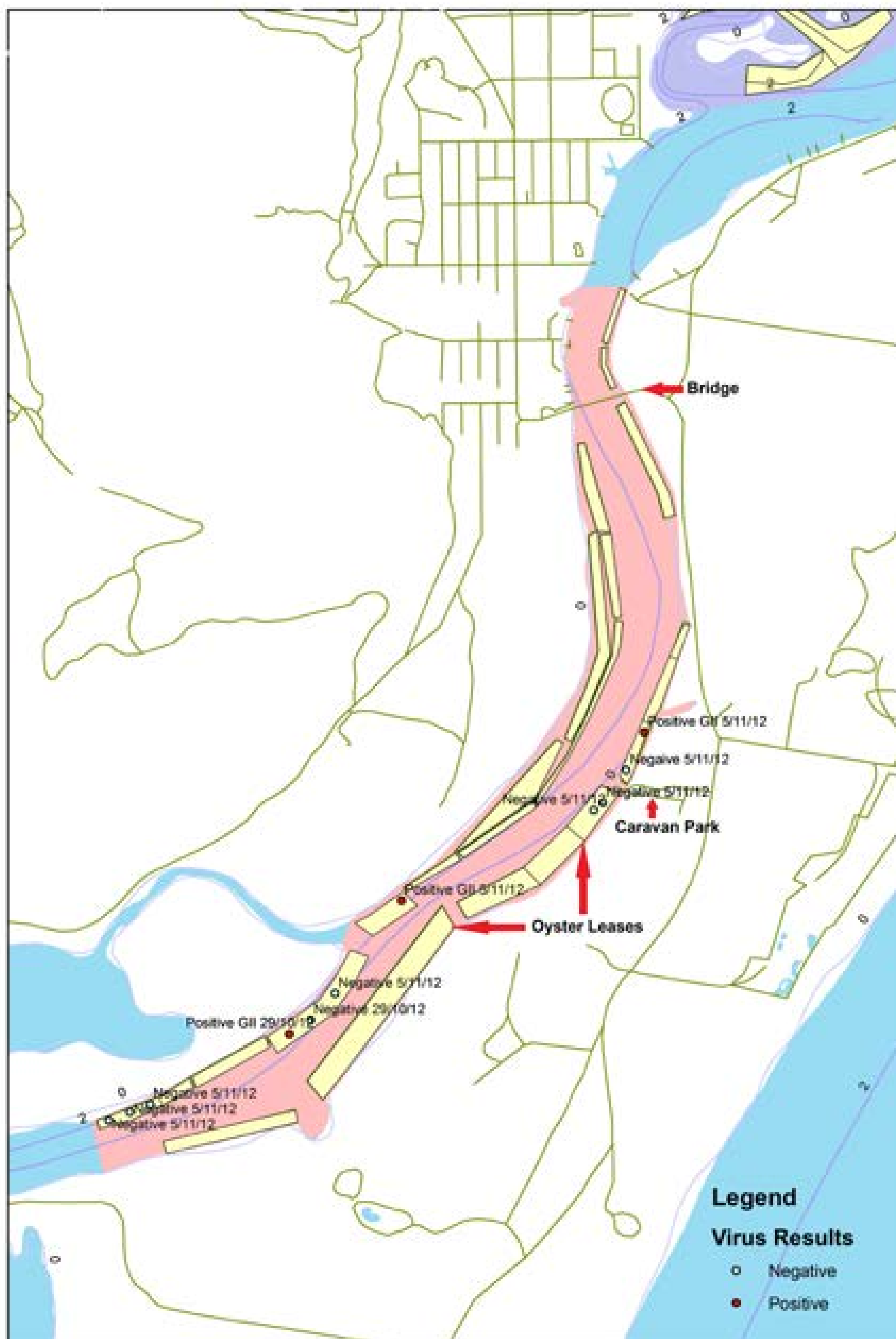
Table: Oyster sampling results, November 2012, implicated harvest area

ID	*E. coli* MPN/100 g	NoV GII	NoV GI
1	<20	Positive*	<LoD
2	<20	<LoD	<LoD
3	40	Positive*	<LoD
4	<20	<LoD	<LoD
5	<20	<LoD	<LoD
6	70	Positive*	<LoD
7	310	<LoD	<LoD
8	70	<LoD	<LoD
9	40	<LoD	<LoD
10	160	<LoD	<LoD
11	110	<LoD	<LoD

* Cyclic threshold values over 39 were considered positive values. In this case, cyclic threshold values ranged from 37 to 39.

LoD Level of detection

Figure. Map of the oyster harvest area



1 November 2012. Only a small amount of product was recalled as there was a limited amount of product in the market at the time of recall. The oyster leases with confirmed contamination were closed for 3 months. Surrounding oyster leases that were not implicated were cleared to resume operation by the NSWFA once the initial environmental investigation was completed.

Discussion

NoV GII RNA was detected in the human and oyster samples, supporting the epidemiological link between cases and oyster consumption. Further genotype discrimination beyond GII was only conducted on the human sample. It is possible that the genotype in the oyster tissue may have differed to that of the human sample, which was GII.[4]

Other possible sources of contamination included pollution events from passing marine vehicles, stormwater and the caravan park sewerage system. However these sources were investigated and deemed unlikely to be the cause of the oyster contamination. The main sewer line had been leaking for some time as evidenced by the road subsidence and the hydrological study was consistent with tidal flows over the implicated oyster lease.

Closure of the harvest area based on epidemiological and trace back evidence occurred on 1 November 2012 in accordance with the requirements of the Australian Shellfish Quality assurance Program (ASQAP).[17] A trade level product retrieval was conducted at this time and oyster farmers who were operating in the same river ceased trading voluntarily until environmental and laboratory investigations were complete. The real-time RT-PCR results identified which oyster leases were affected and provided further evidence to support and maintain the closure of these leases. This was important to prevent further cases and ensure that actions comply with the ASQAP requirements.[17] It was also important as the public health action had trade and financial implications for all of the oyster producers in the harvest area.

Although real-time RT-PCR successfully detected NoV GII in the oyster samples, further subtyping was not possible due to low viral levels. Factors affecting NoV detection in oysters include low viral levels, variability in the NoV genome and the complex extraction process, as well as inhibitory substances that interfere with real-time RT-PCR detection.[1,18] Despite these factors, real-time RT-PCR remains an internationally recognised and validated method of norovirus detection in oysters.[13–16] Further genotyping of the oyster samples to identify the strain beyond the genogroup level would have been useful.

In this outbreak only 1 human sample was collected. It is possible that this may have introduced a selection bias, however, all cases had consumed oysters from the same supplier and the onset of illness in cases reflected the recognised incubation period of norovirus. The sample was positive for NoV RNA by real-time RT-PCR but negative for NoV antigen by ICT (SD-Bioline). This highlights the limitations of ICT for detecting NoV in sporadic cases. Studies that have examined the sensitivity of antigen methods recommend collecting multiple samples to increase the probability of NoV detection, however, this can be difficult in practice.[8,19]

The environmental investigation quickly eliminated a number of potential pollution sources, including overflows from sewerage pumping stations, manholes and domestic on-site sewage management systems.

The results of the basic hydrological survey, coupled with the information from the laboratory analysis and the epidemiological data provided evidence that the leaking sewer line at the bridge was the most likely source of the viral contamination that caused the outbreak. The distance between the impacted leases and the source of the sewage discharge (2.5 km) and the low volume of the sewage discharge (~20 litres per hour) demonstrates the sensitivity of shellfish harvest areas to sewage contamination.

The negative virus results from the follow-up samples collected on 14 January 2013, verified the most likely source of contamination had been successfully remediated following the repair of the sewerage pipe. Subsequently, the affected leases were cleared to resume operation under standard quality assurance operating protocols.[17]

The risk of NoV contamination of oysters is best mitigated by controlling pollution sources. Australia currently has limited laboratory capacity to test oysters for NoV using real-time RT-PCR, which may lead to delayed results in the context of a large outbreak. In the future, cost effective molecular testing for human viruses could be considered for inclusion in oyster quality assurance programs as faecal coliforms and *E. coli* are poor predictors of viral contamination and depuration does not reliably clear viral pathogens, as demonstrated in this investigation.[13,20,21]

Conclusion

The findings of this investigation suggest that real-time RT-PCR testing of human specimens may be valuable where ICT tests are negative, but clinical illness is suggestive of NoV infection. NoV real-

time RT-PCR assisted in linking human infection with oyster contamination and this, together with good descriptive epidemiology led to a thorough environmental investigation and appropriate action to mitigate further public health risk.

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An outbreak of norovirus linked to oysters in Tasmania

Kerryn L Lodo, Mark GK Veitch, Michelle L Green

Abstract

Norovirus is the most commonly reported virus in shellfish related gastroenteritis outbreaks. In March 2013 an investigation was conducted following the receipt of reports of gastroenteritis after the consumption of oysters at private functions in Tasmania. Cases were ascertained through general practitioners, emergency departments, media releases and self-reporting. Of the 306 cases identified in Tasmania, 10 faecal specimens were collected for laboratory testing and eight were positive for norovirus (GII.g). The most common symptoms were vomiting (87%), diarrhoea (85%), myalgia (82%) and fever (56%). The implicated oysters were traced to a single lease from which they were harvested and distributed locally and interstate. Nationally 525 cases were identified from Tasmania (306), Victoria (209), New South Wales (8) and Queensland (2). This report highlights the consequences of norovirus outbreaks in shellfish, even with rapid identification, trace back and removal of the implicated product from the market. *Commun Dis Intell* 2014;38(1)E16–E19.

Keywords: norovirus, oysters, disease outbreak, foodborne disease, multi-jurisdictional

Introduction

On 31 March 2013, 2 clusters of acute gastroenteritis linked to separate private functions were reported to the Tasmanian Department of Health and Human Services (the Department) Public Health Hotline by a General Practitioner (GP) and a member of the public. Symptoms included vomiting and/or diarrhoea with onset approximately 24–30 hours after the functions. Both functions served oysters, which had been purchased from Company A on 28 March 2013. All cases reported oyster consumption. A Tasmanian outbreak investigation team was formed and an investigation initiated.

Methods

Epidemiological investigations

Case ascertainment was conducted through:

- alerts to GPs and Emergency Departments in Southern Tasmania;
- media releases requesting possible cases to contact the Public Health Hotline; and
- follow-up of contacts of self-reported cases notified to public health.

A questionnaire was developed and departmental staff interviewed all cases in Tasmania that self-reported. Ethics approval was not sought as data were collected as part of a routine public health investigation and response.

Laboratory investigations

Cases from the initial 2 clusters were followed up and 10 faecal specimens from these cases were submitted to a local microbiology laboratory for bacterial culture, norovirus and parasitology testing.

Seven of these samples were referred to the University of New South Wales Molecular Virology Laboratory for further characterisation.

Environmental investigations

Trace back investigations were implemented. The oyster processing facility at Company A was inspected by the local environmental health officer (EHO) for possible post-harvest contamination. A sanitary survey of the harvest lease and surrounding area was conducted. This survey included inspection and testing of all sewage management infrastructure in the surrounding area, including inspection of possible waste dumping sites of caravans. Water samples from the lease were collected and tested for *Escherichia coli*.

Data on rainfall trends, tides and hydrology of the area surrounding the lease around the time the oysters were harvested were collected for hydrological assessment.

Seven samples of oyster meat were sent to the South Australian Research and Development Institute (SARDI) for viral testing for hepatitis A and norovirus. These samples were from uneaten product obtained from 3 separate cases, withdrawn product from Company A and oysters collected directly from the harvest lease.

National activity

Chief health officers were informed of the possibility of illness associated with Tasmanian

oysters on 2 April. A national alert was circulated through OzFoodNet (Australian network for the surveillance of foodborne diseases) on 3 April. At an OzFoodNet teleconference on 4 April, a multi-jurisdictional outbreak was declared.

Results

Epidemiological results

In the initial 2 clusters, all those who consumed oysters became ill (Table). These oysters were the only food common to both functions. Five additional clusters of illness were notified the following day. The attack rate of Company A oysters ranged from 86%–100% in groups reported over the course of the outbreak.

Tasmanian cases were defined as:

Confirmed case: A person who had been ill with vomiting and/or diarrhoea with onset on or after the 25 March who has reported eating Tasmanian oysters purchased from Company A between 24 and 31 March, and had a faecal specimen where norovirus has been detected.

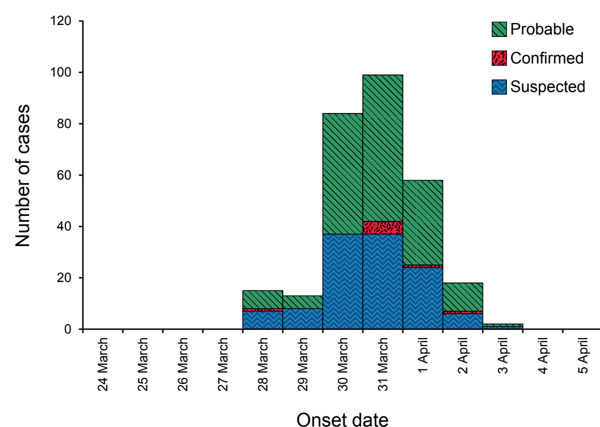
Probable case: A person who had been ill with vomiting and/or diarrhoeas with onset on or after 25 March who has reported eating Tasmanian oysters purchased from Company A between 24 and 31 March.

Suspected case: A person who had been ill with vomiting and/or diarrhoea with onset on or after 25 March, who has been reported as eating oysters purchased from Company A between 24 and 31 March by a confirmed or probable case, but who was not contacted for interview to ascertain all details.

In Tasmania, there were 306 cases identified that met one of the case definitions. Interviews were conducted with 128 confirmed and probable cases who reported a further 178 suspected cases. The suspected cases were not followed further. It was felt that sufficient data had been collected to identify the source of illness and implement control measures. These cases are not included in the following descriptive epidemiological analysis.

Of the 128 confirmed and probable cases interviewed, the average age was 50 years (range 13–78) with a sex distribution of 53% female and 47% male. The median incubation period was 31.5 hours (mean 29.5, range 5–58 hours) and the median duration of illness was 48 hours. Onsets of illness occurred between 28 March and 3 April 2013 (Figure). Symptoms reported included vomiting (87%), diarrhoea (85%), myalgia (82%) and fever (56%). Twenty-two cases (17%) sought medical attention, and 1 case reported hospital admission.

Figure: Cases of gastrointestinal illness associated with Company A oyster consumption in Tasmania, 26 March to 5 April 2013, by onset date



Information on the preparation of oysters consumed by cases was collected during interviews. The majority of cases (66%) ate raw oysters only, 13% of cases ate cooked oysters only, and 18% ate a combination of both. Respondents reported consuming oysters in quantities ranging from one to 36 before illness. The most frequently reported number of oysters eaten was six.

Laboratory results

Of the 10 human samples sent for testing, 8 faecal specimens had norovirus detected and 1 sample also had *Campylobacter* detected. All 7 samples sent to the University of New South Wales Molecular Virology Laboratory had norovirus GII.1 detected with 2 representative samples further tested and identified as being a recombinant strain (GII.g).

Table: Attack rates of gastroenteritis in initial cohorts notified

	Attendees	Number consuming oysters	Number symptomatic	Attack rate
Function 1	9	7	7	100%
Function 2	13	8	8	100%

Environmental results

The sanitary survey of the environment of the area surrounding the harvest lease of Company A, including fluorescein dye testing and visual inspection of waste water infrastructure, identified a private sewerage pipe that was leaking underwater. The sewerage lagoons and pump station were fully functional with no evidence of leakage or overflow. Water samples collected during the sanitary survey of the harvest lease all had *E. coli* levels <10 cfu/100 mL. No evidence of waste dumping by boats or caravans or post-harvest contamination was found.

Hydrological assessment found falling tides flowed over the broken pipe and across the implicated lease. Peak flow times of the broken pipe were compared with tidal movements and high and mid falling tides coincided with the peak flow in the week before the harvest period of the implicated oysters.

Testing for norovirus in oyster meat samples by SARDI using the ISO/CEN standard method found trace levels of norovirus genogroup II (GII) RNA in two live un-shucked oysters from Company A using reverse transcription time polymerase chain reaction. The significance of this result is unknown as there is no threshold infectivity limit detectable by polymerase chain reaction and there was not enough RNA for sequencing to further characterise the virus to genotype level[1]. All tests were negative for hepatitis A.

Outbreak management

The investigation of this outbreak was rapidly undertaken following the initial notification of the two clusters on 31 March 2013. Descriptive evidence suggested oysters were the source and given the timing of the outbreak at Easter, rapid action was necessary to prevent further cases. The harvest lease was closed by Company A on advice from the Tasmanian Shellfish Quality Assurance Program (TSQAP) and the product withdrawn from sale from Company A's shop front the same day. This lease was identified as the only location this producer had harvested oysters sold during this period. Of 1,600 dozen oysters in the Tasmanian market, 600–700 dozen were withdrawn locally.

The damaged sewerage pipe was crimped and the leak stopped. The owners of the pipe were notified and served an abatement notice. A second oyster lease nearby was closed as a precaution. The harvest lease was re-opened on 3 January 2014, in line with TSQAP protocols.

National response

More than 4,500 dozen oysters were distributed outside Tasmania. Authorities in Victoria, New South Wales and Queensland investigated local distribution and implemented withdrawal of Company A oysters. Withdrawal was difficult in some states due to mislabelling of oysters and the common practice of co-mingling product at the point of sale.

In total, 525 cases of illness associated with the consumption of Tasmanian oysters were identified in Australia, from Tasmania (306), Victoria (209), New South Wales (8) and Queensland (2), including 17 confirmed as norovirus. Of those in Victoria, 165 were linked with 4 point source outbreaks at large food premises and the remainder from retail outlets and smaller food outlets. Cases in New South Wales were associated with a single retail outlet and Queensland cases were linked to a restaurant, all supplied with Tasmanian oysters from the implicated harvest of Company A.

Discussion

This outbreak progressed rapidly. Initial descriptive evidence included symptoms and incubation periods consistent with suspected norovirus infection (later confirmed with faecal specimen testing). Oysters from a common supplier were associated with initial clusters with high attack rates. The oysters from Company A were therefore withdrawn from sale as a precautionary measure. Although this action was taken within hours of the initial cluster being notified to the Department, there were more than 300 cases identified in Tasmania, and likely more that did not present to a medical practitioner or contact the Department.

Rapid identification of this outbreak and withdrawal of oysters may have prevented additional cases. However, the low infective dose of norovirus and high risk food, combined with a holiday traditionally associated with increased seafood consumption, still resulted in a large outbreak. Withdrawal or recall was made difficult by the rapid local and interstate distribution, rapid consumption of this product and a lack of labelling at the consumer level. This was further compounded in this outbreak by the common practice of co-mingling of oysters at retail outlets, and mislabelling of some oysters in the distribution chain.

Of the 306 cases in Tasmania, the number of samples available for testing was limited to 10, with 8 positive for norovirus (GII.g). While the number of specimens was small due to the timing

of interviews and the short duration of symptoms, 6 positive specimens are sufficient to confirm the causative pathogen as norovirus in 97% of outbreaks, with any more than 7 samples not improving sensitivity.[2]

The detection and notification of this outbreak among smaller private groups by a local GP was critical to timely action and case ascertainment. This is uncommon in norovirus outbreaks where many cases do not present to health care services and are rarely investigated further when they do.[3] Reported incidents are primarily associated with large functions, where outbreaks are more apparent and associations between exposure and illness more likely to be identified. This was demonstrated in Victoria with the majority of cases associated with large functions over the Easter weekend, though this may have been a result of distribution patterns of the oysters.

Cooking methods typically used to prepare oysters are not sufficient to inactivate enteric viruses present inside shellfish as a result of the faecal contamination of a growing area. However, they can inactivate virus as a result of surface contamination during processing or preparation.[4] Past outbreaks of norovirus linked to cooked oysters support these findings.[5]

The TSQAP program follows the Australian guidelines for the monitoring of oyster growing areas, based on an internationally accepted model. It uses faecal coliforms as indicators of contamination.[6] While faecal coliforms are used as an indicator of both bacterial and viral enteric pathogens, they have been found to be inadequate predictors of the presence of viral pathogens in shellfish.[7–9] Currently, testing of oysters for norovirus is slow, expensive, has limited availability and poor sensitivity. It is not currently a viable method for ongoing monitoring considering the rapid harvest and consumption cycle of oysters.

Norovirus is the most commonly reported virus in shellfish related gastroenteritis outbreaks.[9] This outbreak highlights the significant illness that can occur even with rapid identification and notification of shellfish associated norovirus outbreaks and the challenges in monitoring shellfish growing areas for viral pathogens.

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Short reports

Short incubation periods in *Campylobacter* outbreaks associated with poultry liver dishes

Kirsty G Hope, Tony D Merritt, David N Durrheim

Introduction

Campylobacter is the most common cause of bacterial gastroenteritis in Australia.[1] The incubation period is usually between 2 and 5 days but can range from 1 to 10 days.[2] Symptoms include diarrhoea (often bloody), fever and abdominal pain, and can persist for 7 days or longer.[2] The infective dose required to cause illness is as low as 500 organisms.[3,4] Despite a high number of notifications, outbreaks caused by *Campylobacter* are uncommonly detected.[3,5–7]

Outbreaks of *Campylobacter* have regularly been associated with poultry and dairy products, and in recent years the number of outbreaks associated with poultry liver dishes has increased in the United States of America and the United Kingdom.[5,6,8]

This article describes a point source outbreak of *Campylobacter* associated with duck liver parfait with a possible short incubation period; and the review of the literature prompted by the investigation, which identifies short incubation periods as a common feature of point source outbreaks associated with poultry liver consumption.

Methods

OzFoodNet in Hunter New England was contacted in September 2013 with a report of gastroenteritis amongst guests who attended a wedding reception. A retrospective cohort study was conducted amongst the guests. A standardised questionnaire was completed telephonically by trained interviewers.

A case was defined as any person who consumed food and/or beverages at the wedding who had diarrhoea within 7 days of attending the wedding.

Data analysis was conducted with STATA 11. Univariate analysis included attack rates, *P*-values, relative risks and 95% confidence intervals.

The NSW Food Authority conducted an inspection of the implicated premises. Although there was no food left-over due to the late notification of the outbreak (35 days after the event), information on ingredients and cooking processes of foods served was obtained.

A literature review was conducted using the search term ‘campylobacter’ in combination with ‘poultry’, ‘duck’, ‘chicken’ or ‘liver’. An extract of outbreaks associated with poultry liver (chicken or duck) was obtained from the Australian OzFoodNet outbreak register. Incubation times recorded in hours were converted to days to 1 decimal point.

This outbreak investigation was conducted under the *NSW Public Health Act 2010* and thus ethics approval was not required.

The outbreak

Contact details were available for 33 of the 50 guests, 30 (91%) of whom completed the questionnaire and 17 met the case definition. The median incubation period was 24 hours (range 5–60 hours) and 12 cases reported an incubation period of less than 24 hours. All cases reported diarrhoea, 12 had abdominal pain, nine had fever, seven had nausea and three had vomiting. The median duration of illness was 6 days. One case was hospitalised. One faecal sample was obtained, which was positive for *Campylobacter jejuni*. The incubation period for the confirmed case was 2 days.

In a univariate analysis, the only significant association with illness was for the consumption of the entree that contained duck liver parfait (relative risk 4.3, 95% confidence intervals 1.2–15.5). Fifteen of the 17 (88.2%) cases ate the duck entrée.

The environmental investigation indicated that the duck liver was cooked to a maximum internal temperature of less than 70°C. No food samples were available for testing. No illness was identified in staff or in guests attending other functions at the implicated venue.

Review of *Campylobacter* outbreaks associated with poultry liver dishes

Eight published outbreak reports and 6 additional outbreak records from the OzFoodNet register were reviewed (Table). The median incubation period for *Campylobacter* outbreaks associated with poultry liver in these outbreaks was typically about 2 days, with individual incubation periods ranging from less than 1 day to 9 days. The incubation period for the 1st confirmed case was available for 9 outbreaks, of which 4 (44%) reported an incubation period of less than 1 day.

Table: Characteristics of *Campylobacter* outbreaks associated with poultry liver

Vehicle	Jurisdiction	Number of cases	Number attending	Number confirmed *Campylobacter* only	Number confirmed *Salmonella* and *Campylobacter*	Median incubation (days)	Incubation range (days)	Incubation first confirmed case (days)
Chicken liver pâté	South Australia[4]	15	57	3	0	2	0.3–6	<1
Duck liver pâté	United Kingdom[8]	18	32	8	0	NA	0.5–3	<1
Chicken liver pâté	United Kingdom[9]	59	175	5	3	2.	0–6	<1
Chicken liver pâté	Australian Capital Territory	7	Unknown	3	0	1	0.5–3.8	<1
Chicken liver pâté	United Kingdom[10]	49	102	22	0	2.3	0–5	1
Chicken liver pârfait	United Kingdom[3]	24	67	13	0	2.3	1–5	1
Duck liver pâté	United Kingdom[11]	45	77	4	0	2.8	0.4–8.4	1
Chicken liver pâté	Tasmania	44	Unknown	5	0	2	0.6–4.1	1.5
Duck liver pârfait	New South Wales*	17	50	1	0	1	0.2–2.5	2
Chicken liver pârfait	United Kingdom[12]	11	26	4	0	2.5	0.7–7	NA
Chicken liver pâté	New South Wales	11	34	2	0	1.8	1.3–3.5	NA
Duck liver pâté	Western Australia	65	705	3	3	2.5	0.1–8.1	NA
Chicken liver pâté	Scotland[13]	48	Unknown	15	0	2	0–9	NA
Chicken liver pâté	Queensland	4	Unknown	1	0	1.6	1.1–2.1	NA
Duck liver pan-fried	Queensland	2	Unknown	2	0	2.7	1–3.5	NA

NA Not available

* Current outbreak.

Discussion

This review indicates that short incubation periods are a common feature of *Campylobacter* outbreaks associated with poultry liver dishes. During this outbreak 12 (71%) cases indicated onsets within 24 hours of the function. The review of previous poultry liver related outbreaks indicated that 10 of 14 outbreaks (excluding the current outbreak) had a minimum incubation period of less than a day, with the shortest being 0.1 days. When analysis of the 1st case was limited to confirmed infection only, four of the 9 outbreaks had cases with an incubation period less than 1 day and three had an incubation period of 1 day.

Campylobacter infection in humans usually has a reported incubation period of 2 to 5 days, with some references indicating one to 10 days.[2] Due to the high levels of *Campylobacter* potentially present within liver, it is possible infected individuals may have a shorter incubation period due to a large dose.

During this outbreak investigation, it was clear that the internal temperature achieved during preparation of the liver dish was not adequate to kill *Campylobacter*. Poultry livers should be cooked for 2 to 3 minutes after they reach an internal temperature of 70°C.[14] Inadequate cooking of chicken or duck livers has been associated with numerous *Campylobacter* outbreaks in Australia and internationally.[4,5,8,10,11] Following a review of outbreaks linked to poultry liver dishes in Australia in 2011,[15] Food Standards Australia New Zealand issued advice on the safe cooking of poultry livers.[14] New Zealand studies have shown that *Campylobacter* contaminates both the external and internal tissue of livers and that inactivation of *Campylobacter* is proportional to cooking time.[16–17]

Limitations

The outcome of the initial outbreak investigation that prompted this review is subject to potential recall bias as the investigation did not commence until 35 days after the function had occurred. As not all guests could be contacted (30 out of 50 were interviewed) the possibility of selection bias cannot be excluded.

Many of the outbreaks reviewed only obtained stool specimens from a small proportion of cases and thus the illness reported in some individuals may not have been due to *Campylobacter*. However, there were confirmed cases in previous outbreaks that had incubation periods less than 1 day. Some studies found cases with mixed infections of *Salmonella* and *Campylobacter*. *Salmonella* can have an incubation period as short as 12 hours, and have similar symptoms, therefore it is possible the short incubation periods were the result of another infection rather than *Campylobacter* in some instances. Three of the outbreaks reviewed identified cases with mixed infection; two of these had incubation periods of less than 1 day.

Incubation periods of *Campylobacter* outbreaks associated with other food vehicles were not reviewed as part of this study. Therefore no comparison can be made with other food vehicles.

Conclusion

It is not uncommon to identify cases with short incubation periods (less than a day) in campylobacter point source outbreaks associated with poultry liver consumption. This may result from the potentially high infectious dose in liver. Investigators should not discount suspected gastroenteritis cases with short incubation periods when a poultry liver dish is implicated.

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Chronic carriage and familial transmission of typhoid in western Sydney

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Introduction

Typhoid is a systemic bacterial disease caused by *Salmonella enterica* subspecies *enterica* serovar Typhi (commonly *S.* Typhi).[1] It is usually contracted by ingestion of food or water contaminated by faecal or urinary carriers excreting *S.* Typhi.[2] Typhoid continues to contribute to the global burden of disease, particularly in countries with low and middle gross national income.[1] Locally acquired cases of typhoid in Australia are uncommon, with most cases being associated with international travel.[3]

Humans are the only known hosts for *S.* Typhi and no animal or environmental reservoirs have been identified. People with typhoid typically present with a sustained fever, headache, malaise and anorexia. The clinical picture of typhoid varies depending on the severity of the disease. The case fatality rate is usually below 1% if appropriate antibiotic treatment is given.[1] The incubation period ranges from 3–60 days (usually 8–14 days) and about 10% of untreated typhoid patients excrete *S.* Typhi for 3 months after onset of symptoms.[1] Chronic carriage occurs in 3%–5% of the population infected with *S.* Typhi, with the gallbladder a site of persistence.[4] This is an important public health issue, as carriers act as a reservoir for further spread of the disease through bacterial shedding in faeces.[4]

In New South Wales, typhoid is notifiable under the *Public Health Act 2010* and public health follow-up is required to prevent spread of the disease.[5] An epidemiological review of typhoid in New South Wales identified 250 case notifications between 2005 and 2011, of which 240 were believed to have been acquired overseas.[3] Case notifications remained stable over that period with the highest rates of typhoid (1.8 per 100,000) in Western Sydney Local Health District (LHD).[3]

From January to July 2013, the Western Sydney LHD received 17 case notifications for typhoid, which is an increase compared with 2012 figures (9 case notifications). The Western Sydney Public Health Unit (PHU) investigated the higher-than usual number of typhoid case notifications compared with the same period in the previous year, with particular interest in the cases with no recent history of overseas travel, no overseas visitors and no epidemiological link to other recent cases. This case report examines the investigations and follow-up completed by the Western Sydney PHU for a family cluster of cases.

Background and methods

In May 2013, the Western Sydney PHU received laboratory notifications for 2 cases of *S.* Typhi infection (confirmed from faecal culture). The cases were siblings (aged 3 years and 6 years), with symptom onset in late April 2013. Both children experienced fever, diarrhoea and vomiting and were admitted to hospital.

Following notification, the PHU conducted case management and investigations as per the NSW Health communicable diseases protocol.[5] An interview using a standardised questionnaire was conducted with the children's father to seek further information, to identify contacts and the likely source of infection. All contacts were screened using stool sampling, household contacts included the children's 8-month-old brother and both parents. Other contacts were 4 people in a family household where meals where shared. In addition, isolates that were identified as *S.* Typhi were phage typed at the Microbiological Diagnostic Unit, The University of Melbourne. Ethics approval was not required as the investigation and follow-up was conducted under the *Public Health Act 2010*.[5]

Description of cluster

The results of the initial public health investigations indicated that the 2 cases were locally acquired. None of the family members reported that they had received vaccination for typhoid and there was no recent travel to typhoid endemic countries reported by the family, nor any recent visitors from overseas. The vaccination status of the household where meals were shared was unknown. The family had immigrated to Australia from Bangladesh a number of years previously, and only the father had travelled back to Bangladesh in 2011 for 3 weeks. The food suppliers used by the family were not associated with any other cases in Western Sydney.

Results of the screening of household contacts were negative for *S.* Typhi for the children's 8-month-old brother, their mother and all 4 members of the other household. However, *S.* Typhi was cultured from both screening stool specimens submitted by the children's father. *S.* Typhimurium was also isolated in one of these specimens. The father was asymptomatic, he did not report a history of chronic illness, nor was he taking any medication. He reported providing some assistance with his children's activities of daily living, specifically

with food preparation. Following the detection of *S.* Typhi in stool specimens, the father had an abdomen ultrasound that showed a single mobile gallstone measuring 7 mm in the gallbladder, which can be a site of persistence for *S.* Typhi.

Public health action to minimise the spread of infection included providing education to the family on hand hygiene, establishing there was no occupational, school or child care risks and ongoing management to ensure cases clear *S.* Typhi. All 3 familial cases were phage type E9, which has been shown to be found in Bangladesh.[6]

Discussion

The public health investigations supported the hypothesis that the children's father was a chronic carrier of *S.* Typhi and the likely source of their infection. Chronic *S.* Typhi infections can persist for decades, infected individuals are highly contagious and typically asymptomatic, making the identification of carriers difficult.[7] An epidemiological study has shown a strong link between the development of the chronic carrier state and the presence of gallstones; approximately 90% of chronically infected carriers have gallstones.[7]

It is possible that the father contracted *S.* Typhi on his most recent trip to Bangladesh. Travellers who return to their country of origin to visit friends and relatives are at an increased risk of contracting diseases such as typhoid.[8] They are at higher risk as they are more likely to travel to rural areas, less likely to have received pre-travel advice, less likely to exercise food and water precautions, less likely to receive typhoid vaccination before travelling and have lower levels of perceived risk.[8] Travellers who are returning to their countries of origin to visit family and friends account for a large number of cases of typhoid reported in New South Wales. Of overseas acquired cases from 2005 to 2011, 77% were associated with people returning to their country of origin.[3]

The higher number of typhoid case notifications in Western Sydney LHD may reflect the cultural diversity of the area. Approximately 40% of the population report being born overseas and a high proportion of migrants report coming from typhoid-endemic areas.[9,10] This familial cluster highlights the importance of screening household members for *S.* Typhi carriage, particularly when there is no reported recent overseas travel or visitors from endemic countries.

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Hepatitis A outbreak associated with kava drinking

Jo-Anne M Parker, Thomas Thompumkuzhiyil Kurien, Clare Huppatz

Abstract

Hepatitis A is caused by the hepatitis A virus (HAV), with transmission occurring through the faecal-oral route. In May 2013, a case of hepatitis A infection was reported to a Western Australian regional public health unit, with infection acquired in Fiji. Following this, 2 further cases were linked to the index case by kava drinking and 1 further case was a household contact of a secondary case. This outbreak highlights that the preparation of kava drink and/or the use of a common drinking vessel could be a vehicle for the transmission of HAV. *Commun Dis Intell* 2014;38(1):E26–E28.

Keywords: hepatitis A, outbreak investigation, kava

Introduction

Hepatitis A is an acute infection of the liver caused by hepatitis A virus (HAV). It is predominantly transmitted person-to-person via the faecal-oral route.[1] Sources of infection include contaminated water, infected food handlers and raw or undercooked food sources. A case is considered infectious from a few days before the onset of prodromal symptoms to 1 week after the onset of jaundice or 2 weeks after the onset of the prodromal symptoms, whichever comes first.[2]

In Western Australia, hepatitis A is a notifiable disease. The role of the public health unit (PHU) is to undertake enhanced surveillance of cases to identify the source of infection and to implement measures to reduce further transmission. In the Western Australia Goldfields Region, 36 cases of hepatitis A were reported between 2003 and April 2013, an average of less than 4 cases per year.[3] In May 2013, the PHU was informed of a laboratory confirmed case of hepatitis A in a 40-year-old Fijian male (case 1). The PHU was notified 34 days later of 2 further laboratory confirmed cases of HAV (cases 2 and 3). A 4th case (case 4) was notified in late July.

This report describes the investigation into a cluster of 4 cases of HAV infection.

Methods

Follow-up was conducted for all cases by the PHU staff in accordance with current enhanced surveillance guidelines, using a standardised questionnaire to determine epidemiological links and/or high risk activities. Blood specimens for all cases were sent to the Victorian Infectious Diseases Reference Laboratory (VIDRL), for HAV genotyping to be performed, as is the standard practice for all locally acquired cases of HAV. The investigation was performed as part of routine public health work.

Results

On 26 May 2013, the index case presented with anorexia, nausea, vomiting, lethargy, frontal headache and fever. The index case had travelled to Fiji 4 weeks prior, with symptoms developing 18 days after his return. Clinical examination revealed mild jaundice, noted on the 2nd day of admission. Blood tests showed elevated liver function tests (LFTs) and positive HAV IgM. He was treated symptomatically and his hospital stay was uneventful. He was discharged on day ten.

The PHU interview revealed that during his stay in Fiji, the index case resided in a remote village with poor sanitary conditions including makeshift bathrooms, pit toilets and no running water. He also described consuming street food and untreated drinking water. He had not previously been vaccinated against HAV. The index case did not identify any close contacts who may be at risk of transmission from him, such as immediate family, sexual contacts or persons who consumed food that he had prepared and had not been further cooked. He was provided with verbal and written information regarding HAV, including the infectious period and routes of infection.

On 4 July 2013, 2 further cases of hepatitis A were reported to the PHU by the local hospital medical team. Case 2 presented to the Emergency Department on 1 July 2013 with an 11 day history of fever, myalgia, nausea, vomiting, general fatigue and a 6 day history of jaundice. Investigations showed elevated LFTs and positive HAV IgM and IgG. He was managed symptomatically and was discharged on day four. During interview, case 2 revealed that he had drunk kava with the index case in June, 1 day after the index case was discharged from hospital and 8 days after his onset of jaundice. Case 2 reported that the kava drink was prepared by the index case and they shared a communal drinking vessel.

Case 3 presented to the local hospital the same week as case 2, complaining of a 1 week history of nausea, vomiting and generalised fatigue. He was later noted to be mildly jaundiced with raised LFTs and was positive for HAV IgM and IgG. His recovery was uneventful and he was discharged home on day six. During extensive interviewing with case 3 it was confirmed that he too had shared kava with 2 male friends, later found to be the index case and case 2.

In late July a further case (case 4) was reported to the PHU. Case 4 was the 8-year-old child of case 3. Case 4 presented to the local hospital with a 5 day history of abdominal pain, headache followed by fever, nausea and vomiting. Case 4 had been given HAV vaccination 17 days prior to becoming symptomatic. Hepatitis serology detected hepatitis A IgM and IgG antibodies. Transmission was likely due to normal household activities.

The temporal relationship between all 4 cases is shown in the Figure.

The results of HAV genotyping from blood specimens showed that all 4 cases had the same HAV genotype (IA) and with 100% sequence identity.

Discussion

Kava is an unusual reported vehicle of HAV infection. This outbreak revealed that kava drinking was linked with transmission of 2 cases of HAV infection. Mixing of kava was described by one of the cases as a process by which kava root powder is placed in a muslin cloth and soaked in a vessel filled with cold water. Manual extraction is used, with repeated squeezing of the cloth to extract kava. A second vessel is then used to dip into the mixing vessel and pass around as a communal drinking cup. The source of the HAV was most likely the index case during preparation of kava and/or via the shared drinking vessel.

Multiple interviews assisted in obtaining information to link these cases. At the time of the interview with the index case, a history of kava drinking was not elicited. In contrast, multiple interviews were performed with the subsequent adult cases, which elicited the history of kava sharing. The 3 adult cases were all reluctant to discuss kava use. This reluctance may have been due to cultural differences, a perceived disapproval of the activity or because its importation is restricted. The current standardised questionnaire for hepatitis A is generic and as a result does not identify different cultural practices as a risk factor for the transmission of HAV.

Of concern in this outbreak is the time of apparent infectivity of the index case and that despite provision of written and verbal information to the index case he participated in a high risk activity a day after discharge, facilitating transmission of HAV. The index case participated in kava drinking 8 days after onset of jaundice and 10 days after the onset of his prodromal symptoms. The *Hepatitis A National Guidelines for Public Health Units* outlines the infectious period of cases as being a 'few days before onset of prodromal symptoms to a few days after onset of jaundice and non-infectious 1 week after onset of jaundice or 2 weeks after onset of prodromal symptoms, whichever comes first'.[2] Applying these timeframes to the information provided by the 3 adult cases, case 2 and 3 were exposed outside of the infectious period of case 1. This has implications for the usual advice given to patients. While it is imperative to ensure that an accurate timeline of symptom onset is ascertained, a conservative approach should be used when discussing this timeline with patients. The emphasis should be on preventative measures such as hand hygiene and avoiding high risk activities, rather than the length of time during which they must exercise this caution.

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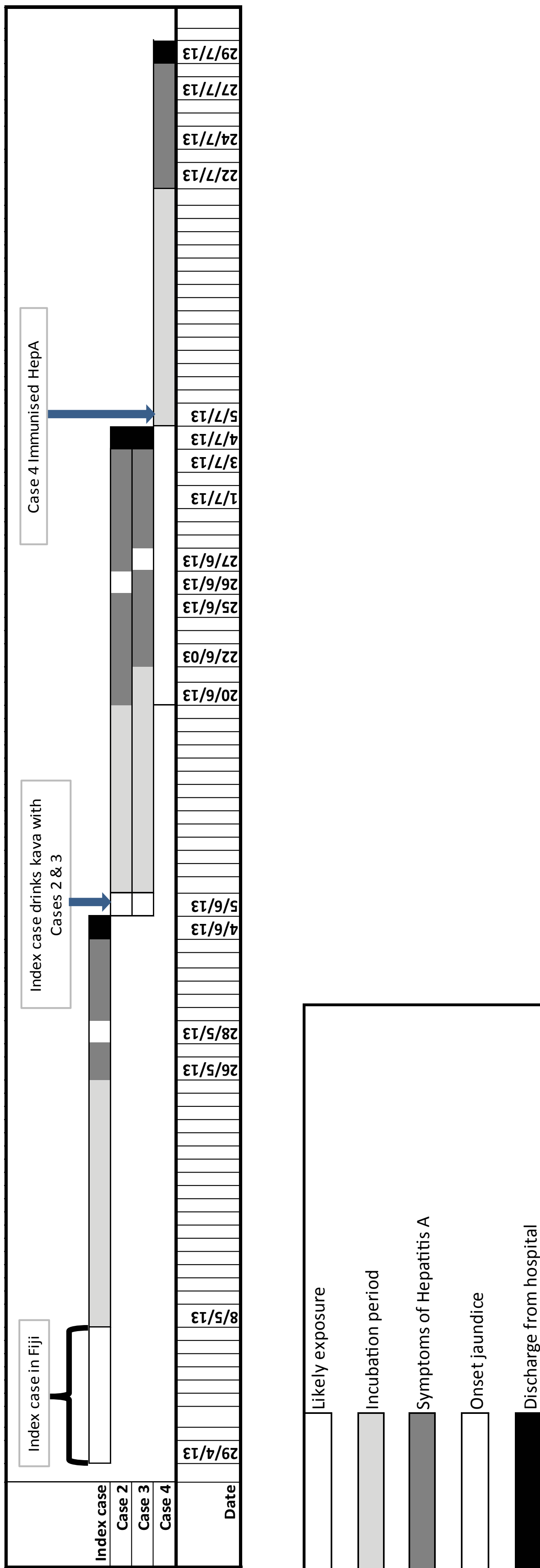


Figure: Timeline of 4 cases of hepatitis A, Western Australian Goldfields Region, 2013

Annual reports

Australian Rotavirus Surveillance Program annual report, 2012

Carl D Kirkwood, Susie Roczo-Farkas, Ruth F Bishop, Graeme L Barnes, and the Australian Rotavirus Surveillance Group

Abstract

This report from the Australian Rotavirus Surveillance Program, together with collaborating laboratories Australia-wide, describes the rotavirus genotypes responsible for the hospitalisation of children with acute gastroenteritis during the period 1 January to 31 December 2012. During the survey period, 1,300 faecal samples were referred to the centre for rotavirus G and P genotype analysis, and of these 748 were confirmed as rotavirus positive. A total of 491 specimens were collected from children under 5 years of age, while 257 were from older children and adults. Genotype analysis revealed that G1P[8] was the dominant type in this reporting period, identified in 35% of strains nationally. Genotype G2P[4] was the second most common strain nationally, representing 28% of samples, followed by genotype G12P[8] (23%). This represents the first report where G12P[8] strains are a major cause of disease in this community. Fluctuations in genotype distribution were also observed based on the vaccine type in use. Genotype G2P[4] was more common in states and territories using Rotarix while G1P[8] was more common in states using RotaTeq. This survey of rotavirus strains circulating in 2012 highlights the continued fluctuations in rotavirus genotypes, with an annual change in dominant genotypes as well as emergence of a previously rare genotype, suggesting a dynamic wild-type population. *Commun Dis Intell* 2014;38(1):E29–E35.

Keywords: rotavirus, gastroenteritis, genotypes, disease surveillance

Introduction

Rotavirus is the major viral cause of severe diarrhoea in young children in all countries worldwide.[1] Rotaviruses belong to the Reoviridae family. They contain 11 segments of dsRNA that encode the 6 structural and 6 non-structural proteins. The significant morbidity and mortality associated with rotavirus infection led to the development of two live oral rotavirus vaccines Rotarix® (GlaxoSmithKline) and RotaTeq® (Merck). Large clinical trials with each vaccine have shown both to be safe and highly effective in the prevention of severe diarrhoea and hospitalisation due to rotavirus infections.[2,3]

Both rotavirus vaccines were included on the National Immunisation Program in Australia free of charge for all infants from 1 July 2007. Each state or territory selected one vaccine for use. RotaTeq is administered in Victoria, South Australia, Western Australia and Queensland, while Rotarix is administered in New South Wales, the Northern Territory, Tasmania and the Australian Capital Territory. Historically, rotavirus infection accounted for up to 10,000 childhood hospitalisations for diarrhoea each year in Australia.[4] The introduction of rotavirus vaccines has seen a significant impact on the disease burden, with national data showing a substantial decline in both rotavirus coded and non-rotavirus coded hospitalisations for diarrhoea since vaccine introduction.[5] State based studies in New South Wales, Queensland, South Australia and Victoria also reported major declines in hospitalisation and emergency room visits since vaccine introduction.[6–8]

The Australian Rotavirus Surveillance Program has reported annual changes in genotypes in the Australian population since 1997, with temporal and geographic changes observed each year.[9] The diversity of rotavirus strains capable of causing disease in children, and the patterns of emergence and circulation provide the baseline information vital to assist vaccine introduction and ongoing evaluation.

The introduction of rotavirus vaccines has increased population immunity to wild-type rotavirus strains. This in turn is likely to impact on the epidemiology of circulating strains. Therefore, investigation of circulating rotavirus genotypes will provide insight into whether vaccine introduction has impacted on virus epidemiology, and provide findings of the consequences of vaccination programs.

This report describes the genotype characterisation of rotavirus strains causing severe gastroenteritis in Australia for the period 1 January to 31 December 2012.

Methods

Rotavirus positive specimens detected by enzyme immunoassay (EIA) or latex agglutination in 21 collaborating laboratories across Australia were collected, stored frozen and forwarded to the National Rotavirus Reference Centre Melbourne, together with relevant age and sex details.

Viral RNA was extracted from a 10%–20% faecal extract prepared for each specimen using the QIAamp Viral RNA mini extraction kit (Qiagen) according to the manufacturer's instructions. The rotavirus G and P genotypes were determined for each sample by application of independent hemi-nested multiplex reverse transcription-polymerase chain reaction (RT-PCR) assays. The first round RT-PCR assays were performed using the Qiagen one step RT-PCR kit, using VP7 conserved primers VP7F and VP7R, or VP4 conserved primers VP4F and VP4R. The second round genotyping PCR reactions were conducted using specific oligonucleotide primers for G types 1, 2, 3, 4, 8, 9 and 12 or P types [4], [6], [8], [9], [10] and [11].[9–14] The G and P genotype of each sample was assigned using agarose gel analysis of second round PCR products.

Any samples that provided a discordant result between the initial antigen detection and genotype assay were further tested using the commercial rotavirus enzyme linked immunosorbent assay ProSpecT (Oxoid, UK), as per manufacturer's instructions to confirm the presence of rotavirus antigen.

Results

Number of isolates

During the period 1 January to 31 December 2012, a total of 1,300 specimens were received for analysis from 16 collaborating centres across Australia; located in Victoria, Western Australia, the Northern Territory, New South Wales, Queensland, South Australia, Tasmania and the Australian Capital Territory.

There were 748 samples confirmed as rotavirus positive by EIA (ProSpecT, OXOID) or RT-PCR analysis. Of these, 491 samples were collected from children under 5 years of age, and 257 samples were from older children and adults. An additional 552 specimens contained either insufficient specimen for genotyping (n=36), or the specimens were not confirmed to be positive for rotavirus (n=516) and were not analysed further.

Age distribution

In the current survey period, a total of 663 rotavirus positive specimens had patient age data available. In the cohort of children aged 5 years or less (n=402), 24.2% of cases were from infants 0–6 months of age, 12.3% were from infants 7–12 months of age, 29.1% from infants 13–24 months of age, 18.5% from infants 25–36 months of age, 6.2% from children 37–48 months of age and 8.9% from children 49–60 months of age.

There were 261 samples from older children and adults, 129 samples were obtained from children 5–10 years of age, 11 were from individuals 10–20 years of age, 91 were from individuals 21–80 years of age, and 30 were from individuals 80 years or older.

Genotype distribution

G1P[8] strains were the most common type identified nationally, representing 35% of all specimens (Table). This strain was identified in all states and territories and was the dominant type in the Northern Territory, Queensland and Victoria, representing between 33% and 65% of strains. It was also equally dominant in South Australia.

G2P[4] strains were the second most common genotype identified nationally, representing 28% of all specimens analysed. It was identified in 7 states and territories, and was the dominant type in New South Wales and the Australian Capital Territory and equal with G1P[8] in South Australia.

G12P[8] strains were the third most common genotype nationally, being identified in 4 states and territories representing 23% of strains. It was the dominant type in Western Australia, representing 43% of strains, and second most common type in the Northern Territory.

G3P[8], G4P[8] and G9P[8] strains each represented 5% or less of the total specimens genotyped (Table). Several rare or unusual genotypes were identified including a single G9P[4] strain identified in Queensland, a single G8P[nt] identified in South Australia. Strains which resembled a component of the RotaTeq vaccine were identified on 6 occasions from Western Australia, Victoria and South Australia. In addition, faecal specimens were received from 28 children who developed rotavirus gastroenteritis after being vaccinated with either RotaTeq or Rotarix. RotaTeq vaccine virus was identified in seven of these cases by RT-PCR and VP6 sequence analysis.

Sixteen samples contained multiple G and/or P genotypes, or a non-typeable G or P genotype. The non-typeable samples are likely to be samples that contain low virus levels, below the limits of our typing assays, or could have contained inhibitors in extracted RNA to prevent the function of the enzymes used in RT and/or PCR steps.

There were 261 confirmed rotavirus samples collected from older children and adults from 7 locations; New South Wales, the Northern Territory, Queensland, Western Australia, South Australia, Victoria and the Australian Capital Territory. The majority of these samples were collected from New

Table: Rotavirus G and P genotype distribution in Australian children ≤ 5 years, 1 January to 31 December 2012

Centre	Type total	G1P[8]		G2P[4]		G3P[8]		G4P[8]		G9P[8]		G9P[4]		G8P[14]		G10P[14]		G12P[8]		Mix*		G1P[6]		Non-type†		Vaccine (RotaTeq)		Neg	Insuff
		%	n	%	n	%	n	%	n	%	n	%	n	%	n	%	n	%	n	%	n	%	n	%	n	%	n	n	n
Australian Capital Territory																													
ACT	3	0	0	100	3	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	N/A	N/A	2	0
New South Wales																													
Sydney (POW)	20	10	2	80	16	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	10	2	N/A	N/A	0	0
Sydney (Westmead)	58	7	4	93	54	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	N/A	N/A	1	0
Newcastle John Hunter	28	0	0	100	28	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	N/A	N/A	19	0
Northern Territory																													
Alice Springs	23	30	7	0	0	0	0	0	0	0	0	0	0	0	0	0	0	61	14	0	0	0	0	9	2	N/A	N/A	18	6
Darwin	27	74	20	4	1	22	6	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	N/A	N/A	3	0
Western Diagnostic (NT)	2	50	1	0	0	50	1	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	N/A	N/A	0	0
PathWest WA	13	92	12	0	0	0	0	0	0	0	0	0	0	0	0	0	0	8	1	0	0	0	0	0	0	N/A	N/A	0	1
Queensland																													
Pathology (Brisbane)	12	83	10	8	1	8	1	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	44	1
Qld Regional	10	50	5	20	2	10	1	0	0	10	1	0	0	0	0	0	0	10	1	0	0	0	0	0	0	0	0	63	0
Pathology – Townsville	7	71	5	0	0	0	0	0	0	0	0	14	1	0	0	0	0	14	1	0	0	0	0	0	0	0	0	11	0
Pathology – -Gold Coast	1	0	0	0	0	0	0	0	0	100	1	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	7	0
South Australia																													
Adelaide	73	29	21	29	21	18	13	3	2	0	0	0	0	0	0	0	0	15	11	3	2	0	0	3	2	1	1	15	2
Tasmania																													
Hobart	1	100	1	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	N/A	N/A	3	0
Victoria																													
Melbourne Pathology	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	14	1
RCH	10	30	3	20	2	10	1	20	2	0	0	0	0	0	0	0	0	0	0	10	1	0	0	0	0	10	1	74	12
Monash	6	50	3	33	2	17	1	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	2	0
Western Australia																													
PathWest WA	161	40	64	4	7	2	3	4	6	2	3	0	0	0	0	0	0	42	67	1	2	0	0	3	5	2	4	8	2
Perth	36	42	15	3	1	0	0	3	1	3	1	0	0	0	0	0	0	50	18	0	0	0	0	0	0	0	0	60	0
Total	491	35	173	28	138	5	27	2	11	1	6	0	1	0	0	0	0	23	113	1	5	0	0	2	11	1	6	344	25

A total of 369 samples were omitted from analysis due to insufficient sample volume or sample was not confirmed as rotavirus positive.

Rotavirus vaccine, either Rotarix or RotaTeq, was identified in an additional 7 vaccinated infants, in whom samples were forwarded directly to ARSP directly from clinicians for analysis

Mix

PathWest, Western Australia: 1x G1/4P[8]; 1x G1/12P[8]

South Australia: 1x G1/3P[8]; 1x G3/9P[8]

RCH, Victoria: 1x G1/3/4P[8]

Non-typeables

Alice Springs, Northern Territory: 1x G1P[nt]; 1x GntP[nt]

PathWest, Western Australia: 3x G-ntP[8]; 2x G1P[nt],

Adelaide, South Australia: 1x G8P[nt]; 1x GntP[nt]

POW, New South Wales: 2x GntP[nt]

South Wales (n=72), Western Australia (n=77), South Australia (n=61) and Queensland (n=24). Genotype analysis of the rotavirus samples from older individuals showed a similar distribution to that observed in young children.

In New South Wales, the majority of the specimens were associated with G2P[4] in children 5–10 years of age. While in Western Australia and South Australia the majority of samples were G12P[8] or G2P[4] respectively, the same as the dominant type in children 5 years of age or younger in each location. A single G3P[14] strain was identified in an 11-year-old child from Victoria.

Analysis of G and P genotyping results revealed that in states where RotaTeq is in use, G1P[8] was the dominant genotype, identified in 40.6% of strains, while G12P[8] was second, identified in 31.6% of strains (Figure). G2P[4] was third most common representing 11.6% of strains. In states using Rotarix, G2P[4] strains were dominant (58.3%), while G1P[8] strains comprised 26.8% of specimens, and G12P[8] was identified in 8.6%. G3P[8] strains were identified at similar rates in both settings. G4P[8] and G9P[8] were only identified in states using RotaTeq vaccine, however, both represented minor types.

There appears to be consistency in genotype distribution within each vaccine type, for example, in 3 of the 4 RotaTeq states (Queensland, Victoria and South Australia), G1P[8] was the dominant type, and was second most common in the remaining location (Western Australia). Similarly, G2P[4] was dominant in 2 of the 3 states using Rotarix (New South Wales and the Australian Capital Territory).

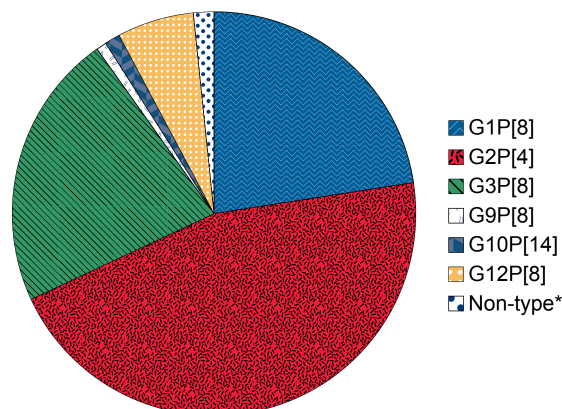
Discussion

The Australian Rotavirus Surveillance Program report for the period 1 January to 31 December 2012 describes the annual distribution of rotavirus genotypes and geographic differences in genotypes causing disease in Australian children.

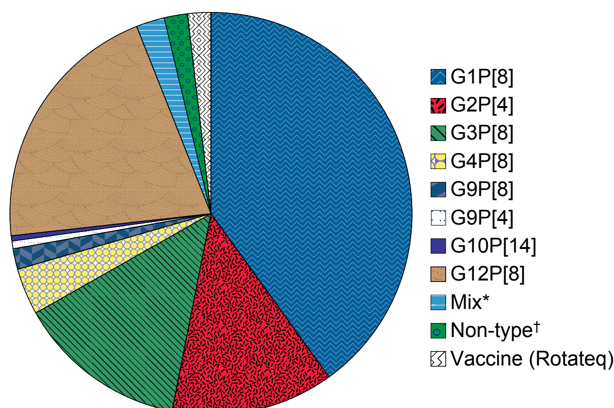
The surveillance program identified that genotype G1P[8] emerged as the dominant genotype nationally, representing 35% of all strains. Genotype G2P[4] was the second predominant type nationally, comprising 28% of all strains. Genotype G12P[8] represented the third most common genotype, representing more than 23% of strains nationally. As observed in the previous post-vaccine years, the dominant genotype continued to fluctuate on a yearly basis, with G1P[8] and G2P[4] constantly alternating.[15–17] In the 11 years pre-vaccine introduction, G1P[8] was the dominant type in 8 of the 11 rotavirus seasons.[9] In each of the post vaccine years, the dominant type

Figure: Overall distribution of rotavirus G and P genotypes identified in children, Australia, 1 January to 31 December 2012, by vaccine usage

Rotarix states



RotaTeq states



* Mix

PathWest, Western Australia: 1x G1/4P[8]; 1x G1/12P[8]

South Australia: 1x G1/3P[8]; 1x G3/9P[8]

RCH, Victoria: 1x G1/3/4P[8]

† Non-typeables

Alice Springs, Northern Territory: 1x G1P[nt]; 1x GntP[nt]

PathWest, Western Australia: 3x G-ntP[8]; 2x G1P[nt],

Adelaide, South Australia: 1x G8 P[nt]; 1x GntP[nt]

POW, New South Wales: 2x GntP[nt]

Rotarix was used in New South Wales, Tasmania, and the Northern Territory. RotaTeq was used in Victoria, South Australia, Western Australia and Queensland.

represented 50% of strains; however, this year the dominant genotype represented only 35% of strains across the country. This may be explained in part by the third most common genotype in this report representing more than 20% of strains (G12P[8]), when in past years the third most common strains fluctuated between 5% and 12%.

This report represents the first occasion that G12P[8] strains have been a major cause of disease

in Australian children. Previously, G12 strains have been identified in a small outbreak in New South Wales during 2005, and as single isolates in Melbourne and Sydney in 2006–07.[18] The emergence of G12P[8] strains in 4 locations in 2012 represents the largest distribution of this genotype observed in Australia. Globally, G12 strains represent a minor type, identified in less than 2% of all strains genotyped from 2003 to 2007[19] G12 strains were recently identified in Belgium in 2007–08, the second season after vaccine introduction.[20] In a recent efficacy trial in South Africa and Malawi, Rotarix was shown to provide comparable protection against a range of circulating genotypes including G12 strains.[21] Both rotavirus vaccines are likely to be effective against the emergence of G12P[8] strains.

The use of different vaccines in Australian states and territories provides a unique opportunity to compare the effect of each vaccine on the circulating wild-type strains. Differences in genotype distribution have been observed during the past 5 years. During years 1 and 2, G2P[4] strains were more common in states and territories using Rotarix, while G3P[8] were more common in RotaTeq locations. During years 3 and 4, the pattern changed such that G2P[4] strains were more common in states using RotaTeq, while G1P[8] strains were more common in locations using Rotarix. G3P[8] remained more common in RotaTeq states only in year 3, after which they occurred at similar rates in years 4 and 5. In the current survey, (year 5), the occurrence of G2P[4] reverted to that observed in years 1 and 2, being more common in locations using Rotarix.[15,17] Thus differences were evident in genotype distribution, but there was no consistent genotype distribution linked to a particular vaccine.

The worldwide interest in uncommon rotavirus genotypes continues because of the possible impact they could have on rotavirus vaccine programs. In previous years, uncommon VP7/VP4 genotype combinations have been identified, and this year the emergence of G12 further highlights this emergence of different genotypes. In 2011, G10P[14] strains were identified in the Northern Territory causing acute gastroenteritis in 5 infants and 1 adult. This represented the first report of this genotype combination in Australia. Full genome sequence analysis identified that the virus was likely the result of an *Artiodactyl*-to-human interspecies transmission.[22] Whether the introduction of vaccine is exerting an increase on immune pressure or simply natural variation is still unclear, but the identification of G10 and G12 strains strengthens the need to continue rotavirus surveillance in both humans and animals.

This report again details a significant number of rotavirus positive samples in older children and adults. A large rotavirus outbreak caused by G2P[4] occurred in New South Wales in 2012, occurring predominantly in children aged 5–9 years. The rates of gastroenteritis in this age group were significantly higher levels than in previous years (J Musto, personal communication). This report also continues the previous reports of an increase in adult rotavirus cases observed in South Australia and Western Australia, as well as in other locations.[23] A reduction in circulating virus in the post-vaccine era may have led to a decrease in protection from rotavirus in older unvaccinated children or adults.

This survey of rotavirus strains causing disease between 1 January and 31 December 2012 highlights the continued fluctuations in rotavirus genotypes across Australia. However, the genotype patterns continue to change on an annual basis and illustrate a more dynamic wild-type population than observed in the pre-vaccine era. This suggests that vaccine pressure may be speeding up the selection process. This is supported by the observation of G12P[8] strains and cases in older children and adults. Therefore, on-going surveillance of the wild-type strains circulating in Australia is required to monitor any changes that may emerge and impact vaccine effectiveness.

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The participating laboratories for this sampling period were:

- Princess Margaret Hospital for Children, Subiaco, Western Australia
- Division of Microbiology, PathWest LM, Western Australia
- Queen Elizabeth Medical Centre, Nedlands, Western Australia
- Microbiology Department, Royal Darwin Hospital, Casuarina, Northern Territory

- Department of Microbiology, Western Diagnostic Pathology, Northern Territory and Western Australia
- Microbiology Department, Alice Springs Hospital, Alice Springs, Northern Territory
- Virology Division, SEALS, Prince of Wales Hospital, New South Wales
- Microbiology Department, The Children's Hospital at Westmead, New South Wales
- Centre for Infectious Diseases and Microbiology, Sydney, New South Wales
- Microbiology Department, John Hunter Hospital, Newcastle, New South Wales
- Forensic and Scientific Services, Queensland Health Herston, Queensland
- Pathology Queensland, Herston, Queensland
- Queensland Paediatric Infectious Diseases Laboratory, Queensland
- Royal Children's Hospital, Brisbane, Queensland
- Queensland Health laboratories in Townsville, Cairns and Gold Coast, Queensland
- Virus Laboratory Institute of Medical and Veterinary Science, Adelaide, South Australia
- Royal Hobart Hospital and the Communicable Disease Prevention Unit, Tasmania
- Department of Health and Human Services, Hobart, Tasmania
- ACT Pathology, Canberra, Australian Capital Territory
- Virology Department, Royal Children's Hospital, Parkville, Victoria
- Department of Microbiology, Melbourne Pathology, Victoria

The National Rotavirus Surveillance Group includes:

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Dr S Lambert, Ms N George, Ms S Ye and members of the Queensland Paediatric Infectious Diseases laboratory, Royal Children's Hospital, Brisbane

Mr R Enbom, Ms G Gilmore, Ms P Derrington and members of the Queensland Health laboratories in Townsville, Cairns and Gold Coast

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Prof G Higgins, Ms S Schepetiuk, Ms L Payne and members of the Virus Laboratory Institute of Medical and Veterinary Science, Adelaide

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Tuberculosis notifications in Australia, 2010

Christina Bareja, Justin Waring, Richard Stapledon and the National Tuberculosis Advisory Committee, for the Communicable Diseases Network Australia

Abstract

The National Notifiable Diseases Surveillance System received 1,353 tuberculosis (TB) notifications in 2010, representing a rate of 6.1 cases per 100,000 population. While rates of 5 to 6 cases per 100,000 population for TB have been maintained in Australia, since first achieved in the mid-1980s, there has been a steady increase in incidence over the past decade. The incidence in the Australian-born Indigenous population was 7.5 per 100,000 population, which is 11 times the incidence reported in the Australian-born non-Indigenous population of 0.7 per 100,000 population. Overseas-born people accounted for 90% of all cases notified in 2010 and represented a rate of 24 per 100,000 population. International students have been recognised as an increasingly important group, representing 25% of all overseas-born cases notified in 2010, and are a focus of this report. Household or other close contact with TB or past residence in a high risk country were the most commonly reported risk factors for TB infection. Outcome data for the 2009 TB cohort indicate that treatment success was attained in more than 95% of cases. As Australia continues to contribute to global TB control it is important to maintain good centralised national reporting of TB to identify populations at risk and monitor trends in TB. *Commun Dis Intell* 2014;38(1):E36–E48.

Keywords: Australia, tuberculosis, communicable disease surveillance; epidemiology, annual report

Introduction

There were 8.8 million incident cases of tuberculosis (TB) globally in 2010, with almost 60% of these cases occurring in Australia's neighbouring countries in South East Asia and the Western Pacific.[1] For the first time, the World Health Organization (WHO) reported in 2010 that both the absolute number of TB cases and TB incidence rates had been falling at a global level. The on-going success of the Stop TB Strategy in our region and globally is important to a low-incidence country like Australia where the TB burden is largely a function of migration.

A crucial component of effective TB control in Australia is the collection of accurate, comprehensive and timely surveillance data. Surveillance of TB in Australia is overseen by the National Tuberculosis Advisory Committee (NTAC), a subcommittee of the Communicable Diseases Network Australia (CDNA). NTAC has the key role of providing expert strategic advice to CDNA on a coordinated national approach to TB control. NTAC also has the role of developing and reviewing nationally agreed strategic and implementation plans for the control of TB in Australia. NTAC relies on quality surveillance data to inform these evidence-based policies.

This report describes the epidemiology of notified cases of TB in Australia in 2010. This report should be considered in conjunction with the Australian Mycobacterium Reference Laboratory Network (MRLN) report on bacteriologically confirmed cases.[2]

Methods

TB is a nationally notifiable disease in Australia and is monitored using the National Notifiable Disease Surveillance System (NNDSS). Medical practitioners, public health laboratories and other health professionals are required under state and territory public health legislation to report cases of TB to jurisdictional health authorities. The *National Health Security Act 2007* provides the legislative basis for the national notification of communicable diseases and authorises the exchange of health information between the Commonwealth and states and territories. State and territory health departments transfer these notifications regularly to the NNDSS. The primary responsibility for public health action resulting from notification resides with state and territory health departments.

The Tuberculosis Data Quality Working Group (TBDQWG) is a working group of NTAC. It has representation from states and territories, the Commonwealth and the MRLN. It ensures routine and timely reporting of trends and emerging issues in TB. The TBDQWG is also responsible for maintaining national consistency and currency in data standards and systems for TB surveillance, relied upon to produce this report.

Data presented in this report represent a point in time analysis of notified cases of TB. This report presents data extracted from NNDSS during

February 2012, with revised data from Tasmania finalised in July 2012 and from New South Wales finalised in November 2012. Due to the dynamic nature of the NNDSS, data in this report may vary from data reported in other NNDSS reports and reports of TB notifications at the state or territory level. Detailed notes on case definition, data collection, quality control and the categorisation of population subgroups are available in the 2007 annual report.[3]

This report presents data analysed by date of diagnosis. This is a derived field within the NNDSS that is the earliest of the reported fields of notification date and notification received date. Crude rates were calculated using the mid-year estimated resident population from the Australian Bureau of Statistics (ABS).[5] Rates specific to population subgroups were based on ABS estimated resident populations as at 30 June 2006, however rates of specific countries of birth were based on the estimated resident population as at 30 June 2010.[5,6]

Results

Epidemiological situation in 2010

In 2010, 1,353 cases of TB were reported to the NNDSS, representing a rate of 6.1 cases per 100,000 population (Figure 1 and Table 1). This is an increase of 3% in the number of notified cases compared with 2009 (n=1,313). While the low rate of TB first achieved in the mid-1980s has been maintained, there has been a steady increase in incidence over the decade leading up to 2010.

A case classification was reported in almost all cases (n=1,350) reported in 2010. Of those with a case classification, the majority of cases were classified as new (96%, n=1,298), that is, a patient who has never been treated for TB or a patient that was treated previously for less than 1 month (Table 1). Relapse was reported in 51 cases, with 12 of those cases reported as relapsing following full treatment in Australia, six following partial treatment in Australia and 33 following full or partial treatment overseas. One case reported by South Australia was reported as treatment after failure, which is a patient who has been treated after remaining culture positive 5 months or more following previous treatment. A report on the treatment after failure case is provided in the Box.

Geographic distribution

As in previous years, New South Wales accounted for the largest number of cases notified by a state or territory (n=518; Table 1). The Australian Capital Territory and Tasmania reported the smallest number of cases in 2010 (n=10, per jurisdiction). The highest jurisdiction-specific rate in 2010 was

Figure 1: Notification rates of tuberculosis, Australia, 1960 to 2010

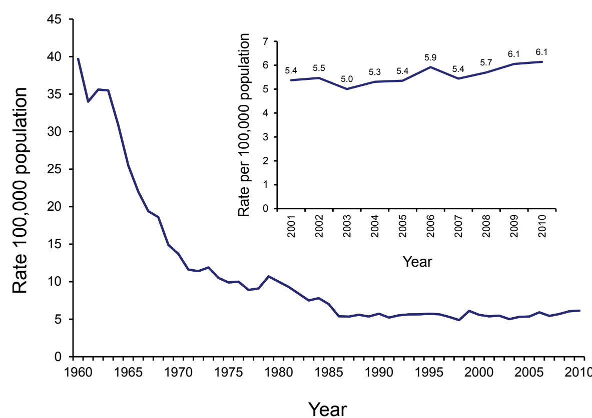


Table 1: Notified cases and rates of tuberculosis, Australia, 2010, by case classification and state or territory

State or territory	New cases		Relapse cases		Total*	
	Notifications	Rate	Notifications	Rate	Notifications	Rate
ACT	9	2.5	0	–	10	2.8
NSW	492	6.9	24	0.3	518	7.3
NT	28	12.2	0	–	28	12.2
Qld	171	3.9	8	0.2	179	4.1
SA	68	4.2	4	0.2	73	4.5
Tas.	10	2.0	0	–	10	2.0
Vic.	420	7.7	9	0.2	429	7.9
WA	100	4.4	6	0.3	106	4.6
Aust.	1,298	5.9	51	0.2	1,353	6.1

* Total includes 1 case of treatment after failure reported by South Australia, and 3 cases reported without a case classification (1 reported by the Australian Capital Territory and 2 by New South Wales).

Box. Case report: Treatment after failure

A case of 'treatment after failure' was the first to have occurred in South Australia in at least the past 2 decades. This arose in an insulin dependent diabetic with pulmonary smear positive disease and initial high level isoniazid resistance. Treatment was supervised throughout by directly observed therapy (DOT). Sputum cultures were negative at the completion of the 2 month intensive phase, but again positive at the completion of nine months of treatment with rifampicin, ethambutol and pyrazinamide (REZ). Drug susceptibility testing confirmed acquired rifampicin resistance, making this a multi-drug-resistant TB (MDR-TB) case. The initial isolate in 2009 was susceptible to rifampicin at 0.25, 0.5, 1.0 and 2.0 mg/L (with the recommended Mycobacterium Growth Indicator Tube, breakpoint for susceptibility being 1.0 mg/L) and contained no rpoB mutation. The later isolate in 2010 was resistant to rifampicin at all 4 concentrations and contained an rpoB mutation known to confer resistance. The 2 isolates had the same12-loci mycobacterial interspersed repetitive units profile. The patient was changed to an appropriate 2nd line regimen and subsequently also had a left upper lobectomy at 2 months to 'debulk' localised disease.

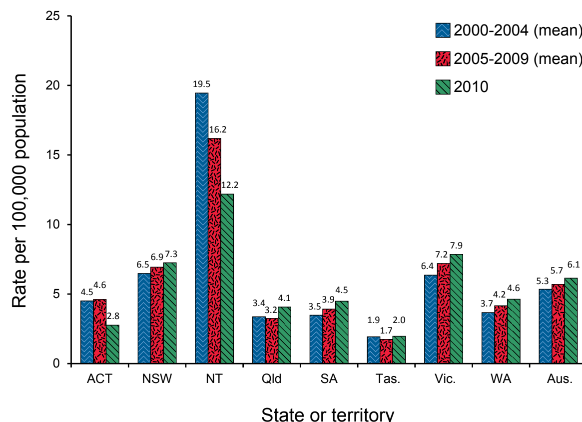
reported by the Northern Territory (12 per 100,000 population), followed by Victoria (7.9 per 100,000 population) and New South Wales (7.3 per 100,000 population).

Compared with the preceding 10 years, the Australian Capital Territory reported its lowest annual rate in 2010 (2.8 per 100,000 population, Figure 2), while Queensland (4.1 per 100,000 population) and Victoria both reported their highest jurisdiction-specific rate. Despite reporting the highest jurisdiction-specific rate in 2010, the rate of TB in the Northern Territory has continuously declined over the past 10 years, with the exception of 2007.

Tuberculosis in the Australian-born population

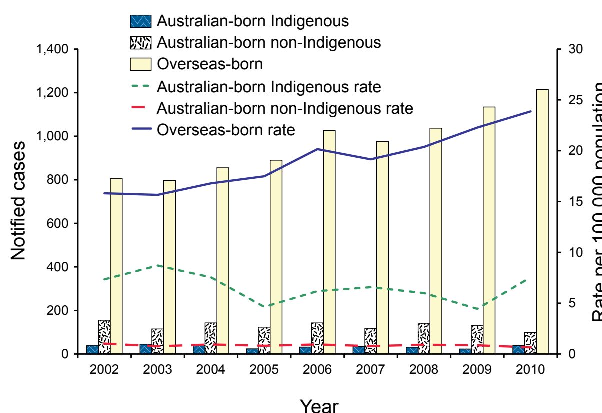
Indigenous status was reported for each of the 138 Australian-born cases reported in 2010 (Table 2). The overall rate of TB in the Australia-born population in 2010 was 0.9 per 100,000 population. The rate in the Australian-born Indigenous population (7.5 per 100,000 population) was 11 times the rate reported in the Australian-born non-Indigenous population (0.7 per 100,000 population).

Figure 2: Notification rates of tuberculosis, Australia, 2000 to 2010, by state or territory



The rate of TB in Australian-born non-Indigenous people has remained relatively stable since 2002 (Figure 3), while the rate in Australian-born Indigenous peoples has demonstrated no clear trend, ranging from 4.4 to 8.7 cases per 100,000 population over this period.

Figure 3: Notified cases and rate of tuberculosis, Australia, 2002 to 2010, by population subgroup



Tuberculosis in the overseas-born population

All cases of TB diagnosed in 2010 were reported with a country of birth, with 90% (n=1,215) of cases reported as overseas-born (Table 2). The proportion of cases that were reported as overseas-born varied across states and territories, ranging from 39% (n=11) in the Northern Territory to 95% (n=409) in Victoria and 100% (n=10) in the Australian Capital Territory.

Table 2: Notified cases and rates of tuberculosis, Australia, 2010, by population subgroup and state or territory

State or territory	Australian-born						Overseas-born	
	Indigenous		Non-Indigenous		Total			
	Notifications	Rate	Notifications	Rate	Notifications	Rate	Notifications	Rate
ACT	0	–	0	–	0	–	10	12.7
NSW	11	7.2	53	1.1	64	1.3	454	25.1
NT	15	23.4	2	1.8	17	9.6	11	33.5
Qld	8	5.5	9	0.3	17	0.5	162	20.1
SA	1	3.6	7	0.6	8	0.7	65	18.9
Tas.	1	5.4	1	0.2	2	0.5	8	14.2
Vic.	1	3.0	19	0.5	20	0.5	409	30.3
WA	2	2.8	8	0.6	10	0.7	96	15.6
Aust.	39	7.5	99	0.7	138	0.9	1,215	23.9

The rate of TB amongst overseas-born people in 2010 was 27 times the rate in Australian-born. The rate of TB in overseas-born people in 2010 was the highest reported in this population group since 2002,with the group experiencing a consistent, steadily increasing trend over this period (Figure 3). This figure should be interpreted with caution, given that completeness of reporting country of birth has improved over this time.

Amongst overseas-born cases notified in 2010, the most frequently reported country of birth was India (n=302, Table 3), followed by Vietnam (n=114), the Philippines (n=91) and Nepal (89). Amongst the most frequently reported countries of birth, the highest estimated rates were amongst those cases born in Somalia (297 per 100,000 population), Nepal (289 per 100,000 population) and Papua New Guinea (PNG) (202 cases per 100,000 population). These estimates of rates must be interpreted with caution as they are biased by temporary visitors being included amongst notified cases but are not necessarily enumerated within the estimated resident population.

Residency status was available for 95% (n=1,152) of TB cases reported as overseas-born in 2010. Residency status is self-reported at the time of diagnosis and is not verified against migration records. The majority of overseas-born cases reported with a residency status were reported as permanent residents (n=603), followed by overseas students (n=282). Of the high-burden overseas-born populations identified in Table 3, the proportion of cases reported as overseas students ranged from 0% of cases born in the Republic of the Union of Myanmar, Somalia, Sudan and Afghanistan to 69% of cases born in Nepal.

There were 36 cases of TB notified amongst PNG nationals accessing health care in the Torres Strait Treaty Zone in 2010. This was a 50% increase on the 24 cases reported in 2009. Treating PNG nationals in the Torres Strait Treaty Zone accounted for 20% of Queensland's caseload in 2010. There was only 1 illegal foreign fisher reported with TB in 2010, similar to the low number of cases in this group in 2009.

Data on the year of arrival were available for 82% (n=996) of the cases reported as overseas-born in 2010. Of these cases, 29% (n=284) presented and were diagnosed within 2 years of arrival in Australia (Figure 4). In 2010 this was the first year where international students contributed to a peak of disease after 2 years of arrival in Australia. Half of all TB cases in the overseas-born occurred within 3 years of arrival in Australia (inter-quartile range, IQR: 1–12 years).

Figure 4: Notified cases of tuberculosis in the overseas-born population, Australia, 2010, by number of years since arrival in Australia

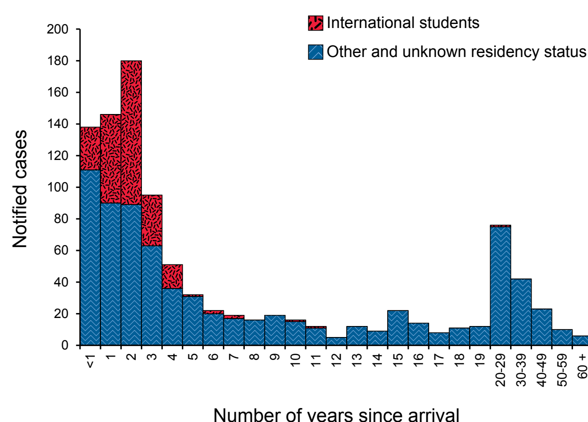


Table 3: Notified cases and rates of tuberculosis for frequently reported countries of birth, Australia, 2010, by residency status

Country of birth	Residency status				Estimated resident population‡	Estimated rate (per 100,000 population)	WHO rate (per 100,000 population)§
	International students*		Other and unknown residency status†	Total			
	n	%					
India	91	30	211	302	343,150	88	185
Vietnam	24	21	90	114	210,090	54	199
Philippines	3	3	88	91	178,570	51	275
Nepal	61	69	28	89	30,840	289	163
China	23	28	59	82	376,980	22	78
Papua New Guinea	5	8	56	61	30,210	202	303
Indonesia	17	33	35	52	68,960	75	189
Bangladesh	9	28	23	32	28,770	111	225
Thailand	8	32	17	25	53,070	47	137
Republic of the Union of Myanmar	0	0	20	20	22,480	89	384
Somalia	0	0	19	19	6,400	297	286
Sudan	0	0	18	18	25,940	69	119
Republic of South Korea	5	29	12	17	89,210	19	97
Pakistan	7	41	10	17	31,890	53	231
Cambodia	1	6	15	16	31,220	51	437
Malaysia	2	13	14	16	129,980	12	82
Afghanistan	0	0	16	16	27,140	59	189
Other overseas-born	26	11	202	228			
Total overseas-born	282	23	933	1,215			
Australian-born	–		–	138			
Total				1,353			

* Students presented as a per cent of total cases, by country of birth.

† An unknown residency status was reported in 5% of overseas-born cases.

‡ The Australian Bureau of Statistics estimated resident population at 30 June 2010, using ABS catalogue 3412.0.[6]

§ Rates from the World Health Organization 2011 Global Tuberculosis Report.[1]

|| China excludes Special Administrative Regions and Taiwan.

Age and sex distribution

Age was reported for all TB cases notified in 2010, while sex was available for close to 100% of cases (n=1,349). Continuing the trend reported in previous years, there were more males than females notified, with a male to female ratio of 1.2:1.

In 2010, TB was predominantly seen in young adults aged 25–34 years, and this was driven by high rates in overseas-born cases in this age group (Figure 5). The burden of disease increased with age in Australian-born cases regardless of Indigenous status.

One of the most important measures of TB control is the incidence in children aged less than 15 years because these cases represent recent TB infection. TB was notified in 49 children aged less than 15 years in 2010, which equated to 4% of the total number of notified cases. Of these, 18 were Australian-born non-Indigenous people including 5 cases reported as having one or more parents born in a high-risk country. An additional 5 cases were Australian-born Indigenous and 26 cases were children born overseas.

Selected risk factors for tuberculosis

Selected risk factor data were provided for 73% (n=985) of notified cases in 2010. Overall, the most frequently reported risk factor was past travel to or residence in a high-risk country that was not their country of birth (n=631, Table 4). Interpretation of this risk factor in overseas-born cases is problematic. At the time these data were collected there were inconsistent practices across states and territories as to the inclusion of a case's country of birth in the assessment of this risk factor. NTAC has agreed that this risk factor is to identify travel-related TB and as such is intended to be exclusive of a case's country of birth. Reporting of this risk factor from 2013 should be clearer.

Having a household member or close contact with TB was the 2nd most common risk factor

Figure 5: Notification rates of tuberculosis, Australia, 2010, by population subgroup and age group

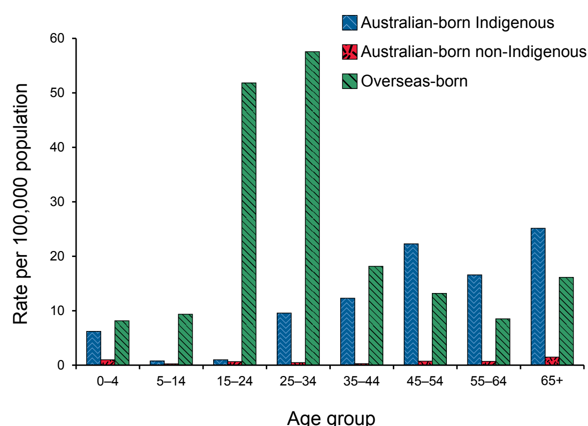


Table 4: Notified cases of tuberculosis, Australia, 2010, by population subgroup and selected risk factors*

Risk factor*	Australian-born Indigenous	Australian-born non-Indigenous	Overseas-born	Total
Household or other close contact with TB	20	22	156	198
Ever resided in a correctional facility†	1	0	5	6
Ever resided in an aged care facility†	0	0	0	0
Ever employed in an institution†,‡	0	2	8	10
Currently or previously† employed in health industry in Australia or overseas	1	4	52	57
Ever homeless	1	0	2	3
Past travel to or residence in a high-risk country	2	23	606	631
Chest X-ray suggestive of old untreated TB	1	3	4	8
Currently receiving immunosuppressive therapy	0	1	9	10
Australian-born child with one or more parent born in a high-risk country	0	4	–	4
None of the above risk factors	4	27	105	136
Total cases assessed for risk factors	29	79	877	985

* More than 1 risk factor may be reported for each notified case of tuberculosis.

† Within the preceding 5 years.

‡ Institution is defined as a correctional facility, aged care facility or homeless shelter.

reported by Australian-born non-Indigenous and overseas-born cases (n=22 and 156, respectively), and it was the most common risk factor reported by Australian-born Indigenous cases (n=20).

A total of 57 cases of TB in 2010 were reported in people who were currently or had previously worked in a health care setting. Of these, 13 were working in a health care setting in Australia at the time of diagnosis or within 12 months of diagnosis. Almost half (n=6) of these cases presented with extrapulmonary disease only, which is generally not communicable. None of these notified cases were deemed to have acquired TB in an Australian health care setting, nor were there any reports of active TB transmission to patients from health care workers in Australia in 2010.

There were a number of cases of TB reported as not having any risk factors identified (n=136).

Tuberculosis and HIV status

The HIV testing history of notified cases of TB was reported in 90% of cases (n=1,215, Table 5). More than half of these cases (n=654) were reported with a known HIV status, of which 3.4% (n=22) were reported as HIV positive. Of the cases with a known HIV status, Australian-born non-Indigenous cases were reported more frequently as HIV positive (4.9%) than Australian-born Indigenous (0%) and overseas-born cases (3.4%). Approximately one-third (n=368) of cases with an HIV testing history were reported as being tested with an unknown result. These cases were almost entirely reported by Victoria, where the HIV status of an individual cannot be reported against their TB notification.

Anatomical site of disease

The anatomical site of TB disease was recorded for all notified cases in 2010 (Table 6). Pulmonary disease was the most frequently reported site of disease (n=830), with most of these cases reported as having pulmonary disease only.

Cases presenting with disease focused in extrapulmonary sites only accounted for 39% (n=523) of cases, with lymph nodes (n=178) and pleura (n=59) reported as the most frequent extrapulmonary sites. Of the more severe forms of TB, there were four classified as miliary and six as meningeal cases. While the treatment outcomes of these severe forms of TB were not finalised at the time of the data being finalised for this report, preliminary data suggested that of the cases with assessable outcomes (n=4), 75% were reported as having completed treatment. Of the remaining cases with non-assessable outcomes (n=6), two-thirds were still under treatment and one-third was transferred overseas and therefore their treatment outcome is unknown.

All extrapulmonary only cases reported with an unknown site were reported by Victoria.

Bacteriologically confirmed cases and drug resistance

The number of cases confirmed bacteriologically by culture of the *Mycobacterium tuberculosis complex* was 1,051 (78%). Of the 830 pulmonary TB cases, 77% (n=642) were culture positive, of which 47% (n=304) were smear positive. Bronchoscopy was used to establish the diagnosis in 137 (21.3%) of the culture proven cases of which 27% (n=37) were smear positive. Five (3.6%) of these bronchoscopy cases were MDR-TB. Of the 523 extra-pulmonary only cases, 64% (n=333) were confirmed by bacteriological means. In children under 15 years, 49 were notified but only 12 (25%) were confirmed by the laboratory. The proportion of culture isolates with available *in vitro* drug susceptibility testing, demonstrating resistance to at least one of the standard first line anti-tuberculous agents was 12% (n=126). Resistance to isoniazid (no rifampicin resistance) was shown in 4.7% (n=49) of isolates. Resistance to at least isoniazid and rifampicin (MDR-TB by definition) was reported in 3.5% (n=37) of cases but 16 of these were from the PNG-Torres Strait Islands cross border region. Overseas-born persons accounted for all of the

Table 5: Notified cases of tuberculosis, Australia, 2010, by population subgroup and HIV status

HIV testing history	Australian-born Indigenous	Australian-born non-Indigenous	Overseas-born	Total
HIV positive	0	3	19	22
HIV negative	29	48	555	632
HIV tested, result unknown*	0	10	358	368
Not tested	4	23	163	190
Refused testing	0	0	3	3
HIV testing history unknown	6	15	117	138
Total	39	99	1,215	1,353

Table 6: Notified cases of tuberculosis, Australia, 2010, by case classification and site of disease

Site of disease	New cases	Relapse cases	Total*	Per cent of cases
Total pulmonary disease	789	38	830	61.3
Pulmonary only	622	34	659	48.7
Pulmonary plus other sites	167	4	171	12.6
Total extrapulmonary only†	509	13	523	38.7
Pleural	59	0	59	4.4
Lymph nodes	155	6	178	11.9
Bone/joint	33	0	35	2.4
Genito/urinary	26	0	27	1.9
Miliary	3	1	7	0.3
Meningeal	5	1	6	0.4
Peritoneal	6	1	14	0.5
Other	62	1	79	4.7
Unknown extrapulmonary site	174	3	174	12.9

* Total includes 3 pulmonary cases (1 reported as treatment after failure and 2 reported without a case classification) and 1 extrapulmonary case reported without a case classification.

† More than 1 extrapulmonary site may be reported for each notified case of TB.

remaining cases including the Philippines (4), India (3), China (2) and Vietnam (3). Extensively drug resistant TB (MDR-TB plus resistance to an injectable agent and a quinolone) was reported in 1 overseas born case and a further case had MDR and quinolone resistance.

A more detailed analysis of bacteriologically confirmed cases, including the drug resistance profiles of isolates, is reported in the Australian Mycobacterium Reference Laboratory Network (MRLN) report.[3]

Treatment outcomes of 2009 tuberculosis patient cohort

Treatment success, including those with bacteriologically confirmed cure and those who completed treatment was reported in 96% (n=1,136) of cases reported with assessable outcomes in 2009 (Table 7). Treatment success by population subgroup ranged from 90% (n=18) in Australian-born Indigenous cases to 96% (n=981) in overseas-born cases. There were 2 cases of a treatment failure reported in cases notified in 2009 and a total of 15 cases reported to have died due to TB.

National performance indicators

The performance criterion for incidence (less than 1 per 100,000 population) developed by NTAC, was met only for the crude incidence rates in non-Indigenous cases (Table 8). Incidence rates in children exceeded the performance criteria (less than 0.1 case per 100,000 population) in all population groups. While the reporting of HIV testing history has declined compared with the previous year, it has improved in recent years and is close to reaching the target of 100%. Outcome reporting came close to meeting the target of 100% for the 2009 patient cohorts, with less than 1% of cases with assessable outcomes reported with an unknown outcome. The performance indicator for cases that reported treatment success was met in 2009. Additionally, this performance indicator was met in each of the population subgroups, including Australian-born Indigenous cases (Table 7).

Discussion

The TB incidence rate in Australia of 6.1 per 100,000 population is low when compared with other countries, including comparable countries with well-developed and resourced health systems. In 2010, TB incidence in the United States of America (USA) was 4 per 100,000 population, the United Kingdom was 15 per 100,000 population and New Zealand was 8 per 100,000 population.[1] This report records a slight increase in the number of notifications and incidence rate compared with 2008 and 2009. This continues the overall trend toward an increase in incidence, as illustrated when the rate is compared with the mean for the 2 preceding intervals of 5 years (Figure 2). This upward trend is particularly noted in New South Wales, Queensland, South Australia, Victoria and Western Australia. It is also in stark contrast with the USA, where there has been a steady decline over the last 10 years.[7]

Table 7: Notified cases of tuberculosis, Australia, 2009, by population subgroup and treatment outcome

Treatment outcome	Australian-born Indigenous		Australian-born non-Indigenous		Overseas-born		Total cases*	
	Notifications	% assessable	Notifications	% assessable	Notifications	% assessable	Notifications	% assessable
Assessable outcomes								
Treatment success	18	90.0	115	95.0	981	95.8	1,136	95.5
Cured (bacteriologically confirmed)†	3	15.0	5	4.1	37	3.6	50	4.2
Completed treatment	15	75.0	110	90.9	944	92.2	1,086	91.3
Interrupted treatment‡	0	–	0	–	2	0.2	2	0.2
Died of tuberculosis	0	–	2	1.7	12	1.2	15	1.3
Defaulted§	2	10.0	1	0.8	23	2.2	27	2.3
Failure‖	0	–	1	0.8	1	0.1	2	0.2
Not followed up, outcome unknown	0	–	2	1.7	5	0.5	7	0.6
Total assessable	20	100.0	121	100.0	1,024	100.0	1,189	100.0
Non-assessable outcomes								
Transferred out of Australia	0	0.0	0	–	65	5.7	65	
Died of other causes	1	4.3	9	6.9	37	3.3	47	
Still under treatment	2	8.7	0	–	8	0.7	12	
Total	23	100.0	130	100.0	1,134	100.0	1,313	

* Total includes 26 cases reported with an unknown population subgroup.

† Cured is defined as the bacteriologically confirmed sputum smear and culture positive at the start of treatment and culture negative in the final month of treatment and on at least 1 previous occasion.

‡ Interrupted treatment is defined as treatment interrupted for 2 months or more but completed.

§ Defaulted is defined as failed to complete treatment.

|| Failure is defined as sputum culture positive at 5 months or later.

Table 8: National tuberculosis performance indicators, performance criteria* and the current status of tuberculosis, Australia, 2009 and 2010

National tuberculosis performance indicator	Performance criterion	2009	2010
Annual incidence of TB (cases per 100,000 population)			
Australian-born Indigenous Australians	< 1	4.4	7.5
Australian-born non-Indigenous Australians	< 1	0.9	0.7
Overseas-born persons	*	22.3	23.9
Incidence in children <15 years, by risk group (per 100,000 population)			
Australian-born Indigenous Australians	< 0.1	1.5	2.6
Australian-born non-Indigenous Australians	< 0.1	0.6	0.5
Overseas-born persons	*	10.6	9.2
Collection of HIV status in tuberculosis cases	100%	95%	90%
Treatment outcome measures (%)			
Cases evaluated for outcomes	100%	91%	TBA
Cases that have treatment completed and are cured	>90%	96%	TBA
Cases recorded as treatment failures	<2%	0.2%	TBA

* Performance criteria currently under review.

TBA To be assessed; treatment outcomes for 2010 patient cohort to be reported in the 2011 annual report.

TB cases in Australia are nearly all new cases, meaning there is no history of prior TB treatment. Only 4% of TB notifications in 2010 were relapsed TB, of which 35% (18 of 51) had previously been treated in Australia. From the data available, it is not clear whether these cases are genuine relapse or reinfection, and the details of the original treatment that may explain relapse, particularly amongst the 12 cases that were considered to have been fully treated in Australia, is also not available. Whether these cases of relapse were associated with acquired drug resistance is also not reported, but NTAC is investigating separately the incidence and characteristics of acquired drug resistance in Australia. Persistence of positive sputum culture despite at least 5 months of treatment, i.e. treatment failure, is extremely rare in Australia. However in 2010 there was a single case in which sputum cultures remained positive after 5 months treatment.

The TB rate in Australian-born people as a whole remains very low, but remains unacceptably high in Australian-born Aboriginal and Torres Strait Islander people. In 2010 the rate in Australian-born Indigenous persons was 11 times the non-Indigenous Australian-born rate and was higher than the previous 5 years (Figure 3). This is contrary to a prior trend downwards, though interpreting trends in this group is difficult, because of the low numbers. Encouragingly, the rate in the Northern Territory, where more than half of cases are Aboriginal, has fallen, which suggests improved control in that population.

The main contributor to TB incidence in Australia and the trend toward increasing TB notifications is increasing levels of migration from high incidence countries. Ninety per cent of TB notifications in 2010 were in overseas-born people, and this proportion and the rate in overseas-born people have steadily increased over the last 10 years. Overseas-born people with TB mainly come from high TB incidence countries from which Australia receives a large number of immigrants, specifically India, Vietnam and the Philippines. Notably, there has been an increasing number of TB cases amongst Nepalese immigrants, who are largely students undertaking tertiary education in Australia. TB notifications in people born in Nepal, together with Somalia, PNG, Bangladesh and Myanmar have the highest rates as a function of the low estimated population of these people resident in Australia. People originating from countries with high rates, such as these, are a target population for screening and an increased vigilance for TB amongst clinicians.

There are a number of other characteristics associated with TB notification in addition to country of birth, which can assist with identifying target groups for future TB control activities. In particular, although TB can occur many years after migration, in TB annual reports over many years it has been consistently noted that half of the TB cases in overseas-born people are young adults diagnosed within 3–4 years of migration including overseas students. These are specifically identified in this report (Table 3 and Figure 4) and represent 25% of

all overseas-born TB notifications. Through jurisdictional TB control programs, NTAC is working towards increased active surveillance for TB in students, and improvement of TB awareness and engagement in this population.

There are three important groups of maritime arrivals impacting TB control in Australia. In 2010 the number of notifications in PNG nationals in Australia nearly doubled compared with 2009 (n=36, 2010 n=55), and more than .half of these were people crossing the Torres Strait Islands Treaty Zone between the Western Province of PNG and Far North Queensland. This group was a significant burden on the Queensland TB Control Program, as it represented one-fifth of Queensland TB notifications and 44% were MDR-TB. This group also represented 43% of all MDR-TB in Australia.[3] In 2010 a collaborative effort was initiated between the TB control programs in PNG, Queensland, and the Australian Government Department of Health, with funding from the then AusAid. The collaboration aims to develop the capacity for managing TB in the Western Province and transfer responsibility for clinical management of TB cases to the TB control program there. Until high level local control of TB is achieved in PNG, the potential for cases to present in Australia from the Western Province remains.

The second group of TB cases arriving by sea is asylum seekers. The number of irregular maritime arrivals (IMAs) increased in 2010. This group is recognised to be at higher risk for TB, because of both the high incidence of TB in the countries from which they originate and transit through, and the circumstances from which they are fleeing.[8] This group is not specifically reported here, because the current TB enhanced surveillance dataset does not clearly distinguish this group because case status as immigration detainees, temporary protection visa holders and permanently settled refugees is often not clearly recorded in notifications, and data on residency status is generally not reliable, because it is not verified. However, the two jurisdictions that receive all IMAs when they first arrive, Western Australia and the Northern Territory, are currently analysing this group and will report their number and characteristics separately.

The third potential group was Indonesians detained for fishing illegally in Australian waters. These represented a significant proportion of Australian TB notifications prior to 2008, but, as in 2009, were not represented significantly in 2010.[9]

Amongst other recorded risk factors for TB, predictably, a history of close contact with TB is commonly reported. This is most important in Australian-born TB cases, as it is marker of possible transmission of TB within Australia. Reported close contact in Australian-born non-Indigenous TB notifications is relatively low (28% of cases assessed for risk factors), whereas 69% of Aboriginal and Torres Strait Islander peoples notified with TB have a history of contact, indicating that transmission to close contacts remains an important cause of higher TB rates in this group. Children diagnosed with TB, by definition, must have acquired their infection recently, so are the best indicator of recent transmission. Amongst Australian-born children, 4 (24% of cases assessed for risk factors) had at least 1 parent born in a high incidence country and 8 (47%) had reported close contact with a case. As a function of all notifications, these data suggest that while transmission within Australia is likely to be uncommon, it does occur. The high proportion of children and Indigenous cases that reported close contact with TB demonstrates the potential for loss of TB control through on-going transmission within Australia and should be monitored closely.

Prior TB annual reports have identified an increasing number of Health Care Workers (HCW) diagnosed with TB. In 2010 this was again seen, with a slight increase compared with 2008 and 2009. These HCWs were nearly all born overseas (91%) and none were considered to have acquired TB from a patient contact in Australia. They therefore do not represent a failure of infection control in Australian health care facilities, but do represent an important risk group for TB that has arisen from an increasing migration of HCWs from high incidence countries to work in Australia. Recognition of the risk posed by this group has led jurisdictional TB control programs to review TB risk management policies for HCWs, and NTAC is currently writing a national guideline for management of TB risk in HCWs.

HIV testing in TB notifications is now well reported (90%), though a result is only available in 54% of notifications. HIV and TB co-infection remains rare in Australia (3.4% of those in which a result was available) and a relatively minor contributor to annual TB incidence, unlike many other parts of the world.

Surveillance benchmarks for areas embarking on a TB elimination strategy are distinct from those for high incidence countries that aim to achieve disease control. Obtaining bacteriological confirmation for at least 80% of all cases is considered a reasonable benchmark for a low prevalence setting.[10] A lower proportion than this might suggest over-diagnosis as a potential issue. The proportion of notifications confirmed by culture in 2012 remained within the range of 70%–80%, which has been the trend of the past decade.[2] The higher rate of bacteriological confirmation noted in pulmonary cases (77%) is expected. However,

the lower culture yields reported in extra-pulmonary (64%) and childhood cases (25%) is likely to reflect the more pauci-bacillary nature of disease and the difficulty in obtaining adequate samples. Additionally, in children, cases strongly suspected on clinical and radiological grounds and related to recent household exposure (where the source case's bacteriologic information is already known) are invariably started on treatment for presumptive infection. Of concern is the number of culture confirmed cases detected by bronchoscopy that were also smear positive (n=37). It is likely that most of these smear positive cases would have been detected if good attempts to collect sputum or induced sputum samples had been undertaken, thereby avoiding bronchoscopy, which in this situation places staff involved in the procedure at unnecessary risk.

In conclusion, Australia maintained effective TB control in 2010 as measured against performance indicators set in the National Strategic Plan.[11] At the time of writing, a new strategic plan for 2011–2015 has been endorsed.[12] This document does not set specific numerical notification targets, as these have been recognised to be largely dependent on factors external to Australian TB control efforts; specifically, migration. However, this strategic plan offers much wider strategic goals, with a goal and objective based work plan. In particular, the plan recommends specific activity toward targeted screening for TB in high risk groups, including latent TB infection treatment. Finally, NTAC acknowledges the continued difficulty in publishing timely notification reports and combination of these reports with Mycobacterial Reference Laboratory reports. Achieving early combined reports is also a key goal in the current strategic plan.

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Hospital-onset Gram-negative Surveillance Program annual report, 2011

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Abstract

The Australian Group on Antimicrobial Resistance performs regular period-prevalence studies to monitor changes in antimicrobial resistance in selected enteric Gram-negative pathogens. The 2011 survey focussed on hospital-onset infections, examining isolates from all specimens presumed to be causing disease. In 2011, 1,827 *Escherichia coli,* 537 *Klebsiella* species and 269 *Enterobacter* species were tested using a commercial automated method (Vitek 2, BioMérieux) and results were analysed using Clinical and Laboratory Standards Institute breakpoints from January 2012. Of the key resistances, non-susceptibilty to the third-generation cephalosporin, ceftriaxone, was found in 9.6% of *E. coli* and 9.5%–12.1% of *Klebsiella* spp. Non-susceptibility rates to ciprofloxacin were 10.6% for *E. coli,* 0.0%–8.3% for *Klebsiella* spp. and 0.0%–5.0% in *Enterobacter* spp. Resistance rates to gentamicin were 8.6%, 2.9%–10.9%, and 0.0%–15.6% for the same 3 groups respectively. Eight strains, 5 *Klebsiella* spp. and 3 *Enterobacter* spp. were shown to harbour a carbapenemase (IMP-4). *Commun Dis Intell* 2014;38 (1):E49–E53.

Keywords: antibiotic resistance; hospital onset; gram-negative; *Escherichia coli; Enterobacter; Klebsiella*

Introduction

Emerging resistance in common pathogenic members of the family Enterobacteriaceae is a world-wide phenomenon, and presents therapeutic problems for practitioners in both the community and in hospital practice. The Australian Group on Antimicrobial Resistance commenced surveillance of the key Gram-negative pathogens, *Escherichia coli* and *Klebsiella* species in 1992. Surveys have been conducted biennially until 2008 when annual surveys commenced alternating between community– and hospital-onset infections (http://www.agargroup.org/surveys). In 2004, another genus of Gram-negative pathogens in which resistance can be of clinical importance, *Enterobacter* species, was added. *E. coli* is the most common cause of community-onset urinary tract infection, while *Klebsiella* species are less common but are known to harbour important resistances. *Enterobacter* species are less common but prominent in hospital-acquired infections, and of high importance due to intrinsic resistance to first-line antimicrobials. Taken together, the 3 groups surveyed are considered to be valuable sentinels for multi-resistance and emerging resistance in enteric Gram-negative bacilli.

Resistances of particular interest include resistance to ß-lactams due to ß-lactamases, especially extended-spectrum ß-lactamases, which inactivate the third-generation cephalosporins that are normally considered reserve antimicrobials. Other resistances of interest include resistance to antibiotics commonly used in the hospital setting such as cefazolin; resistance to agents important for serious infections, such as gentamicin; and resistance to reserve agents such as ciprofloxacin and meropenem.

The objectives of the 2011 surveillance program were to:

1. determine the proportion of resistance to the main therapeutic agents in *E. coli*, *Klebsiella* species and *Enterobacter* species in a subset of Australian diagnostic laboratories;
2. examine the extent of co-resistance and multi-resistance in these species; and
3. detect emerging resistance to extended-spectrum cephalosporins and newer last-line agents such as carbapenems.

Methods

Source of isolates

Isolates were collected from patients hospitalised for more than 48 hours. Each institution collected up to 70 *E. coli,* 20 *Klebsiella* spp. and 10 *Enterobacter* spp.

Species identification

Isolates were identified by one of the following methods: Vitek®; Phoenix™ Automated Microbiology System, Microbact; ATB®; or agar replication. In addition, some *E. coli* isolates were identified using chromogenic agar plus spot indole (DMACA).

Susceptibility testing

Testing was performed by a commercial semi-automated method, Vitek® 2 (BioMérieux), which is calibrated to the ISO reference standard method

of broth microdilution. Commercially available Vitek® AST-N149 cards were utilised by all participants throughout the survey period. The most recent Clinical and Laboratory Standards Institute breakpoints from 2012[1] were employed in the analysis. *E. coli* ATCC 25922 and *E. coli* ATCC 35218 were the quality control strains for this survey. For analysis of cefazolin, breakpoints of ≤4 for susceptible and ≥8 for resistant were applied due to the minimum inhibitory concentration (MIC) range available on the Vitek card, recognising that the January 2012 breakpoint is actually susceptible ≤2 mg/L. Ertapenem MICs were performed using Etest™ strips (BioMérieux). Non-susceptibility, (which includes both intermediately resistant and resistant strains), has been included for some agents because these figures provide information about important emerging acquired resistances.

Molecular confirmation of resistances

E. coli and *Klebsiella* isolates with ceftazidime or ceftriaxone MIC >1 mg/L, or cefoxitin MIC >8 mg/L; *Enterobacter* spp. with cefepime MIC >1 mg/L; and all isolates with ertapenem MIC >0.5 mg/L or meropenem MIC >0.25 mg/L were referred to a central laboratory for molecular confirmation of resistance.

All isolates were screened for the presence of the bla_{TEM}, and bla_{SHV} genes using a real-time polymerase chain reaction (PCR) platform (LC-480) and published primers.[2,3] A multiplex real-time TaqMan PCR was used to detect CTX-M-type genes.[4] Strains were probed for plasmid-borne AmpC enzymes using the method described by Pérez-Pérez and Hanson,[5.] and subjected to molecular tests for MBL (bla_{VIM}, bla_{IMP}, and bla_{NDM}), bla_{KPC}, and $bla_{OXA\text{-}48\text{-}like}$ genes using real-time PCR.[6,7]

Results

In 2011, 2,633 isolates were examined comprising 1,827 *E. coli*, 537 *Klebsiella* spp. and 269 *Enterobacter* spp. (Table 1). The majority of isolates were from urine, while 5.6% of isolates overall were from blood cultures (comprising 4.8% of *E. coli* isolates, 7.3% of *Klebsiella* and 8.2% of *Enterobacter* species). Other sites of isolation reflect the high incidence of these species in nosocomial and pre– and post-operative surgical infections.

Major resistances and non-susceptibilities are listed in Table 2. Multi-resistance was detected in 12.6% of *E. coli* isolates, 10.6% of *Klebsiella* species, and 8.7% of *Enterobacter* species (Table 3). A more detailed breakdown of resistances and non-susceptibilities by state and territory is provided in the online report from the group (http://www.agargroup.org/surveys). By way of summary, there were no substantial differences across the states and territories in resistance patterns in contrast to what is seen with resistance patterns in *Staphylococcus aureus* and *Enterococcus* spp.

Table 1: Species tested

Group	Species	Total
E. coli	*E. coli*	1,827
Klebsiella	*K. pneumoniae*	396
	K. oxytoca	137
	K. pneumoniae subsp *ozaenae*	3
	Klebsiella not speciated	1
Total		537
Enterobacter	*E. cloacae*	180
	E. aerogenes	83
	E. asburiae	3
	E. gergoviae	2
	Enterobacter not speciated	1
Total		269

Escherichia coli

Moderately high levels of resistance to ampicillin (and therefore amoxycillin) were observed (50.5%), with lower rates for amoxycillin-clavulanate (16.1% intermediate, and 7.7% resistant) (Table 2). Non-susceptibility to third-generation cephalosporins has increased slowly compared with the 2009 survey (ceftriaxone 9.6%, ceftazidime 5.8%, compared with 7.2% and 4.2% respectively in 2009). Most of the strains with extended-spectrum ß-lactamase (ESBL) genes harboured genes of the CTX-M type (68%, 128/189). Moderate levels of resistance were detected to cefazolin (22.3%) and trimethoprim (23.4%). Ciprofloxacin non-susceptibility was found in 10.6% of *E. coli* isolates. Ciprofloxacin resistance was found in 51.1% and gentamicin resistance was found in 42.6% of ESBL-producing strains. Resistance to ticarcillin-clavulanate, cefepime, and gentamicin were below 5%. Two isolates had elevated meropenem MICs (≥0.5 mg/L) but 73 strains (4.0%) had ertapenem MICs above wild-type (>0.06 mg/L), 89% of which contained CTX-M or plasmid-borne *AmpC* genes. None harboured a known carbapenemase.

Klebsiella species

These showed slightly higher levels of resistance to cefazolin and ceftriaxone compared with *E. coli*, but lower rates of resistance or non-susceptibility to ticarcillin-clavulanate, cefazolin, ceftriaxone, ceftazidime, and gentamicin (Table 2). ESBLs were

Table 2: Non-susceptibility and resistance rates for the main species tested

Antimicrobial	Category*	*E. coli* (%)	*K. pneumoniae* (%)	*K. oxytoca* (%)	*E. cloacae* (%)	*E. aerogenes* (%)
Ampicillin	I	0.9	†	†	†	†
Ampicillin	R	50.5	†	†	†	†
Amoxycillinclavulanate	I	16.1	8.8	4.4	†	†
Amoxycillinclavulanate	R	7.7	6.1	10.2	†	†
Ticarcillin-clavulanate	R	8.0	9.1	11.7	33.9	21.7
Cefazolin	R	22.3	18.4	68.6	†	†
Cefoxitin	R	4.8	4.3	2.2	†	†
Ceftriaxone	NS	9.6	12.1	9.5	43.3	33.7
Ceftazidime	NS	5.8	9.8	3.6	40.6	28.9
Cefepime	NS	1.8	2.3	0.0	4.4	0.0
Meropenem	NS	0.1	0.5	0.0	0.6	0.0
Ertapenem	NS	0.2	1.0	0.0	16.1	4.8
Ciprofloxacin	NS	10.6	8.3	0.0	5.0	0.0
Norfloxacin	NS	10.2	4.8	0.0	4.4	0.0
Gentamicin	NS	8.6	10.9	2.9	15.6	0.0
Trimethoprim	R	23.4	18.7	4.4	27.2	2.4
Nitrofurantoin	NS	5.0	†	†	†	†

* R = resistant, I = intermediate, NS = non-susceptible (intermediate + resistant).

† Considered largely intrinsically resistant due to natural β-lactamases.

Testing for resistance to piperacillin-tazobactam was not available for this survey due to a global recall from BioMérieux.

Table 3: Multiple acquired resistances, by species

Species	Total	Number of acquired resistances												
		Non-multi-resistant					Multi-resistant							
		0	1	2	3	Cumulative %	4	5	6	7	8	9	10	Cumulative %
E. coli	1,827	828	340	278	150		68	48	55	29	26	4	1	
%		45.3	18.6	15.2	8.2	87.4	3.7	2.6	3.0	1.6	1.4	0.2	0.1	12.6
Klebsiella spp.*	537	280	158	22	20		20	12	10	11	3	1		
%		52.1	29.4	4.1	3.7	89.4	3.7	2.2	1.9	2.0	0.6	0.2		10.6
Enterobacter spp.†	269	107	56	62	18		16	6	3	1				
%		39.8	20.8	23.0	6.7	90.3	5.9	2.2	1.1	0.4				9.7

* Antibiotics included: amoxycillin-clavulanate, cefazolin, cefoxitin, ceftriaxone, ceftazidime, cefepime, gentamicin, amikacin, ciprofloxacin, nitrofurantoin, trimethoprim, meropenem.

Antibiotics excluded: ampicillin (intrinsic resistance), ticarcillin-clavulanate, tobramycin, norfloxacin, nalidixic acid, sulfamethoxazole-trimethoprim (high correlation with antibiotics in the included list).

† Antibiotics included, ceftriaxone, ceftazidime, cefepime, gentamicin, amikacin, ciprofloxacin, nitrofurantoin, trimethoprim, meropenem.

Antibiotics excluded: ampicillin, amoxycillin-clavulanate, cefazolin, and cefoxitin, (all four due to intrinsic resistance); also excluded were ticarcillin-clavulanate, tobramycin, norfloxacin, nalidixic acid, sulfamethoxazole-trimethoprim (high correlation with antibiotics in the included list).

present in 48 of 53 presumptively ESBL-positive isolates of *K. pneumoniae*, 35 of which proved to be of the CTX-M type. Five of 7 *Klebsiella* species (5 *K. pneumoniae* and 1 *K. oxytoca*) with elevated meropenem MICs (≥ 0.5 mg/L) harboured bla_{IMP-4}, while 30 additional strains had elevated ertapenem MICs (>0.06 mg/L), but none of these harboured a known carbapenemase.

Enterobacter species

Acquired resistance was common to ticarcillin-clavulanate (29.7%), ceftriaxone (40.1%), ceftazidime (36.4%) and trimethoprim (19.3%) (Table 2). Rates of resistance to cefepime, ciprofloxacin, and gentamicin were all less than 11%. Twenty-seven of 88 strains tested for ESBL based on a suspicious phenotype, harboured ESBL-encoding genes. Five strains had elevated meropenem MICs (≥ 0.5 mg/L) three of which harboured bla_{IMP-4}, while 39% of strains had ertapenem MICs above wild type (>0.125 mg/L), related to the presence of stably-derepressed chromosomal *AmpC* ß-lactamase.

Discussion

Comparing these results with those from the first hospital-onset survey in 2009, there is a small but noticeable increase in resistance or non-susceptibility rates to some reserve antibiotics. For example, rates of resistance in *E. coli* for ceftriaxone rose from 7.2% to 9.6% and for non-susceptibility to ciprofloxacin rose from 8.1% to 10.6%. Such rises were not observed in *Klebsiella* or *Enterobacter* species. Although originally thought to be primarily community-associated, the great bulk of extended-spectrum ß-lactamases detected were of the CTX-M type, suggesting that this group has become the dominant form in hospital infections as well. Plasmid-borne AmpC ß-lactamases also appear to be increasing substantially, up from 31 strains with genes detected encoding one of these enzymes in 2009, to 51 strains in 2011.

The greatest concern is the emergence of carbapenemases which affect the 'last-line' ß-lactams such as meropenem. In 2009, we detected 5 strains of *Klebsiella* with a carbapenemase, all of which were bla_{IMP-4}. In this 2011 survey, we found 8 strains, 5 *Klebsiella* spp. and 3 *Enterobacter* sp., all of which were also bla_{IMP-4}. This carbapenemase appears to have become endemic in Australia, albeit at a very low level presently. So far our surveys have not detected other carbapenemases, such as KPC-2 and NDM-1, which are known to be prevalent in other countries. However, there are published reports of the detection on these carbapenemases in Australia, all so far imported by overseas visitors or Australian returning from overseas.[10,11] Surveys such as those conducted by AGAR are critical to determining whether such unwelcome resistances might become established in Australia.

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Community-onset Gram-negative Surveillance Program annual report, 2012

John D Turnidge, Thomas Gottlieb, David H Mitchell, Geoffrey W Coombs, Denise A Daley, Jan M Bell for the Australian Group on Antimicrobial Resistance

Abstract

The Australian Group on Antimicrobial Resistance performs regular period-prevalence studies to monitor changes in antimicrobial resistance in selected enteric Gram-negative pathogens. The 2012 survey focussed on community-onset infections, examining isolates from urinary tract infections from patients presenting to outpatient clinics, emergency departments or to community practitioners. In 2012, 2,025 *Escherichia coli,* 538 *Klebsiella* species and 239 *Enterobacter* species were tested using a commercial automated method (Vitek 2, BioMérieux) and results were analysed using Clinical and Laboratory Standards Institute breakpoints from January 2012. Of the key resistances, non-susceptibility to the third-generation cephalosporin, ceftriaxone, was found in 4.2% of *E. coli* and 4.6%–6.9% of *Klebsiella* spp. Non-susceptibility rates to ciprofloxacin were 6.9% for *E. coli,* 0.0%–3.5% for *Klebsiella* spp. and 0.8%–1.9% in *Enterobacter* spp, and resistance rates to piperacillin-tazobactam were 1.7%, 0.7%–9.2%, and 8.8%–11.4% for the same 3 groups respectively. Only 1 *Enterobacter cloacae* was shown to harbour a carbapenemase (IMP-4). *Commun Dis Intell* 2014;38(1):E54–E58.

Keywords: antibiotic resistance; community onset; gram-negative; *Escherichia coli; Enterobacter; Klebsiella*

Introduction

Emerging resistance in common pathogenic members of the family Enterobacteriaceae is a world-wide phenomenon, and presents therapeutic problems for practitioners in both the community and in hospital practice. The Australian Group on Antimicrobial Resistance commenced surveillance of the key Gram-negative pathogens, *Escherichia coli* and *Klebsiella* species in 1992. Surveys have been conducted biennially until 2008 when annual surveys commenced alternating between community– and hospital-onset infections (http://www.agargroup.org/surveys). In 2004, another genus of Gram-negative pathogens in which resistance can be of clinical importance, *Enterobacter* species, was added. *E. coli* is the most common cause of community-onset urinary tract infection, while *Klebsiella* species are less common but are known to harbour important resistances. *Enterobacter* species are less common in the community, but of high importance due to intrinsic resistance to first-line antimicrobials in the community. Taken together, the 3 groups of species surveyed are considered to be valuable sentinels for multi-resistance and emerging resistance in enteric Gram-negative bacilli.

Resistances of particular interest include resistance to ß-lactams due to ß-lactamases, especially extended-spectrum ß-lactamases, which inactivate the third-generation cephalosporins that are normally considered reserve antimicrobials. Other resistances of interest include resistance to antibiotics commonly used in the community such as trimethoprim; resistance to agents important for serious infections, such as gentamicin; and resistance to reserve agents such as ciprofloxacin and meropenem.

The objectives of the 2012 surveillance program were to:

1. determine proportions of resistance to the main therapeutic agents in *Escherichia coli*, *Klebsiella* species and *Enterobacter* species in a subset of Australian diagnostic laboratories;
2. examine the extent of co-resistance and multi-resistance in these species; and
3. detect emerging resistance to newer last-line agents such as carbapenems. Isolates from the urinary tract were selected for this program.

Methods

Source of isolates

Isolates were collected from non-hospitalised patients with urinary tract infections, including those presenting to emergency departments, outpatient departments or to community practitioners. Each institution collected up to 70 *E. coli,* 20 *Klebsiella* spp. and 10 *Enterobacter* spp. isolates. Urinary tract isolates were selected because of their high frequency and high rates of exposure to antimicrobial agents in the community.

Species identification

Isolates were identified by one of the following methods: Vitek®; Phoenix™ Automated Microbiology System, Microbact; ATB®; or agar

replication. In addition, some *E. coli* isolates were identified using chromogenic agar plus spot indole (DMACA).

Susceptibility testing

Testing was performed by a commercial semi-automated method, Vitek® 2 (BioMérieux), which is calibrated to the ISO reference standard method of broth microdilution. Commercially available Vitek® AST-N246 cards were utilised by all participants throughout the survey period. The most recent Clinical and Laboratory Standards Institute breakpoints from 2013[1] were employed in the analysis. *E. coli* ATCC 25922 and *E. coli* ATCC 35218 were the quality control strains for this survey. For analysis of cefazolin, breakpoints of ≤4 for susceptible and ≥8 for resistant were applied due to the minimum inhibitory concentration (MIC) range available on the Vitek card, recognising that the January 2013 breakpoint is actually susceptible ≤2 mg/L. Non-susceptibility, (which includes both intermediately resistant and resistant strains), has been included for some agents because these figures provide information about important emerging acquired resistances.

Molecular confirmation of resistances

E. coli and *Klebsiella* isolates with ceftazidime or ceftriaxone MIC >1 mg/L, or cefoxitin MIC >8 mg/L; *Enterobacter* spp. with cefepime MIC >1 mg/L; and all isolates with meropenem MIC >0.25 mg/L were referred to a central laboratory for molecular confirmation of resistance.

All isolates were screened for the presence of the bla_{TEM}, and bla_{SHV} genes using a real-time polymerase chain reaction (PCR) platform (LC-480) and published primers.[2,3] A multiplex real-time TaqMan PCR was used to detect CTX-M-type genes.[4] Strains were probed for plasmid-borne AmpC enzymes using the method described by Pérez-Pérez and Hanson,[5] and subjected to molecular tests for MBL (bla_{VIM}, bla_{IMP}, and bla_{NDM}), bla_{KPC}, and $bla_{OXA\text{-}48\text{-}like}$ genes using real-time PCR.[6,7]

Results

In 2012, 2,802 isolates were examined, comprising 2,025 *E. coli,* 538 *Klebsiella* spp. and 239 *Enterobacter* spp. (Table 1). Major resistances and non-susceptibilities are listed in Table 2. Multi-resistance was detected in 7.6% of *E. coli* isolates, 5.1% of *Klebsiella* spp. and 5.4% of *Enterobacter* spp. (Table 3). A more detailed breakdown of resistances and non-susceptibilities by state and territory is provided in the online report from the group (http://www.agargroup.org/surveys). By way of summary, there were no substantial differences across the states and territories in resistance patterns in contrast to what is seen with resistance patterns in *Staphylococcus aureus* and *Enterococcus* spp.

Table 1: Species tested

Group	Species	Total
E. coli	*E. coli*	2,025
Klebsiella	*K. pneumoniae*	434
	K. oxytoca	101
	K. pneumoniae subsp *ozaenae*	3
Total		538
Enterobacter	*E. cloacae*	128
	E. aerogenes	107
	E. asburiae	2
	E. gergoviae	1
	Enterobacter not speciated	1
Total		239

Escherichia coli

Moderately high levels of resistance to ampicillin (and therefore amoxycillin) were observed (44.3%), with lower rates for amoxycillin-clavulanate (11.3% intermediate, 5.3% resistant) (Table 2). Non-susceptibility to third-generation cephalosporins was low but appears to be increasing slowly compared with the 2010 survey (ceftriaxone 4.2%, ceftazidime 2.2%). In line with international trends amongst community strains of *E. coli*, most of the strains with extended-spectrum ß-lactamase (ESBL) genes harboured genes of the CTX-M type (75%, 68/91). Moderate levels of resistance were detected to cefazolin (14.3%) and trimethoprim (22.7%). Ciprofloxacin non-susceptibility was found in 6.9% of *E. coli* isolates. Ciprofloxacin resistance was found in 51.8% and gentamicin resistance was found in 30.1% of ESBL-producing strains. Resistance to ticarcillin-clavulanate, piperacillin-tazobactam, cefepime, and gentamicin were below 5%. No isolates had elevated meropenem MICs.

Klebsiella species

These isolates showed slightly higher levels of resistance to cefazolin, ceftriaxone and piperacillin-tazobactam compared with *E. coli*, but lower rates of resistance to amoxycillin-clavulanate, ticarcillin-clavulanate, ciprofloxacin, gentamicin, and trimethoprim (Table 2). ESBLs were present in 17 of 21 presumptively ESBL-positive isolates of *K. pneumoniae*, 14 of which proved to be of the CTX-M type. No *Klebsiella* species had elevated meropenem MICs.

Table 2: Non-susceptibility and resistance rates for the main species tested

Antimicrobial	Category*	*E. coli* (%)	*K. pneumoniae* (%)	*K. oxytoca* (%)	*E. cloacae* (%)	*E. aerogenes* (%)
Ampicillin	I	1.9	†	†	†	†
Ampicillin	R	44.3	†	†	†	†
Amoxycillinclavulanate	I	11.3	2.8	1.0	†	†
Amoxycillinclavulanate	R	5.3	2.1	9.9	†	†
Ticarcillin-clavulanate	R	5.7	1.8	12.5	16.8	19.8
Piperacillintazobactam	R	1.7	0.7	9.2	8.8	11.4
Cefazolin	R	14.3	6.9	75.8	†	†
Cefoxitin	R	1.5	1.4	0.0	†	†
Ceftriaxone	NS	4.2	4.6	6.9	27.3	21.5
Ceftazidime	NS	2.2	3.0	0.0	19.5	18.7
Cefepime	NS	0.7	0.5	0.0	0.8	0.0
Meropenem	NS	0.0	0.0	0.0	1.6	0.0
Ciprofloxacin	NS	6.9	3.5	0.0	0.8	1.9
Norfloxacin	NS	6.8	2.3	0.0	0.0	1.9
Gentamicin	NS	4.5	3.0	0.0	5.5	0.0
Trimethoprim	R	22.7	9.9	3.0	17.2	1.9
Nitrofurantoin	NS	5.4	†	†	†	†

* R = resistant, I = intermediate, NS = non-susceptible (intermediate + resistant).

† Considered largely intrinsically resistant due to natural β-lactamases.

Table 3: Multiple acquired resistances, by species

Species	Total	Number of acquired resistances													
		Non-multi-resistant					Multi-resistant								
		0	1	2	3	Cumulative %	4	5	6	7	8	9	10	11	Cumulative %
E. coli	1,871	940	368	304	117		62	33	23	16	4	3		1	
%		50.2	19.7	16.2	6.3	92.4	3.3	1.8	1.2	0.9	0.2	0.2		0.1	7.6
Klebsiella spp.*	508	303	150	21	8		12	4	5	3	2				
%		59.6	29.5	4.1	1.6	94.9	2.4	0.8	1.0	0.6	0.4				5.1
Enterobacter spp.†	224	122	51	19	20		7	4		1					
%		54.5	22.8	85.0	8.9	94.6	3.1	1.8		0.4					5.4

* Antibiotics included: amoxycillin-clavulanate, piperacillin-tazobactam, cefazolin, cefoxitin, ceftriaxone, ceftazidime, cefepime, gentamicin, amikacin, ciprofloxacin, nitrofurantoin, trimethoprim, meropenem.

Antibiotics excluded: ampicillin (intrinsic resistance), ticarcillin-clavulanate, tobramycin, norfloxacin, nalidixic acid, sulfamethoxazole-trimethoprim (high correlation with antibiotics in the included list).

† Antibiotics included: piperacillin-tazobactam, ceftriaxone, ceftazidime, cefepime, gentamicin, amikacin, ciprofloxacin, nitrofurantoin, trimethoprim, meropenem

Antibiotics excluded: ampicillin, amoxycillin-clavulanate, cefazolin, and cefoxitin, (all four due to intrinsic resistance); also excluded were ticarcillin-clavulanate, tobramycin, norfloxacin, nalidixic acid, sulfamethoxazole-trimethoprim (high correlation with antibiotics in the included list).

Enterobacter species

Acquired resistance was common to ticarcillin-clavulanate (17.8%), piperacillin-tazobactam (9.8%), ceftriaxone (24.3%), ceftazidime (18.8%) and trimethoprim (10.0%) (Table 2). Rates of resistance to cefepime, ciprofloxacin, and gentamicin were all less than 5%. Three of 4 strains tested for ESBL based on a suspicious phenotype, harboured ESBL-encoding genes. Two strains had elevated meropenem MICs (≥ 0.5 mg/L) one of which harboured $bla_{\text{IMP-4}}$.

Discussion

The Australian Group on Antimicrobial Resistance has been tracking resistance in sentinel enteric Gram-negative bacteria since 1992. Until 2008, surveillance was segregated into hospital– versus community-onset infections. The first year of community-onset only surveillance was 2008.[8] Comparing results from that year with 2012, there has been a noticeable increase in resistance rates to some important and reserve antibiotics. For example, rates of resistance in *E. coli* for ceftriaxone rose from 2.1% to 4.2% and for non-susceptibility to ciprofloxacin rose from 4.2% to 6.9%. Intermediate percentages were observed in 2010, confirming the definite upward trend.

Overall though, there are worrying trends in the emergence of CTX-M-producing *E. coli* and *Klebsiella* species and gentamicin– and ciprofloxacin-resistant *E. coli* now presenting in or from the community. Other resistance patterns appear stable. Carbapenem resistance attributable to acquired carbapenemases are still rare in community onset infections in Australia. Compared with many other countries in our region, resistance rates in Australian Gram-negative bacteria are still relatively low.[9]

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Community-onset *Staphylococcus aureus* Surveillance Programme annual report, 2012

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Abstract

In 2012, the Australian Group on Antimicrobial Resistance (AGAR) conducted a community-onset period-prevalence survey of clinical *Staphylococcus aureus* isolated from hospital outpatients and general practice patients including nursing homes, long term care facilities and hospice patients. Day surgery and dialysis patients were excluded. Twenty-nine medical microbiology laboratories from all state and mainland territories participated. Isolates were tested by Vitek2® (AST-P612 card). Results were compared with previous AGAR community surveys. Nationally, the proportion of *S. aureus* that were methicillin-resistant *S. aureus* (MRSA) increased significantly from 11.5% in 2000 to 17.9% in 2012 (P<0.0001). Resistance to the non-ß-lactam antimicrobials varied between regions. No resistance was detected to vancomycin, teicoplanin or linezolid. Resistance in methicillin susceptible *S. aureus* was rare apart from erythromycin (12.8%) and was absent for vancomycin, teicoplanin, linezolid and daptomycin. The proportion of *S. aureus* characterised as health care-associated MRSA (HA-MRSA) was 5.1%. Three HA-MRSA clones were characterised, with 72.9% and 26.4% of HA-MRSA classified as ST22-IV [2B] (EMRSA-15) and ST239-III [3A] (Aus-2/3 EMRSA) respectively. Multi-clonal community-associated MRSA (CA-MRSA) accounted for 12.5% of all *S. aureus*. Regional variation in resistance in MRSA was primarily due to the differential distribution of the 2 major HA-MRSA clones; ST239-III [3A] (Aus-2/3 EMRSA), which is resistant to multiple non-ß-lactam antimicrobials, and ST22-IV [2B] (EMRSA-15), which is resistant to ciprofloxacin and typically erythromycin. Although the majority of CA-MRSA were non-multi-resistant, a significant expansion of Panton-Valentine leukocidin (PVL) positive CA-MRSA clones has occurred nationally. The mean age of patients (31.7 years, 95% CI 28.9–34.5) with a PVL positive CA-MRSA infection was significantly lower (P<0.0001), than the mean age of patients with a PVL negative CA-MRSA infection (55.7 years, 95% CI 50.7–60.6). This shift in the molecular epidemiology of MRSA clones in the Australian community will potentially increase the number of young Australians with skin and soft tissue infections requiring hospitalisation. *Commun Dis Intell* 2014;38(1):E59–E69.

Keywords: antimicrobial resistance surveillance; *Staphylococcus aureus;* community-onset; methicillin resistance

Introduction

Staphylococcus aureus continues to be the causative organism of a wide range of community-acquired infections ranging from relatively minor skin and soft tissue infections to serious and life threatening systemic sepsis with a high mortality.[1,2] In Australia, methicillin-resistant *S. aureus* (MRSA) was first detected in Sydney in the 1960s,[3] but really became an endemic problem in hospitals, in particular in the eastern states, with the appearance of a multi-resistant strain, (Aus-2/3 EMRSA), in the 1970s and 80s.[4,5] In Australia, community-associated MRSA (CA-MRSA) strains emerged in the 1990s, initially in Western Australia and the Northern Territory,[6–8] and subsequently in the eastern states.[9–11] These MRSA strains are generally less resistant to a range of antimicrobials and associated with skin and soft tissue infection (SSTI). Strains harbouring the genes encoding Panton-Valentine leukocidin (PVL) were first detected in Australia in the late 1990s (the South Western Pacific [SWP] or Oceania clone: ST30-IV [2B].[12] The PVL positive Queensland clone (ST93-IV [2B]) was characterised in 2000 and is now the dominant CA-MRSA in Australia.[13,14] Importation of several overseas PVL positive clones has occurred: USA300 (ST8-IV [2B]), the Bengal Bay Clone (ST772-V [5C2]), Taiwan CA-MRSA (ST59-V [5C2 and 5]) and European CA-MRSA (ST80-IV [2B]).[15] PVL is associated with recurrent furunculosis and more severe infections including osteomyelitis, septicaemia and necrotising pneumonia.

The Australian Group on Antimicrobial Resistance (AGAR) has conducted surveillance of antimicrobial resistance in *S. aureus* for over 20 years.[16] This surveillance role is very important given the ability of *S. aureus* strains to acquire new resistance and virulence determinants and to undergo rapid clonal expansion. Since the 1960s multiple waves of MRSA clones have occurred in Australia influencing the susceptibility profiles of the isolates seen in clinical practice. Results of previous AGAR surveys provide the only longitudinal record of the epidemiology of MRSA at a national level.[17–19] Given the emergence

of hyper-virulent community MRSA strains, AGAR changed its methodology in 2000 to conduct surveys of community isolates biennially. The community-based surveys performed in 2000, 2002, 2004 and 2006 have been reported previously.[20–22]

The results of the 7th community-based survey of *S. aureus* infection conducted in 2012 are reported here.

Methods

Twenty-nine laboratories from all 8 Australian states and territories participated in the 2012 *S. aureus* AGAR survey.

From 1 July to 30 November 2012 each laboratory collected up to 100 clinically significant consecutive *S. aureus* isolates from different patients. Isolates were collected from hospital outpatients. Day surgery and dialysis patients were excluded. Isolates from nursing homes, long-term care facilities and hospice patients were included. Each *S. aureus* isolate was from an individual patient and was judged to have come from a potentially infected site.

Susceptibility methodology

All isolates were tested using the Vitek2® AST-P612 card. All isolates with a penicillin minimum inhibitory concentration (MIC) of ≤0.125 mg/L were screened for the presence of ß-lactamase using nitrocefin or disc diffusion using a Penicillin 10 unit disc (CLSI) or Penicillin 1 unit disc (EUCAST). High-level mupirocin resistance was determined by disc diffusion (200 ug). CLSI breakpoints were utilised for all antimicrobials[23] except fusidic acid (http://www.eucast.org/clinical_breakpoints/). Isolates with an MIC in the intermediate resistance category have been called resistant in this report.

Epidemiological typing of methicillin-resistant *Staphylococcus aureus*

Of the 510 MRSA identified, 499 (97.8%) were referred to the Australian Collaborating Centre for Enterococcus and Staphylococcus Species (ACCESS) Typing and Research for epidemiological typing.

Electrophoresis of chromosomal DNA using a contour-clamped homogeneous electric field DRIII System (Bio-Rad Laboratories Pty Ltd) was performed as previously described on all MRSA isolates.[24] Multilocus sequence typing (MLST) and SCC*mec* typing was performed as previously described on selected MRSA isolates.[25–27]

PCR for the detection of PVL determinants was performed as previously described on all MRSA isolates.[28]

Methicillin-resistant *Staphylococcus aureus* nomenclature

MRSA clones were defined by the combination of the multilocus sequence type (ST) and the SCC*mec* type.[29] Clones are reported with their ST and SCC*mec* type followed by their colloquial name in parenthesis; e.g. ST22-IV [2B] (EMRSA-15). SCC*mec* nomenclature is used as proposed by the International Working Group on the Classification of Staphylococcal Cassette Chromosome Elements.[30] Briefly, the structural type is indicated by a Roman numeral, with a lowercase letter indicating the subtype, and the *ccr* complex and the *mec* complex are indicated by an Arabic numeral and an uppercase letter respectively in parenthesis. Where there is an extra *ccr* element, this is indicated by '&' and an Arabic numeral designating the *ccr* type. When there is an extra *ccr* element present whose precise location is unknown it is indicated by an '&' and *ccr* number outside the parentheses. Clones were classified into two groups on the basis of previously published evidence: those implicated in healthcare-associated infection (HA-MRSA) and those implicated in CA-MRSA.

Clones that diverged at no more than one of the 7 MLST loci were considered to belong to the same clonal complex (CC). Double locus variants were included in the same CC if the linking single locus variant was present in the MLST database (http://www.mlst.net/).

Statistical analysis

The difference between proportions was tested using a Chi-square test and Fisher's exact test (GraphPad® Prism Software). Relative risk and 95% confidence intervals (CI) were calculated using VassarStats (http://vassarstats.net).

Results

The survey included 2,844 isolates (Table 1) with the majority (1,792, 63.0%) being contributed by New South Wales, Queensland and Victoria.

SSTI specimens contributed the majority of isolates (2,575, 90.5%) followed by respiratory specimens (106, 3.7%) and bacteraemia (89, 3.1%). There were significantly (P<0.0001) more isolates causing non-invasive (2,740, 96.3%) than invasive (104, 3.7%) infections (Table 2).

Table 1: *Staphylococcus aureus* isolates, Australia, 2012, by state or territory

State or territory	Number of institutions	Total	%
ACT	1	100	3.5
NSW	7	693	24.4
NT	1	100	3.5
Qld	6	599	21.1
SA	3	296	10.4
Tas.	2	159	5.6
Vic.	5	500	17.6
WA	4	397	14.0
Total	29	2,844	100.0

The proportion of *S. aureus* that were MRSA was 17.9% (95% CI 16.6–19.4%) nationally (Table 3), ranging from 4.0% in the Australian Capital Territory to 25.5% in New South Wales.

Table 2: Source of *Staphylococcus aureus* isolates, Australia, 2012

Specimen source	Number	%	95% CI
Skin and soft tissue	2,575	90.5	89.4–91.6
Respiratory	106	3.7	3.1–4.5
Blood	89	3.1	2.6–3.8
Urine	58	2.0	1.6–2.6
Sterile body cavity	16	0.6	0.4–0.9
Total	2,844		
Invasive*	104	3.7	3.0–4.4
Non-invasive	2,740	96.3	95.6–97.0

* Blood or sterile body cavity

The proportion of invasive isolates (blood/sterile body cavity sites) that were MRSA was 21.2% (95% CI 14.4–30.0%) and was similar (P=0.4037) to the proportion of non-invasive isolates at 17.8% (95% CI 16.4–19.3%) (Table 3). The proportion of MRSA was highest in blood at 21.3% (95% CI 14.1–31.0%) (Table 4).

There were significant differences (P<0.0001) in the proportion of MRSA seen in different patient groups with patients from long term care facilities (46.7%, 95% CI 24.8–70.0%), patients attending emergency departments (20.9%, 95% CI 18.9–23.1%) and hospital outpatients (17.0%, 95% CI 14.2–20.1%) having high rates of MRSA. In general practice patients the proportion of *S. aureus* that were MRSA was 12.7% (95% CI 10.5–15.3%).

Resistance in MRSA to non-ß-lactam antimicrobials (with the exception of rifampicin, high-level mupirocin and fusidic acid) varied between regions (Table 5). Two isolates were non-susceptible to daptomycin. No resistance was detected to vancomycin, teicoplanin or linezolid. There were differences in the proportion of isolates resistant to non-ß-lactam antimicrobials in MRSA associated with

Table 4: Proportion of *Staphylococcus aureus* that were methicillin resistant, Australia, 2012, by specimen type

Source of infection	n/N	%	95% CI
Blood	19/89	21.3	14.1–31.0
Sterile body cavity	3/16	18.8	6.6–43.1
Skin and soft tissue	462/2,575	17.9	16.5–19.5
Urine	10/58	17.2	9.6–28.9
Respiratory	16/106	15.1	9.5–23.1
Total	510/2,844	17.9	16.6–19.4

Table 3: Proportion of *Staphylococcus aureus* that were methicillin resistant, Australia, 2012, by state or territory and source

State or territory	All isolates			Invasive isolates*			Non-invasive isolates		
	n/N	%	95% CI	n/N	%	95% CI	n/N	%	95% CI
ACT	4/100	4.0	1.2–10.2	0/0	0.0		4/100	4.0	1.2–10.2
NSW	177/693	25.5	22.4–28.9	13/33	39.4	24.7–56.4	164/660	24.8	21.7–28.3
NT	24/100	24.0	16.6–33.3	1/2	50.0	9.5–90.6	23/98	23.5	16.1–32.8
Qld	103/599	17.2	14.4–20.4	2/17	11.8	2.0–35.6	101/582	17.4	14.5–20.7
SA	43/296	14.5	10.9–19.0	1/5	20.0	2.0–64.0	42/291	14.4	10.8–19.0
Tas.	9/159	5.7	2.9–10.6	0/11	0.0	0–30.0	9/148	6.1	3.1–11.3
Vic.	87/500	17.4	14.3–21.0	3/23	13.0	3.7–33.0	84/477	17.6	14.4–21.3
WA	63/397	15.9	12.6–19.8	2/13	15.4	3.1–43.5	61/384	15.9	12.6–19.0
Aus.	510/2,844	17.9	16.6–19.4	22/104	21.2	14.4–30.0	488/2,740	17.8	16.4–19.3

* Blood/sterile body cavity

Table 5: Number and proportion of methicillin-resistant *Staphylococcus aureus* resistant to the non-ß-lactam antimicrobials, Australia, 2012, by state or territory

Drug	ACT (n=4)		NSW (n=177)		NT (n=24)		Qld (n=103)		SA (n=43)		Tas. (n=9)		Vic. (n=87)		WA (n=63)		Aus. (n=510)		Differences across regions	
	n	%	n	%	n	%	n	%	N	%	n	%	n	%	n	%	n	%	X^2	*P*
Erythromycin	3	75.0	77	43.5	7	29.2	22	21.4	14	30.2	4	44.4	51	58.6	23	36.5	200	39.2	31.05	<0.0001
Clindamycin*	0	0.0	28	15.8	3	12.5	10	9.7	5	11.6	0	0.0	20	23.0	2	3.2	68	13.3	16.89	0.0182
Tetracycline	0	0.0	28	15.8	5	20.8	9	8.7	5	11.6	0	0.0	26	29.9	1	1.6	74	14.5	31.34	<0.0001
Co-trimoxazole	0	0.0	20	11.3	3	12.5	4	3.9	3	7.0	0	0.0	19	21.8	3	4.8	52	10.2	21.73	0.0028
Ciprofloxacin	1	25.0	91	51.4	4	16.7	20	19.4	8	18.6	4	44.4	51	58.6	13	20.6	191	37.5	64.53	<0.0001
Gentamicin	0	0.0	22	12.4	5	20.8	3	2.9	2	4.7	0	0.0	14	16.1	2	3.2	48	9.4	20.59	0.0044
Fusidic acid	0	0.0	8	4.5	0	0.0	7	6.8	2	4.7	0	0.0	7	8.0	2	3.2	26	5.1	4.786	0.6861
Mupirocin†	0	0.0	4	2.3	0	0.0	4	3.9	0	0.0	0	0.0	0	0.0	0	0.0	8	1.6	7.788	0.3517
Rifampicin	0	0.0	2	1.1	0	0.0	1	1.0	0	0.0	0	0.0	2	2.3	1	1.6	6	1.2	2.027	0.9583

* Constitutive resistance.

† High-level resistance.

Table 6: Number and proportion of methicillin sensitive *Staphylococcus aureus* resistant to the non-ß-lactam antimicrobials, Australia, 2012, by state or territory

Drug	ACT (n=96)		NSW (n=516)		NT (n=76)		Qld (n=496)		SA (n=253)		Tas. (n=150)		Vic. (n=413)		WA (n=334)		Aus. (n=2,334)		Differences across regions	
	n	%	n	%	n	%	n	%	N	%	n	%	n	%	n	%	n	%	X^2	*P*
Penicillin	81	84.4	451	87.4	69	90.8	436	87.9	213	84.2	125	83.3	363	87.9	275	82.3	2,013	86.2	10.56	0.1589
Erythromycin	14	14.6	64	12.4	12	15.8	61	12.3	39	15.4	18	12.0	59	14.3	32	9.6	299	12.8	6.616	0.47
Clindamycin*	1	1.0	12	2.3	0	0	8	1.6	5	2.0	1	0.7	7	1.7	4	1.2	38	1.6	4.487	0.7223
Tetracycline	5	5.2	14	2.7	0	0.0	8	1.6	8	3.2	5	3.3	17	4.1	16	4.8	73	3.1	12.28	0.0918
Co-trimoxazole	5	5.2	21	4.1	0	0.0	5	1.0	12	4.7	3	2.0	24	5.8	7	2.1	87	3.7	24.94	0.0008
Ciprofloxacin	2	2.1	11	2.1	1	1.3	13	2.6	8	3.2	8	5.3	21	5.1	9	2.7	65	2.8	11.12	0.1337
Gentamicin	1	1.0	3	0.6	0	0.0	3	0.6	3	1.2	0	0.0	3	0.7	0	0.0	13	0.6	5.590	0.5884
Fusidic acid	7	7.3	33	6.4	2	2.6	56	11.3	3	1.2	10	6.7	13	3.1	11	3.3	135	5.8	48.84	<0.0001
Mupirocin†	2	2.1	8	1.6	1	1.3	47	9.5	2	0.8	0	0.0	4	1.0	4	1.2	68	2.9	97.36	<0.0001
Rifampicin	0	0.0	3	0.6	0	0.0	2	0.4	0	0.0	0	0.0	2	0.5	0	0.0	7	0.3	4.748	0.6907

* Constitutive resistance.

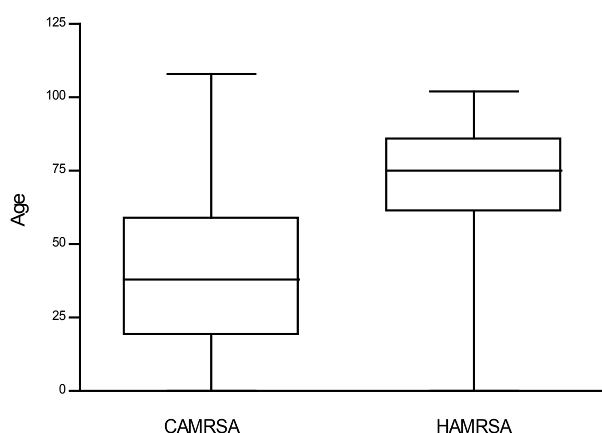
† High-level resistance.

various patient types. MRSA resistance for many antimicrobials was high in hospital outpatients, emergency and long-term care, which is consistent with a higher proportion of these having been acquired in healthcare-related settings.

Susceptibility testing of methicillin sensitive *S. aureus* (MSSA) (Table 6) show resistance to non-ß-lactam antimicrobials remains uncommon except for erythromycin where overall resistance was 12.8% (95% CI 11.5–14.2%). All isolates were susceptible to vancomycin, teicoplanin, linezolid and daptomycin. Resistance to penicillin was high and in similar proportions ranging from 82.3% to 90.8% across all regions.

Based on molecular typing, of the 499 MRSA referred to ACCESS Typing and Research, 28.9% (144) and 71.1% (355) were classified as HA-MRSA and CA-MRSA strains respectively. The mean age of patients with a CA-MRSA infection (40.6 years, 95% CI 37.8–43.4) was significantly lower ($P<0.0001$), than the mean age of patients with a HA-MRSA infection (69.8 years 95% CI 66.2–73.4) (Figure 1).

Figure 1: Box plot of age of patients infected with community-associated methicillin-resistant *Staphylococcus aureus* and healthcare-associated methicillin-resistant *Staphylococcus aureus*, Australia, 2012



Community-associated methicillin-resistant *Staphylococcus aureus* Mean age 40.6 years (95% CI: 37.8–43.4).

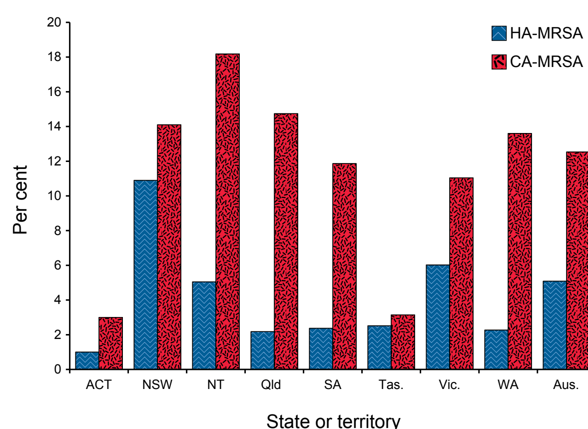
Healthcare-associated methicillin-resistant *Staphylococcus aureus* Mean age 69.8 years (95% CI: 66.2–73.4).

Throughout Australia, the percentage of *S. aureus* characterised as HA-MRSA was 5.1%, ranging from 1.0% in the Australian Capital Territory to 10.8% in New South Wales (Figure 1). Three HA-MRSA clones were identified: ST22-IV [2B] (EMRSA-15) (72.9% of HA-MRSA), ST239-III [3A] (Aus-2/3 EMRSA) (26.4%),and 1 isolate of ST5-II [2A] (New York Japan MRSA/USA100).

ST22-IV [2B] (EMRSA-15) has become the predominant HA-MRSA clone in the Australian community accounting for 21.0% of MRSA ranging from 0% in the Northern Territory to 44.4% in Tasmania (Table 7). Typically PVL negative, 99% and 61% of ST22-IV [2B] (EMRSA-15) isolates were resistant to ciprofloxacin and erythromycin respectively.

ST239-III [3A] (Aus-2/3 EMRSA) accounted for 7.6% of MRSA ranging from 0% in the Australian Capital Territory and Tasmania to 21.7% in the Northern Territory (Table 7). PVL negative ST239-III [3A] (Aus-2/3 EMRSA was typically resistant to tetracycline (100%), erythromycin (100%), gentamicin (97%),ciprofloxacin (92%), and cotrimoxazole (92%).

Figure 2: Percentage of *Staphylococcus aureus* characterised as healthcare-associated methicillin-resistant *Staphylococcus aureus* and community-associated methicillin-resistant *Staphylococcus aureus* strains, Australia, 2012, by state or territory



Throughout Australia, the percentage of *S. aureus* characterised as CA-MRSA was 12.5%, ranging from 3.0% in the Australian Capital Territory to 18.0% in the Northern Territory (Figure 2). Thirty-two CA-MRSA clones were identified by pulsed-field gel electrophoresis, corresponding to 25 MLST/SCC*mec* clones (Table 8). Overall, 82.5% of CA-MRSA were classified into 6 clones: ST93-IV [2B] (Qld CA-MRSA) (36.3% of CA-MRSA);ST30-IV [2B] (SWP MRSA) (16.9%); ST1-IV [2B] (WA1) (13.5%); ST45-V [5C2&5] (WA84) (5.9%); ST78-IV [2B] (WA2) (5.1%); and ST5-IV [2B] (WA3) (4.8%).

Table 7: Proportion of methicillin-resistant *Staphylococcus aureus* characterised as ST22-IV [2B] (EMRSA-15) and ST239-III [3A] (Aus-2/3 EMRSA), Australia, 2012, by state or territory

	ACT	`NSW	NT	Qld	SA	Tas.	Vic.	WA	Aus.
ST22-IV [2B]	25.0	34.3	0.0	9.9	11.9	44.4	21.2	12.7	21.0
ST239-III [3A]	0.0	8.7	21.7	3.0	4.8	0.0	14.1	1.6	7.6

Table 8: Proportion of community-associated methicillin-resistant *Staphylococcus aureus*, Australia, 2012, by clone and Panton Valentine leukocidin carriage

Clone	Clonal complex	Alternative name	n (%)	PVL pos (%)
ST93-IV [2B]	Singleton	Queensland MRSA	129 (36.3)	127 (98.4)
ST30-IV [2B]	30	SWP MRSA	60 (16.9)	56 (93.3)
ST1-IV [2B]	1	WA1	48 (13.5)	3 (6.3)
ST45-V [5C2&5]	45	WA84 (Vic CA-MRSA)	21 (5.9)	0
ST78-IV [2B]	88	WA2	18 (5.1)	1 (5.6)
ST5-IV [2B]	5	WA3	17 (4.8)	5 (29.4)
ST73-IV [2B]	5	WA65	10 (2.8)	0
ST8-IV [2B]	8	USA300	10 (2.8)	9 (90.0)
ST952-V [5C2&5]	59	Taiwan A MRSA	5 (1.4)	5 (100)
ST59-V [5C2&5]	59	Taiwan MRSA	5 (1.4)	5 (100)
ST5-IV [2B]	5	WA121	4 (1.1)	4 (100)
ST6-IV [2B]	5	WA51	3 (0.8)	3 (100)
ST8-IV [2B]	8	WA5	3 (0.8)	0
ST953-IV [2B]	97	WA54	3 (0.8)	0
ST772-V [5C2]	1	Bengal Bay	2 (0.6)	2 (100)
ST1-V [5C2]	1		1 (0.3)	0
ST188-IV [2B]	1	WA38	1 (0.3)	0
ST5-IV [2B]	5	WA96	1 (0.3)	0
ST5-IV [2B]	5	WA71	1 (0.3)	1 (100)
ST5-V [5C2]	5	WA109	1 (0.3)	0
ST5-V [5C2]	5		1 (0.3)	0
ST835-IV [2B]	5	WA48	1 (0.3)	0
ST2471-V [5C2]	8	WA120	1 (0.3)	0
ST12-novel	12	WA59	1 (0.3)	0
ST30-V [5C2]	30	WA124	1 (0.3)	1 (100)
ST45-IV [2B]	45	WA75	1 (0.3)	0
ST59-IV [2B]	59	WA15	1 (0.3)	0
ST59-IV [2B]	59	WA55	1 (0.3)	1 (100)
ST72-IV [2B]	72	Korean Clone	1 (0.3)	0
ST577-IV [2B]	121	WA22	1 (0.3)	0
ST883-IV [2B]	Singleton	WA47	1 (0.3)	0
ST1303-IV [2B]	U	WA76	1 (0.3)	0
Total			355	223 (62.8)

PVL Panton Valentine leukocidin.

Percentage figures in parenthesis relate to CA-MRSA isolates.

ST93-IV [2B] (Qld CA-MRSA) accounted for 25.9% of MRSA ranging from 12.9% in Victoria to 47.8% in the Northern Territory (Table 9). PVL positive ST93-IV[2B] (Qld CA-MRSA) were typically resistant to the ß-lactam antimicrobials only (110/129) or additionally to erythromycin (17/129).

ST30-IV [2B] (SWP MRSA) accounted for 12.0% of MRSA ranging from 0% in the Australian Capital Territory to 25.7% in Queensland (Table 9). Overall 90% of PVL positive ST30-IV [2B] were resistant to the ß-lactam antimicrobials only.

ST1-IV [2B] (WA1) accounted for 9.6% of MRSA ranging from 0% in Tasmania and the Australian Capital Territory to 25.4% in Western Australia (Table 9). Typically PVL negative, 95.8% of isolates were non-multi-resistant (resistant to less than 3 ß-lactam antimicrobials).

The remaining 3 major CA-MRSA clones, ST45-V [5C2&5] (WA84), ST78-IV [2B] (WA2) and ST5-IV [2B] (WA3) accounted for 4.2%, 3.6% and 3.4% of MRSA respectively.

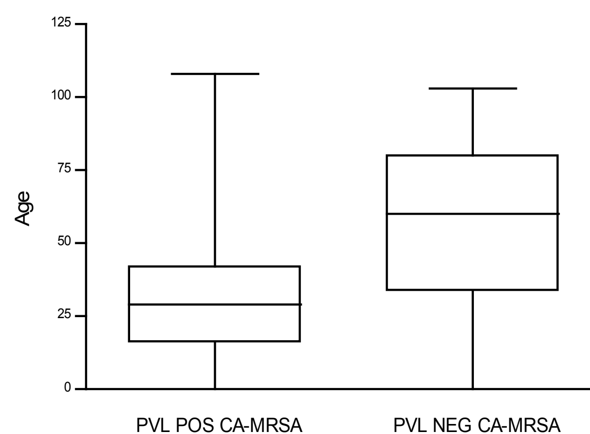
Overall, 94.4% of CA-MRSA were non-multi-resistant, with 61.4% of isolates resistant to ß-lactam antimicrobials only. However, 20 isolates (5.6% of CA-MRSA) were multi-resistant including 2 PVL positive ST772-V [5C2] (Bengal Bay MRSA) isolates, which, in addition to ß-lactam antimicrobials, were resistant to gentamicin, erythromycin, ciprofloxacin, and cotrimoxazole. Two CA-MRSA, ST188-IV [2B] (WA38) and ST8-IV [2B] (USA300), were resistant to 5 non-ß-lactam antimicrobials: gentamicin, erythromycin, ciprofloxacin, cotrimoxazole and tetracycline; and gentamicin, erythromycin, ciprofloxacin, mupirocin and tetracycline respectively.

PVL determinants were detected in 45.5% of MRSA:223 (62.8%) CA-MRSA (Table 8) and 4 ST22-IV [2B] (EMRSA-15) isolates. In addition to ST93-IV [2B] (Qld CA-MRSA) and ST30-IV [2B] (SWP MRSA), PVL-positive CA-MRSA clones included the international clones ST8-IV [2B] (USA300), ST59-V [5C2&5] (Taiwan MRSA) and ST772-V [5C2] (Bengal Bay MRSA). The mean age of patients (31.7 years: 95% CI 28.9–34.5) with a PVL positive CA-MRSA infection was significantly lower (P<0.0001) than the mean age of patients with a PVL negative CA-MRSA infection (55.7 years, 95% CI 50.7–60.6) (Figure 3).

Discussion

This survey demonstrates MRSA has become a significant burden in the Australian community. Over the 7 biennial AGAR community surveys (2000 to 2012), the percentage of *S. aureus* identified as MRSA has increased significantly (P<0.0001) by 6 percentage points over the 12-year period (11.5% in 2000 to 17.9% in 2012). Molecular typ-

Figure 3: Box plot of age of patients infected with Panton Valentine leukocidin positive and Panton Valentine leukocidin negative community-associated methicillin-resistant *Staphylococcus aureus*, Australia, 2012



Panton Valentine leukocidin positive community-associated methicillin-resistant *Staphylococcus aureus*: Mean age 31.7 years (95% CI: 28.9–34.5)

Panton Valentine leukocidin negative community-associated methicillin-resistant *Staphylococcus aureus*: Mean age 55.7 years (95% CI: 50.7–60.6)

Table 9: Proportion of methicillin-resistant *Staphylococcus aureus* characterised as community-associated methicillin-resistant *Staphylococcus aureus*, Australia, 2012, by state or territory

	ACT	NSW	NT	Qld	SA	Tas.	Vic.	WA	Aus.
ST93-IV [2B] (Qld)	25.0	21.5	47.8	37.6	33.3	22.2	12.9	23.8	25.9
ST30-IV [2B] (SWP)	0.0	8.7	13.0	25.7	9.5	22.2	7.1	6.3	12.0
ST1-IV [2B] (WA1)	0.0	6.4	4.3	8.9	14.3	0.0	5.9	25.4	9.6
ST45-V [5C2&5] (WA84)	0.0	2.9	0.0	2.0	2.4	0.0	15.3	0.0	4.2
ST78-IV [2B] (WA2)	25.0	0.6	0.0	1.0	7.1	11.1	2.4	14.3	3.6
ST5-IV [2B] (WA3)	25.0	1.7	0.0	5.0	2.4	0.0	4.6	4.8	3.4
Other	0.0	25.8	16.7	8.0	17.1	0.0	25.5	12.9	12.2

ing has shown this increase in community-onset MRSA has primarily been due to the emergence and expansion of non-multi-resistant clones.

In the 2012 study, resistance in MRSA to erythromycin, ciprofloxacin, tetracycline, gentamicin, clindamycin and cotrimoxazole significantly varied across regions. These differences can be explained by the different MRSA clones in circulation in each region; for example Aus-2/3 EMRSA (ST239-III), which is reliably resistant to gentamicin, erythromycin, tetracycline, ciprofloxacin and cotrimoxazole are commonly found in New South Wales, the Northern Territory and Victoria.

There were significant differences in the proportion of resistance to non-ß-lactam antimicrobials in MRSA associated with various patient types with gentamicin, tetracycline, ciprofloxacin, clindamycin, cotrimoxazole and fusidic acid resistance higher in hospital outpatients that other patient types. This is consistent with their having a higher proportion of healthcare-related acquisition.

In the 2012 study, apart from erythromycin, resistance to the non-ß-lactam antimicrobials amongst the MSSA was uncommon. Over the 7 AGAR surveys, no trends in resistance, increase or decrease, were evident for erythromycin, tetracycline, gentamicin or rifampicin. Nationally, small but significant increases were seen for clindamycin, ciprofloxacin, fusidic acid, high-level mupirocin and cotrimoxazole.

The mean age of patients with infections due to CA-MRSA strains (41 years; median 38 years) was found to be significantly lower (P<0.0001) than the mean age of patients with infections due to HA-MRSA strains (70 years; median 75 years). Although the percentage of *S. aureus* characterised as HA-MRSA in this survey (5.1%) was lower when compared with the 2010 survey (5.9%), ST22-IV [2B] (EMRSA-15) remains a major HA-MRSA clone in most Australian communities surveyed, accounting for 21.0% of all community-onset MRSA infections. Of continuing concern has been the rapid emergence of this clone in the community in Victoria (0% in 2002 to 21.2% in 2012), and New South Wales (18.0% in 2000 to 34.3% in 2012). In 2012, CA-MRSA accounted for 71.1% of MRSA and 12.5% of all *S. aureus*. Since 2000, the percentage of *S. aureus* characterised as CA-MRSA has more than doubled (5.3% in 2000). As in previous surveys although CA-MRSA was multi-clonal (32 clones,) 82.5% of strains could be characterised into 6 clones. ST93-IV [2B] (Qld CA-MRSA), a PVL-positive clone, remains the most frequently isolated CA-MRSA clone in the Australian community accounting for 36.3% of all CA-MRSA and 25.9% of all MRSA infections. Overall, 62.8% of CA-MRSA were PVL positive, a 21% increase when compared with the 2006 survey. The mean age of patients with PVL positive CA-MRSA infections (32 years; median 29 years) was significantly lower (P<0.0001) than the mean age of patients with PVL negative CA-MRSA infections (56 years; median 57 years). However, the increase in PVL-positive MRSA is not only due to the expansion of the ST93-IV [2B] clone but also due to ST30-IV [2B] (SWP MRSA) and due to the introduction of several international CA-MRSA clones including ST8-IV [2B] (USA300) ST59-V [5C2&5] (Taiwan CA-MRSA) and the hypervirulent multi-resistant ST772-V [5C2] (Bengal Bay). Four ST22-IV [2B] (EMRSA-15) isolates carrying the PVL determinant were also identified. Acquisition of the PVL determinant in this clone, which has been demonstrated to have enhanced transmission in the Australian community, continues to be a major public health concern.

In summary, resistance in MSSA remains uncommon with the exception of erythromycin and penicillin. Resistance in MRSA appears dynamic due to the success or decline of MRSA clones circulating in Australia. The national rate continues to rise in strains causing infections in people in the community. Since the initial AGAR community-onset *S. aureus* survey in 2000 there has been a significant increase in the percentage of patients with community onset MRSA infections in most regions of Australia, such that in 2012 one in 6 patients with a staphylococcal infection have MRSA and one in eight are infected with a CA-MRSA clone. This makes the empiric choice for the correct antibiotic therapy of community *S. aureus* infections increasingly difficult. Of further concern is that this increase in MRSA has primarily been due to the expansion of the PVL positive clones such as ST93-IV [2B] (Qld CA-MRSA) and ST30-IV [2B] (SWP CA-MRSA). This shift in the molecular epidemiology of MRSA clones in the Australian community will potentially increase the number of SSTIs in young Australians. As SSTIs caused by PVL-positive *S. aureus* frequently results in hospitalisation the emergence of these strains in the community as well as the detection of PVL-positive HA-MRSA (EMRSA-15) is a major health concern.

A full detailed report on this study may be found on the AGAR web site: (http://www.antimicrobial-resistance.com/) under AMR surveillance.

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Quarterly report

OzFoodNet enhanced foodborne disease surveillance, 1 January to 31 March 2013

The OzFoodNet Working Group

Introduction

The Australian Government Department of Health established the OzFoodNet network in 2000 to collaborate nationally to investigate foodborne disease. In each Australian state and territory, OzFoodNet epidemiologists investigate outbreaks of enteric infection. OzFoodNet conducts studies on the burden of illness and coordinates national investigations into outbreaks of foodborne disease. This quarterly report documents investigations of outbreaks of gastrointestinal illness and clusters of disease potentially related to food, which occurred in Australia between 1 January and 31 March 2013.

Data were received from OzFoodNet epidemiologists in all Australian states and territories. The data in this report are provisional and subject to change, as the results of outbreak investigations can take some months to finalise.

During the 1st quarter of 2013, OzFoodNet sites reported 522 outbreaks of enteric illness, including those transmitted by contaminated food. Outbreaks of gastroenteritis are often not reported to health agencies or the reports may be delayed, meaning that these figures under-represent the true burden of enteric disease outbreaks. In total, these outbreaks affected 8,378 people, of whom 221 were hospitalised. There were 20 deaths reported during these outbreaks. The majority of outbreaks (74%, n=385) were due to person-to-person transmission (Table 1), with 50% (194/385) of these occurring in aged care facilities.

Foodborne and suspected foodborne disease outbreaks

There were 34 outbreaks during this quarter where consumption of contaminated food was suspected or confirmed as being the primary mode of transmission (Table 2). These outbreaks affected 347 people and resulted in 36 hospitalisations. There were no deaths reported during these outbreaks. This compares with 37 outbreaks during the 4th quarter of 2012[1] and a 5-year mean of 39 outbreaks for the 1st quarter between 2008 and 2012.

Table 1: Outbreaks and clusters of gastrointestinal illness reported by OzFoodNet, Australia, 1 January to 31 March 2013, by mode of transmission

Transmission mode	Number of outbreaks and clusters	Per cent of total
Foodborne and suspected foodborne	34	6.5
Waterborne and suspected waterborne	17	3.3
Person-to-person	385	73.8
Animal-to-person	1	<1
Unknown (*Salmonella* cluster)	19	3.6
Unknown (*Listeria* cluster)	1	<1
Unknown (other pathogen cluster)	3	0.6
Unknown	62	11.9
Total	522	100.0*

* Percentages do not add up due to rounding.

A limitation of the outbreak data provided by OzFoodNet sites for quarterly reports is the potential for variation in the categorisation of the features of outbreaks depending on circumstances and investigator interpretation. Changes in the number of foodborne outbreaks should be interpreted with caution due to the small number each quarter.

Salmonella was the aetiological agent for 17 (50%) outbreaks this quarter, with *Salmonella* Typhimurium being the most common serotype (n=13). Of the remaining outbreaks, 4 (12%) were due to norovirus and 1 (3%) each due to *Campylobacter jejuni*, ciguatera fish poisoning, *Shigella sonneii* biotype a, *Staphylococcus aureus*, and a suspected bacterial toxin. In 8 (24%) outbreaks, the aetiological agent was unknown.

There were 16 outbreaks (47% of foodborne or suspected foodborne outbreaks) reported in this quarter associated with food prepared in restaurants (Table 3).

Table 2: Outbreaks of foodborne or suspected foodborne disease reported by OzFoodNet sites, 1 January to 31 March 2013 (n=34)

State or territory	Month*	Setting prepared	Agent responsible	Number affected	Hospitalised	Evidence	Responsible vehicles
ACT	Jan	Private residence	*Salmonella* Typhimurium PT 44 / MLVA) profile 03-11-07-11-523	10	0	D	Unknown
ACT	Feb	Private residence	Suspected bacterial toxin	6	0	D	Assorted chicken, beef and bean dishes with rice
NSW	Jan	Private residence	*S.* Typhimurium MLVA profile 03-27-08-21-496	8	2	D	Unknown
NSW	Jan	Restaurant	*S.* Cerro	3	1	D	Unknown
NSW	Jan	Restaurant	*S.* Typhimurium	5	0	D	Unknown
NSW	Jan	Restaurant	Norovirus	3	0	D	Salad
NSW	Jan	Takeaway	*S.* Typhimurium	3	3	D	Unknown
NSW	Jan	Takeaway	Unknown	3	0	D	Chicken burger
NSW	Feb	Private residence	*S.* Birkenhead	12	3	D	Unknown
NSW	Feb	Restaurant	*S.* Typhimurium MLVA profile 03-09-07/08-14-523	7	3	M	Fried ice cream with raw egg
NSW	Feb	Restaurant	Unknown	4	0	D	Beef taco
NSW	Feb	Takeaway	Unknown	2	0	D	Unknown
NSW	Mar	Private residence	*S.* Typhimurium MLVA profile 03-17-09-12-523	4	4	D	Raw egg smoothies
NT	Feb	Private residence	*S.* Typhimurium PT 9	4	2	D	Caesar salad dressing containing raw egg
NT	Mar	Private residence	*Shigella sonnei* biotype a	5	1	D	Curried meat (unknown type of meat)
Qld	Jan	Restaurant	*S.* Typhimurium MLVA profile 03-25-16-11-524	3	1	D	Duck liver paté
Qld	Jan	Restaurant	*Staphylococcus aureus*	8	0	M	Chicken sushi
Qld	Feb	National franchised fast food	Unknown	3	0	D	Pizza
Qld	Mar	Primary produce	Ciguatera fish poisoning	4	Unknown	D	Red coral trout
SA	Mar	Restaurant	Norovirus	14	1	D	Unknown
SA	Mar	Restaurant	*Salmonella* Typhimurium PT 9 MLVA profile of 03-15-06-11-550	9	1	D	Eggs
Tas.	Jan	Restaurant	*S.* Mississippi	36	2	D	Suspected salad
Vic.	Jan	Commercial caterer	Unknown	32	1	A	Sandwiches
Vic.	Jan	Correctional facility	*S.* Typhimurium PT 135	3	1	D	Raw egg drink
Vic.	Jan	Private residence	*S.* Typhimurium PT 135a	10	1	D	Tiramisu containing raw eggs
Vic.	Jan	Restaurant	Norovirus	6	0	D	Infectious food handler
Vic.	Jan	Restaurant	Norovirus	7	0	D	Salad

Table 2 *continued*: Outbreaks of foodborne or suspected foodborne disease reported by OzFoodNet sites, 1 January to 31 March 2013 (n=34)

State or territory	Month*	Setting prepared	Agent responsible	Number affected	Hospitalised	Evidence	Responsible vehicles
Vic.	Feb	Aged care facility	Unknown	9	0	D	Unknown
Vic.	Feb	Bakery	*S.* Infantis	21	5	D	Pork rolls
Vic.	Feb	Restaurant	*Campylobacter jejuni*	2	1	D	Variety of Chinese dishes
Vic.	Mar	Commercial caterer	Unknown	35	0	D	Unknown
Vic.	Mar	Restaurant	*S.* Typhimurium PT 44	22	2	A	Scrambled eggs
WA	Feb	Restaurant	Unknown	39	1	A	Duck pancakes
WA	Mar	Restaurant	*S.* Typhimurium PFGE profile 151	5	0	D	Unknown
Total				347	36		

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

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

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

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

MLVA Multi-locus variable number tandem repeat analysis.

PFGE Pulsed-field gel electrophoresis.

PT Phage type.

Table 3: Outbreaks of foodborne or suspected foodborne disease reported by OzFoodNet, 1 January to 31 March 2013, by food preparation setting

Food preparation setting	Outbreaks
Restaurant	16
Private residence	8
Takeaway	3
Commercial caterer	2
Aged care	1
Bakery	1
Correctional facility	1
National franchised fast food	1
Primary product	1
Total	34

To investigate these outbreaks, sites conducted 4 cohort studies, 3 case control studies and collected descriptive case series data for 23 investigations, while for 4 outbreaks, no individual patient data were collected. The evidence used to implicate food vehicles included analytical association between illness and food in 3 outbreaks and microbiological confirmation in 2 outbreaks. Descriptive evidence alone was obtained in 29 outbreak investigations.

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

Australian Capital Territory

There were 2 reported outbreaks of foodborne or suspected foodborne illness during the quarter. The aetiological agents were identified as *Salmonella* Typhimurium phage type (PT) 44 multi-locus variable number tandem repeat analysis (MLVA) profile[*,2] 03-11-07-11-523 and a suspected bacterial toxin.

New South Wales

There were 11 reported outbreaks of foodborne or suspected foodborne illness during the quarter. The aetiological agent was identified in eight of these outbreaks: five were due to *S.* Typhimurium and one each due to norovirus, *S.* Birkenhead and *S.* Cerro.

Description of key outbreaks

Ten people from 3 groups ate at a restaurant and seven of the ten subsequently became ill with gastrointestinal illness. All seven developed illness had consumed fried ice cream. All cases had stool samples that tested positive for *S.* Typhimurium MLVA profile 03-09-07-14-523 or 03-09-08-14-523. The New South Wales Food Authority (NSWFA) inspected the restaurant and took 5 samples of frozen and cooked fried ice cream balls that were made subsequent to the visits by the salmonellosis cases. All 5 sampled fried ice cream balls tested positive for *S.* Typhimurium MLVA profile 03-09-07-14-523 or 03-09-08-14-523. The restaurant proprietor was advised to only make fried ice cream using a pasteurised egg product. The NSWFA also inspected the egg farm that supplied the restaurant and found *Salmonella* with the same MLVA profile on an egg rinse sample. The egg farm was instructed to complete a clean-up of operations.

A cluster of 3 cases of an unusual *S.* Typhimurium MLVA profile (03-27-08-21-496) was investigated in January 2013. The cases occurred in December 2012 and were members of the same social club who had shared a dinner organised for 52 attending members. Further interviews found that eight of the members had gastroenteritis symptoms with two of those requiring admission to hospital. The group ate a menu of chicken and corn soup, roast chicken, potato salad, coleslaw, commercial frozen cheesecake, lemon meringue pie and trifle. The items were prepared in members' homes or bought from a grocery store. The group was not willing to provide further information about specific foods consumed and the exact cause remains unknown.

In January, investigators were notified of gastrointestinal illness in 3 people from a group of five that had shared a meal at a hotel restaurant. This was the only shared exposure for the group with no contact between their 4 different households or contact with ill people in the week prior to the meal. Symptoms included nausea, vomiting, abdominal cramping and diarrhoea with one or more of joint or muscle pain, headache and lethargy. The median incubation period was approximately 25 hours with a duration of 3 days. Foods consumed included chicken schnitzel and salad. The NSWFA conducted an inspection of the premises and identified food handlers and staff who had been unwell with symptoms of vomiting and had returned to work before the recommended 48 hour exclusion period. One clinical sample obtained from a case was positive for norovirus by polymerase chain reaction. A verbal warning was given to the hotel restaurant.

Investigators were notified of a cluster of *S.* Typhimurium MLVA profile 03-17-09-12-523, after a family of four was hospitalised. The only common high risk food consumed was banana smoothie made with milk and raw eggs. The eggs

* MLVA profiles are reported using the Australian coding convention agreed at a MLVA typing harmonisation meeting in Sydney in November 2011.

were from a small free-range egg farm. The family was provided with information about salmonellosis and the risks associated with consuming raw eggs.

Northern Territory

There were 2 reported outbreaks of foodborne illness during the quarter. The aetiological agents were identified as *S.* Typhimurium PT 9 and *Shigella sonnei* biotype a.

Description of key outbreaks

A mother and a 4-day-old neonate were notified with *S.* Typhimurium PT 9 positive stool cultures. The mother ate a home prepared Caesar salad with a raw egg dressing 1 day prior to the onset of symptoms. A day later she was admitted to hospital with diarrhoea and vomiting and gave birth the following day. The neonate's stool culture tested positive 4 days later for *S.* Typhimurium PT 9. Routine follow-up revealed 2 other family members had symptoms of gastroenteritis and had also consumed the Caesar salad. The salad was refrigerated after initial preparation and eaten over a period of 3 days, which resulted in staggered onset dates for the adults. No leftover food was available for testing.

An outbreak of diarrhoea affecting 5 people was attributed to consumption of a contaminated meal of unknown curried meat. Three people experienced symptoms of abdominal cramps and diarrhoea following the meal. Two of these people provided stool samples, which tested positive for *Shigella sonneii* biotype a. Routine follow-up of cases revealed that the meal had been prepared by an elderly woman with diarrhoea whose grandson also had diarrhoea.

Queensland

There were 4 reported outbreaks of foodborne or suspected foodborne illness during the quarter. The aetiological agent was identified in three of these outbreaks: one each due to *S.* Typhimurium, *Staphylococcus aureus* and ciguatera fish poisoning.

Description of key outbreaks

Eight cases of gastrointestinal illness were linked to the consumption of chicken sushi rolls, purchased from the same sushi venue in January. The outbreak was initially identified among 4 attendees of a birthday party held at a children's play café. Sushi rolls from the venue were included in the birthday party menu. Investigations identified further cases within the community who had not attended the party, but who had consumed sushi meals from the same establishment. Symptoms experienced by the cases included vomiting, diarrhoea and stomach cramps with onsets of illness approximately 1 to 3.5 hours after consuming the sushi (median incubation period 2.8 hours). Environmental swabs and food samples (including beef or chicken sushi, tuna, pork buns, rice and chicken) were collected for microbiological testing. A moderate to heavy growth of coagulase positive staphylococci were detected in 3 of 4 faecal specimens with 1 specimen positive for staphylococcal enterotoxin. Coagulase positive staphylococci (2 x 10^7 cfu/g) and staphylococcal enterotoxin were detected in 1 sample of teriyaki chicken, while *Bacillus cereus* (>10^4 cfu/g) was detected in beef and teriyaki chicken samples. Environmental investigations identified numerous time-temperature issues on site as well as poor food handling practices. The license of this food establishment was suspended until these issues were rectified.

Three cases of *S.* Typhimurium with the same MLVA profile (03-25-16-11-524) were notified in January. Investigations identified that the cases were from 2 separate groups of diners who had reported attending the same restaurant on the same night. Onset of symptoms ranged from 20 to 36 hours following their respective meals with 1 case hospitalised overnight due to their illness. The consumption of duck liver paté was common to all 3 cases. While no duck liver paté was available for microbiological testing, undercooked duck liver paté was the likely contributing factor for this outbreak. Restaurant management voluntarily removed this item from the menu following identification of this outbreak.

South Australia

There were 2 reported outbreaks of foodborne or suspected foodborne illness during the quarter. The aetiological agents were identified as *S.* Typhimurium PT 9 and norovirus.

Description of key outbreak

Investigators were notified of an outbreak of *S.* Typhimurium PT 9 associated with a restaurant in March. Nine people who consequently had gastrointestinal illness dined at the restaurant on the same day. Seven of the cases submitted faecal samples; all were confirmed as *S.* Typhimurium PT 9 with the MLVA profile 03-15-06-11-550. Through hypothesis generating interviews, it was found that all cases had consumed or tasted poached eggs and hollandaise sauce. An environmental investigation was conducted that identified the use of raw eggs in the hollandaise sauce, temperature abuse of eggs and the need for improvements in food handling as contributing factors. The restaurant kitchen has since been refurbished.

Tasmania

There was 1 reported outbreak of foodborne illness during the quarter. The aetiological agent was identified as *S.* Mississippi.

Description of outbreak

Investigators identified an outbreak of *S.* Mississippi associated with eating at a hotel restaurant over a 3-day period in December 2012. This outbreak was identified in early January through routine follow-up of salmonellosis cases. There were 11 confirmed cases and an estimated 25 suspected cases. Symptoms reported by confirmed cases were diarrhoea (100%), vomiting (50%), fever (50%) and abdominal pain (80%). The median incubation time was 30 hours. Of the 11 confirmed cases identified, all had consumed the salad that was served with the main meals in the exposure period. Two food handlers had reported illness and been excluded from work around the time of the outbreak. These food handlers did not have faecal samples collected at the time of illness.

A retrospective cohort study was performed utilising data collected from 19 cases that had been identified during the investigation and the co-diners of these cases. The salad was the only meal with a statistical association with illness (risk ratio (RR) undefined, $P=0.0007$). There were limitations to the study as cases were not reinterviewed with a specific questionnaire detailing all the individual specific exposures at the restaurant and several co-diners were not interviewed directly and their exposure information was relayed by their contacts (the confirmed cases). Due to these limitations, the findings of the study undertaken have to be interpreted with caution.

Victoria

There were 10 reported outbreaks of foodborne or suspected foodborne illness during the quarter. The aetiological agent was identified for seven of these outbreaks: three were due to *S.* Typhimurium; two were due to norovirus; and one each was due to *S.* Infantis and *Campylobacter jejuni*.

Description of key outbreaks

In January, investigators were notified of an outbreak of gastroenteritis amongst a group that attended a function catered by an off-site caterer. Of approximately 100 attendees, 61 were interviewed and 32 reported vomiting and/or diarrhoea with a median incubation period of 48 hours after eating morning tea. Six different types of sandwiches were served as well as scones with jam and cream and tea and coffee. Analysis of food exposures revealed that eating any food at the function had a statistically significant association with illness (odds ratio (OR) undefined; $P<0.0000$). Specifically, consumption of 1 of 2 different types of sandwiches (both containing ham and tomato) was statistically associated with illness. No faecal specimens were collected but the symptoms, duration and median incubation period were consistent with a viral pathogen as the cause.

In January, routine review of surveillance data identified an increase in cases of salmonellosis with residential addresses in 1 of 4 local government areas and a cluster investigation commenced. A day later the Microbiological Diagnostic Unit reported a cluster of cases of *S.* Infantis in this same geographical area. Initial case interviews identified the likely food source for this outbreak as being Vietnamese pork rolls, all purchased from the same bakery. Twenty-one cases (13 confirmed *S.* Infantis and 8 suspected cases) reported eating a variety of rolls from the bakery over a 7-day period. Egg butter made with raw eggs was consumed by the majority of cases. Shell eggs and raw chicken were sampled during the investigation, however, these were not from the same batch used during the outbreak period. *S.* Singapore was isolated from a rinse of the eggs and *S.* Infantis was isolated from the raw chicken. It is suspected that the outbreak was caused either by consumption of an ingredient of the pork roll that was cross contaminated by raw chicken or the egg butter.

In January, a *Salmonella* outbreak affecting 3 inmates of a prison was investigated. All had shared protein shakes containing raw eggs on multiple occasions during their incubation period. These drinks were prepared in a kitchenette and the eggs were not kept refrigerated. The eggs used in the drink were purchased from the canteen and not sourced from the main kitchen. *S.* Typhimurium PT 135a was isolated from the faecal specimens of 2 cases.

In January, an outbreak affecting 10 people who dined at a private residence was investigated. Sixteen people attended and 13 of these were interviewed. Eight people reported an onset of gastroenteritis with a median incubation period of 20.5 hours after eating the meal. An additional 2 cases were children of one of the cases. They were suspected to have been secondary cases as their onsets were 5 and 10 days after the dinner. A cohort analysis did not show an association with consumption of any of the foods and illness. It is suspected that the outbreak was caused by consumption of tiramisu which contained raw eggs, as this was the only food consumed by all of the cases.

Three people who consumed the tiramisu did not report any symptoms. *S.* Typhimurium PT 135a was isolated from the faecal specimens of 5 cases.

In March, a case of salmonellosis was notified by a general practitioner who reported that one of his patients had become unwell after attending a golf breakfast. His patient reported that a number of other attendees at this function had also become unwell. Investigation revealed that the case ate with a group of 31 golfers. Food served to the group was limited to scrambled eggs, bacon, mushrooms, tomatoes, bread and fruit. A retrospective cohort study was conducted, with 30 questionnaires completed. Twenty-two attendees reported having an onset of diarrhoea with a median incubation period of 22 hours from the breakfast meal. Nine cases had *S.* Typhimurium PT 44 isolated from their faecal specimen. Analysis of food exposures revealed a statistically significant association with consumption of scrambled eggs and illness (RR undefined, P=0.0004). Since 2007, *S.* Typhimurium PT 44 has been responsible for multiple outbreaks associated with consumption of raw or undercooked eggs in Victoria.

Western Australia

There were 2 reported outbreaks of foodborne or suspected foodborne illness during the quarter. *S.* Typhimurium was identified as the aetiological agent for one of the outbreaks.

Description of key outbreaks

At least 39 people became ill following a function held at a restaurant in February, with a median incubation period of 32.5 hours and duration of 1.3 days. The incubation period, duration of illness and symptoms suggested that the infectious agent was likely to be a virus such as norovirus. The 1 faecal specimen collected from an ill person was negative for common bacterial pathogens, but was not tested for viruses. In univariate analyses, illness was statistically associated with consumption of the Peking duck pancake (OR 56, 95% CI 7.8—undefined, P=<0.0001). The environmental investigation found that there was no documented temperature control for foods stored or received, that duck had been transported from the supplier without temperature control, and that the supplier inappropriately stored the duck in their cool room. Whilst there were potential food safety risks from these food handling, storage and transportation issues, these were not likely to be the cause of this outbreak. No staff illness was reported at the time of the outbreak at the restaurant or the supplier of the duck. It was concluded that the Peking duck pancake was the most likely food contaminated with a viral like pathogen but the means of contamination of the pancake was not identified.

Two cases with *S.* Typhimurium pulsed field gel electrophoresis (PFGE) type 151 ate independently at a restaurant on the same day in March. This is a rare *S.* Typhimurium PFGE type in Western Australia, with only 7 cases previously reported. Three other people who dined with these guests were also ill. Cases fell ill within 24 hours of the meal, but could not detail specific menu items eaten when interviewed. The local government area environmental health officer investigated the food business and found it had adequate food storage and handling and no staff reported illness. The cause of this suspected foodborne outbreak was not determined.

Multi-jurisdictional investigation

A previously reported multi-jurisdictional outbreak investigation of listeriosis (PFGE type 119A:44A:1) associated with the consumption of brie and/or camembert cheese[1] continued this quarter.

Cluster investigations

During the quarter, OzFoodNet sites investigated a number of clusters of infection for which no common food vehicle or source of infection could be identified. Aetiological agents identified during these investigations included: *S.* Typhimurium, *Cryptosporidium, Listeria monocytogenes* and Shiga toxin-producing *Escherichia coli*.

In January, an investigation commenced in New South Wales into a cluster of *S.* Wangata as part of an ongoing investigation to identify the source of this *Salmonella* serovar. Twenty-two cases were notified over a 2 month period. Cases had a median age of 42 years (range 1—81) and 50% were female. Place of residence included Hunter New England (6), North Coast (8), Northern Sydney Central Coast (3), South East Sydney (3), Sydney South West (1) and Greater Western (1). Seventeen cases were interviewed. Exposures of greatest interest included close proximity to rivers/creeks (65%), lizards (53%) and dogs (40%). The investigation is ongoing.

Comments

The majority of reported outbreaks of gastrointestinal illness in Australia are due to person-to-person transmission, and in this quarter, 74% of outbreaks (n=385) were transmitted via this route. The number of foodborne outbreaks this quarter

(n=34) is similar to the previous quarter (n=37) and to the 5-year mean of 39 outbreaks for the 1st quarter between 2008 and 2012.

Salmonella was identified as the aetiological agent in 50% of all foodborne or suspected foodborne outbreaks this quarter (17/34). *S.* Typhimurium was identified as the aetiological agent in 13 (38%) of the foodborne or suspected foodborne outbreaks (Table 2). Of the 17 outbreaks where *Salmonella* was implicated as the responsible agent, 47% (8/17) were associated with raw or undercooked egg products (raw egg dressings, butter and drinks and raw or undercooked egg meals and desserts).

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National Notifiable Diseases Surveillance System, 1 October to 31 December 2013

A summary of diseases currently being reported by each jurisdiction is provided in Table 1. There were 55,328 notifications to the National Notifiable Diseases Surveillance System (NNDSS) with a notification received date between 1 October to 31 December 2013 (Table 2). The notification rate of diseases per 100,000 population for each state or territory is presented in Table 3.

Table 1: Reporting of notifiable diseases by jurisdiction

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

Table 1 *continued*: Reporting of notifiable diseases by jurisdiction

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

* Infections with Shiga-like toxin (verotoxin) producing *Escherichia coli*.

NEC Not elsewhere classified.

Table 2: Notifications of diseases received by state and territory health authorities, 1 October to 31 December 2013, by date of diagnosis*

Disease	State or territory								Total 4th quarter 2013	Total 3rd quarter 2012	Total 4th quarter 2012	Last 5 years mean 4th quarter	Ratio	Year to date 2013	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	9	3	12	1	0	7	12	44	34	55	53.4	0.8	168	226.8
Hepatitis B (unspecified)‡	28	663	60	258	63	15	470	185	1,742	1,887	1,590	1,606.2	1.1	7,026	6,707.0
Hepatitis C (newly acquired)†	5	8	0	NN	14	2	47	36	112	99	118	102.8	1.1	404	410.2
Hepatitis C (unspecified)‡	36	900	49	653	121	51	553	243	2,606	2,773	2,412	2,546.8	1.0	10,314	10,504.2
Hepatitis D	0	1	0	3	0	0	6	3	13	8	10	7.8	1.7	53	36.4
Gastrointestinal diseases															
Botulism	0	0	0	0	0	0	0	0	0	1	0	0.0	0.0	4	0.6
Campylobacteriosis	82	NN	47	1,256	392	237	1,821	635	4,470	3,520	3,894	4,405.8	1.0	14,677	16,407.0
Cryptosporidiosis	6	147	14	176	27	9	137	59	575	420	438	392.6	1.5	3,848	2,612.0
Haemolytic uraemic syndrome	0	4	0	0	1	0	1	0	6	2	5	5.0	1.2	15	17.4
Hepatitis A	0	10	0	14	4	0	5	0	33	45	47	85.4	0.4	189	283.4
Hepatitis E	1	4	0	2	0	0	2	0	9	2	8	6.6	1.4	31	38.0
Listeriosis	0	4	2	0	0	0	7	2	15	15	28	19.4	0.8	76	78.8
STEC, VTEC§	1	8	0	7	10	0	1	2	29	78	34	32.8	0.9	179	102.6
Salmonellosis	43	929	84	1,066	230	48	680	308	3,388	2,249	2,823	2,635.8	1.3	12,795	10,649.0
Shigellosis	3	51	24	15	9	2	39	27	170	128	132	145.2	1.2	556	608.0
Typhoid	3	13	0	5	2	0	12	2	37	16	31	27.6	1.3	150	115.2
Quarantinable diseases															
Cholera	0	2	0	0	0	0	0	0	2	0	0	0.8	2.5	3	4.6
Highly pathogenic avian influenza in humans	0	0	0	0	0	0	0	0	0	0	0	0.0	0.0	0	0.0
Plague	0	0	0	0	0	0	0	0	0	0	0	0.0	0.0	0	0.0
Rabies	0	0	0	0	0	0	0	0	0	0	0	0.0	0.0	0	0.0
Severe acute respiratory syndrome	0	0	0	0	0	0	0	0	0	0	0	0.0	0.0	0	0.0
Smallpox	0	0	0	0	0	0	0	0	0	0	0	0.0	0.0	0	0.0
Viral haemorrhagic fever	0	0	0	0	0	0	0	0	0	0	0	0.0	0.0	0	0.0
Yellow fever	0	0	0	0	0	0	0	0	0	0	0	0.0	0.0	0	1.0

Table 2 *continued*: Notifications of diseases received by state and territory health authorities, 1 October to 31 December 2013, by date of diagnosis*

Disease	State or territory								Total 4th quarter 2013	Total 3rd quarter 2012	Total 4th quarter 2012	Last 5 years mean 4th quarter	Ratio	Year to date 2013	Last 5 years YTD mean
	ACT	NSW	NT	Qld	SA	Tas	Vic	WA							
Sexually transmissible infections															
Chlamydial infection[‖,¶]	306	5,279	758	4,934	1,112	354	4,718	2,751	20,212	20,419	19,785	17,391.6	1.2	82,463	71,923.0
Donovanosis	0	0	0	0	0	0	0	0	0	0	0	0.0	0.0	0	1.0
Gonococcal infection[¶]	29	1,012	451	626	161	22	796	521	3,618	3,622	3,521	2,640.6	1.4	14,932	10,443.6
Syphilis – congenital	0	0	1	0	0	0	0	0	1	3	0	1.0	1.0	7	4.0
Syphilis < 2 years duration[¶]	1	138	6	78	13	4	179	20	439	462	393	311.2	1.4	1,755	1,327.8
Syphilis > 2 years or unspecified duration[‡,¶]	4	98	14	71	30	1	149	24	391	474	322	324.6	1.2	1,729	1,346.6
Vaccine preventable diseases															
Diphtheria	0	0	0	1	0	0	0	0	1	0	0	0.0	0.0	2	0.8
Haemophilus influenzae type b	0	2	0	2	0	0	0	0	4	8	3	3.8	1.1	20	19.2
Influenza (laboratory confirmed)	79	1,078	199	1,488	1,694	114	1,202	600	6,454	16,983	3,070	2,485.0	2.6	28,279	30,693.6
Measles	1	15	0	32	13	0	16	10	87	44	30	20.0	4.4	158	126.4
Mumps	0	17	3	8	2	1	5	3	39	44	40	36.0	1.1	218	181.0
Pertussis	84	633	19	844	284	37	821	471	3,193	2,978	5,975	9,105.0	0.4	12,337	28,415.2
Pneumococcal disease (invasive)	6	94	11	54	27	11	91	45	339	554	354	363.2	0.9	1,547	1,705.8
Poliomyelitis	0	0	0	0	0	0	0	0	0	0	0	0.0	0.0	0	0.0
Rubella	1	0	0	3	1	0	1	0	6	11	6	6.2	1.0	25	40.4
Rubella – congenital	0	0	0	0	0	0	1	0	1	0	0	0.0	0.0	2	0.2
Tetanus	0	0	0	0	0	0	0	0	0	0	4	0.8	0.0	4	3.8
Varicella zoster (chickenpox)	7	NN	16	56	119	10	330	124	662	569	515	586.2	1.1	2,038	1,894.6
Varicella zoster (shingles)	15	NN	74	14	508	60	356	360	1,387	1,137	1,215	904.8	1.5	5,007	3,340.2
Varicella zoster (unspecified)	36	NN	3	1,467	34	26	858	321	2,745	2,712	2,295	1,908.8	1.4	9,935	6,913.2
Vectorborne diseases															
Arbovirus infection (NEC)	0	0	0	4	0	0	0	0	4	8	1	2.8	1.4	21	11.8
Barmah Forest virus infection	0	63	33	283	1	0	6	73	459	798	654	424.4	1.1	4,245	1,723.2
Dengue virus infection	1	70	10	61	24	6	78	84	334	512	229	247.2	1.4	1,845	1,110.2
Japanese encephalitis virus infection	0	0	0	0	0	0	0	0	0	2	0	0.0	0.0	4	0.4
Kunjin virus infection**	0	0	0	3	0	0	0	0	3	0	0	0.2	15.0	3	1.4
Malaria	2	19	5	19	3	1	17	17	83	107	96	105.8	0.8	414	440.0
Murray Valley encephalitis virus infection**	0	0	0	0	0	0	0	0	0	1	0	0.2	0.0	1	4.6
Ross River virus infection	1	92	88	379	36	3	42	322	963	680	707	840.6	1.1	4,309	5,061.0

Table 2 *continued*: Notifications of diseases received by state and territory health authorities, 1 October to 31 December 2013, by date of diagnosis

Disease	State or territory								Total 4th quarter 2013	Total 3rd quarter 2012	Total 4th quarter 2012	Last 5 years mean 4th quarter	Ratio	Year to date 2013	Last 5 years YTD mean
	ACT	NSW	NT	Qld	SA	Tas	Vic	WA							
Zoonoses															
Anthrax	0	0	0	0	0	0	0	0	0	0	0	0.0	0.0	0	0.2
Australian bat lyssavirus	0	0	0	0	0	0	0	0	0	0	0	0.0	0.0	1	0.0
Brucellosis	0	2	0	3	0	0	0	0	5	1	7	8.0	0.6	14	33.4
Leptospirosis	0	3	2	11	1	0	5	0	22	23	12	21.4	1.0	95	142.6
Lyssavirus (NEC)	0	0	0	0	0	0	0	0	0	0	0	0.0	0.0	0	0.0
Ornithosis	0	1	0	1	0	0	12	1	15	9	31	24.8	0.6	47	79.0
Q fever	0	53	1	52	3	0	8	3	120	116	86	89.2	1.3	475	348.0
Tularaemia	0	0	0	0	0	0	0	0	0	0	0	0.5	0.0	0	1.0
Other bacterial infections															
Legionellosis	2	22	4	36	22	2	14	23	125	172	98	86.0	1.5	507	323.8
Leprosy	0	1	0	1	1	0	1	0	4	3	2	2.6	1.5	13	8.6
Meningococcal infection††	0	14	0	10	3	0	5	2	34	49	41	54.6	0.6	149	247.8
Tuberculosis	4	118	10	30	20	1	99	45	327	336	378	383.4	0.9	1,274	1,319.4
Total	787	11,577	1,991	14,038	4,986	1,017	13,598	7,334	55,328	64,114	51,495			224,363	

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

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

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

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

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

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

** In the Australian Capital Territory, Murray Valley encephalitis virus infection and Kunjin virus infection are combined under Murray Valley encephalitis virus infection.

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

NN Not notifiable

NEC Not elsewhere classified

Totals comprise data from all states and territories. Cumulative figures are subject to retrospective revision so there may be discrepancies between the number of new notifications and the increment in the cumulative figure from the previous period.

Table 3: Notification rates of diseases, 1 October to 31 December 2013, 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	0.5	5.1	1.1	0.2	0.0	0.5	2.0	0.8
Hepatitis B (unspecified)§	29.9	36.3	102.0	22.6	15.2	11.7	33.4	30.4	30.7
Hepatitis C (newly acquired)‡	5.3	0.4	0.0	NN	3.4	1.6	3.3	5.9	2.5
Hepatitis C (unspecified)§	38.4	49.3	83.3	57.2	29.2	39.8	39.3	40.0	45.9
Hepatitis D	0.0	0.1	0.0	0.3	0.0	0.0	0.4	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	87.5	NN	79.9	110.0	94.7	185.0	129.4	104.4	116.0
Cryptosporidiosis	6.4	8.1	23.8	15.4	6.5	7.0	9.7	9.7	10.1
Haemolytic uraemic syndrome	0.0	0.2	0.0	0.0	0.2	0.0	0.1	0.0	0.1
Hepatitis A	0.0	0.5	0.0	1.2	1.0	0.0	0.4	0.0	0.6
Hepatitis E	1.1	0.2	0.0	0.2	0.0	0.0	0.1	0.0	0.2
Listeriosis	0.0	0.2	3.4	0.0	0.0	0.0	0.5	0.3	0.3
STEC, VTEC‖	1.1	0.4	0.0	0.6	2.4	0.0	0.1	0.3	0.5
Salmonellosis	45.9	50.9	142.9	93.4	55.5	37.5	48.3	50.6	59.7
Shigellosis	3.2	2.8	40.8	1.3	2.2	1.6	2.8	4.4	3.0
Typhoid fever	3.2	0.7	0.0	0.4	0.5	0.0	0.9	0.3	0.7
Quarantinable diseases									
Cholera	0.0	0.1	0.0	0.0	0.0	0.0	0.0	0.0	0.0
Human pathogenic avian influenza in humans	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
Plague	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
Rabies	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
Severe acute respiratory syndrome	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
Smallpox	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
Viral haemorrhagic fever	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
Yellow fever	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
Sexually transmitted infections									
Chlamydial infection¶,**	326.5	289.2	1,289.2	432.3	268.6	276.4	335.3	452.3	356.0
Donovanosis	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
Gonococcal infection**	30.9	55.4	767.1	54.8	38.9	17.2	56.6	85.7	63.7
Syphilis – congenital	0.0	0.0	1.7	0.0	0.0	0.0	0.0	0.0	0.0
Syphilis < 2 years duration**	1.1	7.6	10.2	6.8	3.1	3.1	12.7	3.3	7.7
Syphilis > 2 years or unspecified duration§,**	4.3	5.4	23.8	6.2	7.2	0.8	10.6	3.9	6.9
Vaccine preventable diseases									
Diphtheria	0.0	0.0	0.0	0.1	0.0	0.0	0.0	0.0	0.0
Haemophilus influenzae type b	0.0	0.1	0.0	0.2	0.0	0.0	0.0	0.0	0.1
Influenza (laboratory confirmed)	84.3	59.1	338.5	130.4	409.1	89.0	85.4	98.7	113.7
Measles	1.1	0.8	0.0	2.8	3.1	0.0	1.1	1.6	1.5
Mumps	0.0	0.9	5.1	0.7	0.5	0.8	0.4	0.5	0.7
Pertussis	89.6	34.7	32.3	73.9	68.6	28.9	58.3	77.4	56.2
Pneumococcal disease (invasive)	6.4	5.1	18.7	4.7	6.5	8.6	6.5	7.4	6.0
Poliomyelitis	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
Rubella	1.1	0.0	0.0	0.3	0.2	0.0	0.1	0.0	0.1
Rubella – congenital	0.0	0.0	0.0	0.0	0.0	0.0	0.1	0.0	0.0
Tetanus	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0

Table 3 *continued*: Notification rates of diseases, 1 October to 31 December 2013, by state or territory. (Annualised rate per 100,000 population)*,†

Disease	State or territory								Aust.
	ACT	NSW	NT	Qld	SA	Tas.	Vic.	WA	
Vaccine preventable diseases *cont'd*									
Varicella zoster (chickenpox)	7.5	NN	27.2	4.9	28.7	7.8	23.4	20.4	17.2
Varicella zoster (shingles)	16.0	NN	125.9	1.2	122.7	46.8	25.3	59.2	36.0
Varicella zoster (unspecified)	38.4	NN	5.1	128.5	8.2	20.3	61.0	52.8	71.3
Vectorborne diseases									
Arbovirus infection (NEC)	0.0	0.0	0.0	0.4	0.0	0.0	0.0	0.0	0.1
Barmah Forest virus infection	0.0	3.5	56.1	24.8	0.2	0.0	0.4	12.0	8.1
Dengue virus infection	1.1	3.8	17.0	5.3	5.8	4.7	5.5	13.8	5.9
Japanese encephalitis virus infection	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
Kunjin virus infection††	0.0	0.0	0.0	0.3	0.0	0.0	0.0	0.0	0.1
Malaria	2.1	1.0	8.5	1.7	0.7	0.8	1.2	2.8	1.5
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	1.1	5.0	149.7	33.2	8.7	2.3	3.0	52.9	17.0
Zoonoses									
Anthrax	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
Australia bat lyssavirus	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
Brucellosis	0.0	0.1	0.0	0.3	0.0	0.0	0.0	0.0	0.1
Leptospirosis	0.0	0.2	3.4	1.0	0.2	0.0	0.4	0.0	0.4
Lyssavirus (NEC)	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
Ornithosis	0.0	0.1	0.0	0.1	0.0	0.0	0.9	0.2	0.3
Q fever	0.0	2.9	1.7	4.6	0.7	0.0	0.6	0.5	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	2.1	1.2	6.8	3.2	5.3	1.6	1.0	3.8	2.2
Leprosy	0.0	0.1	0.0	0.1	0.2	0.0	0.1	0.0	0.1
Meningococcal infection‡‡	0.0	0.8	0.0	0.9	0.7	0.0	0.4	0.3	0.6
Tuberculosis	4.3	6.5	17.0	2.6	4.8	0.8	7.0	7.4	5.8

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

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

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

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

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

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

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

†† In the Australian Capital Territory, Murray Valley encephalitis virus infection and Kunjin virus infection are combined under Murray Valley encephalitis virus infection.

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

NEC Not elsewhere classified.

NN Not notifiable.

Australian childhood immunisation coverage, 1 April to 30 June cohort, assessed as at 30 September 2013

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

Introduction

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

The data show the percentage of children 'fully immunised' at 12 months, 24 months and 60 months, for 3-month birth cohorts of children at the stated ages between 1 April to 30 June 2013. 'Fully immunised' refers to vaccines on the National Immunisation Program Schedule, but excludes rotavirus, pneumococcal conjugate, varicella, and meningococcal C conjugate vaccines, and is outlined in more detail below.

'Fully immunised' at 12 months of age is defined as a child having a record on the ACIR of 3 doses of a diphtheria (D), tetanus (T) and pertussis-containing (P) vaccine, 3 doses of polio vaccine, 2 or 3 doses of PRP-OMP containing *Haemophilus influenzae* type b (Hib) vaccine or 3 doses of any other Hib vaccine, and 2 or 3 doses of Comvax hepatitis B vaccine or 3 doses of all other hepatitis B vaccines. 'Fully immunised' at 24 months of age is defined as a child having a record on the ACIR of 3 or 4 doses of a DTP-containing vaccine, 3 doses of polio vaccine, 3 or 4 doses of PRP-OMP Hib vaccine or 4 doses of any other Hib vaccine, 3 or 4 doses of Comvax hepatitis B vaccine or 4 doses of all other hepatitis B vaccines, and 1 dose of a measles, mumps and rubella-containing (MMR) vaccine. 'Fully immunised' at 60 months of age is defined as a child having a record on the ACIR of 4 or 5 doses of a DTP-containing vaccine, 4 doses of polio vaccine, and 2 doses of an MMR-containing vaccine.

A full description of the basic methodology used can be found in *Commun Dis Intell* 1998;22(3):36–37.

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

Results

The percentage of children 'fully immunised' by 12 months of age for Australia increased from the previous quarter by 0.5 of a percentage point to 90.9% (Table 1). Except for the Australian Capital Territory, all jurisdictions experienced small increases in coverage for all individual vaccines due at 12 months of age, ranging from 0.1 of a percentage point to 1.2 percentage points.

The percentage of children 'fully immunised' by 24 months of age for Australia increased marginally from the previous quarter by 0.2 of a percentage point to 92.3% (Table 2). There were no important changes in coverage for any individual vaccines due at 24 months of age or by jurisdiction.

Table 1: Percentage of children immunised at 12 months of age for the birth cohort 1 April to 30 June 2012, preliminary results, by disease and state or territory; assessment date 30 September 2013

Vaccine	State or territory								Aust.
	ACT	NSW	NT	Qld	SA	Tas.	Vic.	WA	
Total number of children	1,311	25,142	1,035	16,026	4,981	1,451	18,588	8,473	77,007
Diphtheria, tetanus, pertussis (%)	93.5	90.8	92.9	91.9	91.9	91.8	92.0	91.6	91.6
Poliomyelitis (%)	93.4	90.6	92.9	91.9	91.8	91.8	92.0	91.6	91.5
Haemophilus influenzae type b (%)	93.2	90.3	92.6	91.7	91.6	91.8	91.7	91.3	91.2
Hepatitis B (%)	93.1	90.3	92.6	91.6	91.4	91.7	91.6	90.9	91.1
Fully immunised (%)	92.7	90.0	92.4	91.5	91.2	91.6	91.3	90.7	90.9
Change in fully immunised since last quarter (%)	-0.7	+0.3	+1.0	+0.1	+1.0	+0.6	+0.8	+0.9	+0.5

The percentage of children 'fully immunised' by 60 months of age for Australia increased marginally from the previous quarter by 0.3 of a percentage point to 92.1% (Table 3). This maintains the improvement in coverage for this age milestone. There were no important changes in coverage for any individual vaccines due at 60 months of age or by jurisdiction.

The Figure shows the trends in vaccination coverage from the first ACIR-derived published coverage estimates in 1997 to the current estimates. There is a clear trend of increasing vaccination coverage over time for children aged 12 months, 24 months and 60 months (from December 2007). Coverage at 24 months is still higher than coverage at 12 months of age.

Figure: Trends in vaccination coverage, Australia, 1997 to 30 June 2013, by age cohorts

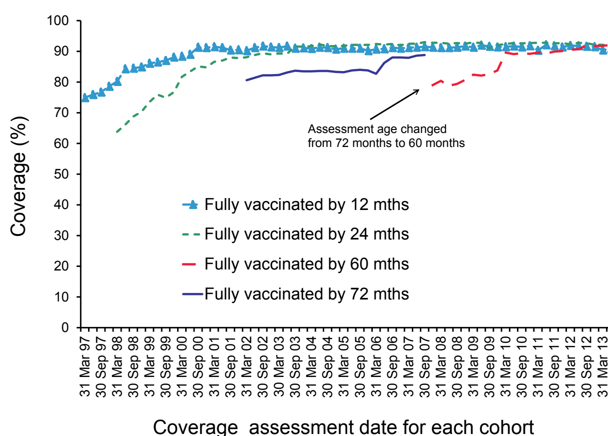


Table 2: Percentage of children immunised at 24 months of age for the birth cohort 1 April to 30 June 2011, preliminary results, by disease and state or territory; assessment date 30 September 2013*

Vaccine	State or territory								Aust.
	ACT	NSW	NT	Qld	SA	Tas.	Vic.	WA	
Total number of children	1,340	24,945	1,001	16,066	4,925	1,529	18,028	8,404	76,238
Diphtheria, tetanus, pertussis (%)	94.9	94.7	96.6	94.9	94.7	95.7	95.4	94.1	94.9
Poliomyelitis (%)	94.9	94.6	96.6	94.9	94.7	95.7	95.4	94.1	94.9
Haemophilus influenzae type b (%)	94.0	93.4	95.8	93.9	93.5	94.6	94.1	92.8	93.7
Measles, mumps, rubella (%)	94.1	93.4	96.0	94.1	93.8	94.6	94.2	93.2	93.8
Hepatitis B (%)	94.0	94.2	96.3	94.5	94.4	95.4	94.9	93.2	94.4
Fully immunised (%)	92.1	91.8	94.9	92.9	92.3	93.6	92.9	91.1	92.3
Change in fully immunised since last quarter (%)	-1.2	-0.1	+1.8	+0.6	-0.4	-0.6	+0.2	+0.6	+0.2

* The 12 months age data for this cohort were published in *Commun Dis Intell* 2013;37(4):E143.

Table 3: Percentage of children immunised at 60 months of age for the birth cohort 1 April to 30 June 2008, preliminary results, by disease and state or territory; assessment date 30 September 2013

Vaccine	State or territory								Aust.
	ACT	NSW	NT	Qld	SA	Tas.	Vic.	WA	
Total number of children	1,237	24,296	900	16,351	4,938	1,592	17,970	8,484	75,768
Diphtheria, tetanus, pertussis (%)	93.8	92.8	90.6	92.5	92.2	93.8	93.3	90.7	92.6
Poliomyelitis (%)	93.5	92.7	90.4	92.5	92.2	93.7	93.3	90.7	92.5
Measles, mumps, rubella (%)	93.5	92.6	90.9	92.5	92.2	93.8	93.1	90.8	92.5
Fully immunised (%)	93.0	92.3	90.4	92.1	91.9	93.2	92.8	90.3	92.1
Change in fully immunised since last quarter (%)	+1.8	+0.1	-0.8	+0.6	+0.3	-0.4	+0.4	+0.6	+0.3

HIV surveillance, 1 January to 31 March 2013

The Kirby Institute

Introduction

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

Tabulations of newly diagnosed HIV infections are based on data available 3 months after the end of the reporting interval indicated, to allow for reporting delay and to incorporate newly available information. More detailed information on diagnoses of HIV infection is published in the quarterly Australian HIV Surveillance Report, and annually in the *HIV, Viral Hepatitis and Sexually Transmissible Infections in Australia, Annual Surveillance Report*. The reports are available from the Kirby Institute, University of New South Wales, SYDNEY NSW 2052. Internet: http://www.kirby.unsw.edu.au Telephone: +61 2 9385 0900. Facsimile: +61 2 9385 0920. For more information see *Commun Dis Intell* 2014;38(1):E96–E97.

Results

Newly diagnosed cases of HIV infection reported for 1 January to 31 March 2013, are shown in Tables 1 and 2).

Table 1: Number of new diagnoses of HIV infection, 1 January to 31 March 2013, by sex and state or territory of diagnosis

Sex	ACT	NSW	NT	Qld	SA	Tas.	Vic.	WA	Total 1st quarter 2013	Total 1st quarter 2012	YTD 2013	YTD 2012
Female	0	6	0	11	3	0	12	5	37	44	37	44
Male	0	71	1	54	11	0	73	23	233	296	233	296
Not reported	0	0	0	0	0	0	0	0	0	0	0	0
Total*	0	77	1	65	14	0	85	28	270	340	270	340

* Totals include people whose sex was reported as transgender.

Table 2: Cumulative number of new diagnoses of HIV infection since the introduction of HIV antibody testing, 1985 to 31 March 2013, by sex and state or territory

Sex	ACT	NSW	NT	Qld	SA	Tas.	Vic.	WA	Aus.
Female	42	1,136	40	468	162	26	578	366	2,818
Male	333	16,492	195	3,960	1,222	171	7,086	1,687	31,146
Not reported	0	227	0	0	0	0	22	0	249
Total*	375	17,896	235	4,437	1,385	197	7,714	2,060	34,299

* Totals include people whose sex was reported as transgender.

Invasive pneumococcal disease surveillance Australia, 1 October to 31 December 2013

Rachel de Kluyver for the Enhanced Invasive Pneumococcal Disease Surveillance Working Group

Introduction

Invasive pneumococcal disease (IPD) is caused by the bacterium *Streptococcus pneumoniae* and results in illnesses such as pneumonia, bacteraemia and meningitis. There are currently more than 90 serotypes recognised worldwide, approximately half of which are found in Australia where IPD has been a nationally notifiable disease since 2001. The Communicable Diseases Network Australia (CDNA) established the Enhanced Invasive Pneumococcal Disease Surveillance Working Group (EIPDSWG) in 2000 to assist in developing and implementing a nationally standardised approach to the enhanced surveillance of IPD in Australia. This quarterly report documents trends in notified cases of IPD occurring in Australia in during 1 October to 31 December 2013.

Notification data are collected by all Australian states and territories under jurisdictional public health legislation and are forwarded to the Commonwealth under the National Health Security Act 2007. Notified cases are collated nationally in the National Notifiable Diseases Surveillance System (NNDSS). The data in this report are provisional and subject to change as laboratory results and additional case information become available. The data are analysed by diagnosis date, which is the onset date or where the onset date was not known, the earliest of the specimen collection date, the notification date, and the notification receive date. Data for this report were extracted on 31 October 2013. Crude rates were calculated using the Australian Bureau of Statistics estimated resident populations for Australia at 30 June of each year. Consideration of vaccination status of cases is outside the scope of this report. For more detailed reports readers are referred to the regular *Communicable Diseases Intelligence* supplements *Vaccine Preventable Diseases in Australia*.

In Australia, pneumococcal vaccination is recommended as part of routine immunisation for children, the medically at risk and older Australians. The 7-valent pneumococcal conjugate vaccine (7vPCV) was added to the National Immunisation Program (NIP) schedule for Indigenous and medically at-risk children in 2001 and for all children up to 2 years of age in 2005. The 13-valent pneumococcal conjugate vaccine (13vPCV) replaced the 7vPCV in the childhood immunisation program from July 2011. The 23-valent pneumococcal polysaccharide vaccine (23vPPV) was added to the NIP schedule for Aboriginal and Torres Strait Islander peoples aged 50 years or older in 1999 and for non-Indigenous Australians aged 65 years or older from January 2005.

Results

There were 336 cases of IPD reported to the NNDSS in the 4th quarter of 2013, bringing the year to date total to 1,543 cases (Table). The number of cases notified in the reporting period fell 39% from quarter 3 (n=552). Total number of cases in 2013 was a 15% reduction on the number of cases reported in 2012 (n=1,822).

Overall, Indigenous status was reported for 91% (n=307) of cases, ranging from 83% of cases reported by New South Wales to 100% of cases reported by the Australian Capital Territory, the Northern Territory, Tasmania and Western Australia. New South Wales continued its practice of targeted follow-up of notified cases of IPD aged 5 years or under and 50 years or older for core and enhanced data, whereas follow-up of all cases is undertaken in other states and territories. For this reporting period, Victoria temporarily returned to follow-up of all IPD cases. This may account for missing data among cases falling outside these age groups. Of cases with a reported Indigenous status, Aboriginal and Torres Strait peoples accounted for 13% (n=38) of all cases notified in the quarter (Table).

Serotype information was available for 89% (n=297) of all cases reported in the quarter, ranging from 7% of cases reported by South Australia to 100% of cases reported by the Australian Capital Territory and Tasmania. There were 2 cases reported in the quarter that were deemed by the reference laboratory as non-typable. For figures in this report, cases deemed non-typable are included in the 'Serotype not specified' category with respect to vaccine serotype group.

In the 4th quarter of 2013, notified cases were highest in children aged under 5 years (n=43), followed by the 85 years or over age group (n=38). This age distribution was evident in cases reported as non-Indigenous Australian (Figure 1). However in cases reported as Indigenous, the most prevalent age

Table: Notified cases of invasive pneumococcal disease, Australia, 1 October to 31 December 2013, by Indigenous status, serotype and state or territory

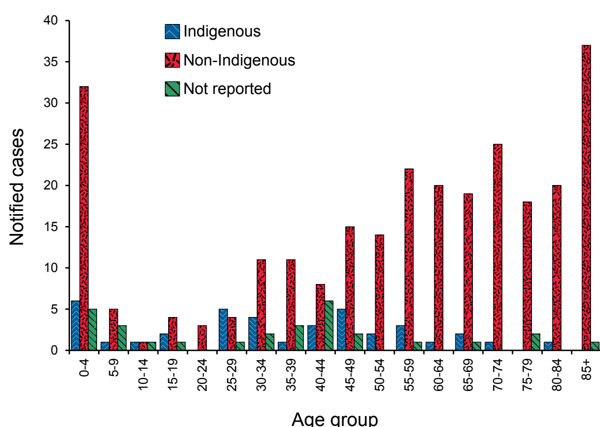
Indigenous status	ACT	NSW	NT	Qld	SA	Tas.	Vic.	WA	Total 4th qtr 2013	Total 3rd qtr 2013	Total 4th qtr 2012	Year to date 2013
Indigenous	0	2	10	15	0	0	1	10	38			
Non-Indigenous	6	76	1	34	26	11	80	35	269			
Not stated/ unknown	0	16	0	2	1	0	10	0	29			
Total	6	94	11	51	27	11	91	45	336	552	354	1,543
Indigenous status completeness* (%)	100	83	100	96	96	100	89	100	91			
Serotype completeness† (%)	100	94	91	94	7	100	99	96	89			

* Indigenous status completeness is defined as the reporting of a known Indigenous status, excluding the reporting of not stated or unknown Indigenous status.

† Serotype completeness is the proportion of all cases of invasive pneumococcal disease that were reported with a serotype or reported as non-typable. Serotype incompleteness may include when no isolate was available as diagnosis was by polymerase chain reaction and no molecular typing was performed; the isolate was not referred to the reference laboratory or was not viable; typing was pending at the time of reporting or no serotype was reported by the notifying jurisdiction to the National Notifiable Diseases Surveillance System.

groups were the under 5 years (n=6) followed by the 25–29 and 45–49 years age groups (n=5 each). In this quarterly report, 3 age groups have been selected for focused analyses. These age groups align with groups that carry the greatest burden of disease and against which the NIP is targeted.

Figure 1: Notified cases of invasive pneumococcal disease, Australia, 1 October to 31 December 2013, by Indigenous status and age group



Invasive pneumococcal disease in children aged less than 5 years

In the 4th quarter of 2013, 13% (n=43) of notified cases were aged less than 5 years. This was a marked decrease on the number of cases reported in the previous quarter (n=65) and similar to the number reported during the same period of 2012 (n=41) (Figure 2). The annual rate of notified cases in children less than 5 years of age did not change from 2012 and was the lowest on record at 13 per 100,000 population.

The majority of cases aged less than 5 years (95%, n=41) were reported with serotype information. Of these, 39% (n=16) were reported with a serotype included in the 7vPCV or the 13vPCV.

Notified cases aged less than 5 years with disease caused by the 6 additional serotypes targeted by the 13vPCV increased steadily over the period 2007 to 2011, particularly those caused by serotype 19A (Figure 3). However, cases of this type have decreased since the 4th quarter of 2011, reflecting the introduction of the 13vPCV on the universal childhood immunisation program in mid-2011. In the 4th quarter of 2013, there were 12 cases aged less than 5 years with disease due to serotype 19A, 2 cases due to serotype 3 and 2 cases of serotype 7F. Similar to the 4th quarter 2012, no cases in this age group were reported with disease caused by serotypes 1, 5 or 6A.

Invasive pneumococcal disease in Indigenous Australians aged 50 years or older

In the 4th quarter of 2013, 3% (n=10) of notified cases were reported as Indigenous Australians aged 50 years or over. This was the second lowest reported so far this year behind the 1st quarter (n=8) and was similar to the number reported

Figure 2: Notified cases and rates of invasive pneumococcal disease in those aged less than 5 years, Australia, 2002 to 31 December 2013, by vaccine serotype group

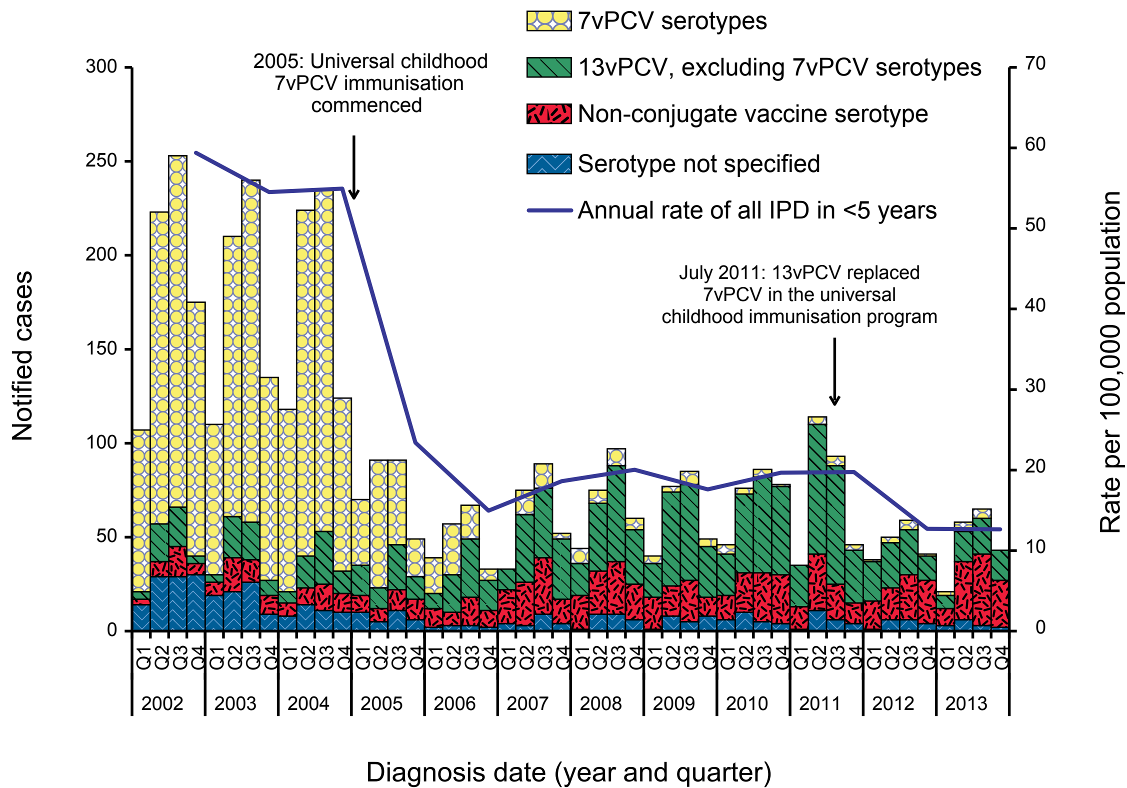


Figure 3: Notified cases of invasive pneumococcal disease caused by serotypes targeted by the 13-valent pneumococcal conjugate vaccine (excluding those targeted by 7-valent pneumococcal conjugate vaccine) and rates of all invasive pneumococcal disease, aged less than 5 years, Australia, 2002 to 31 December 2013

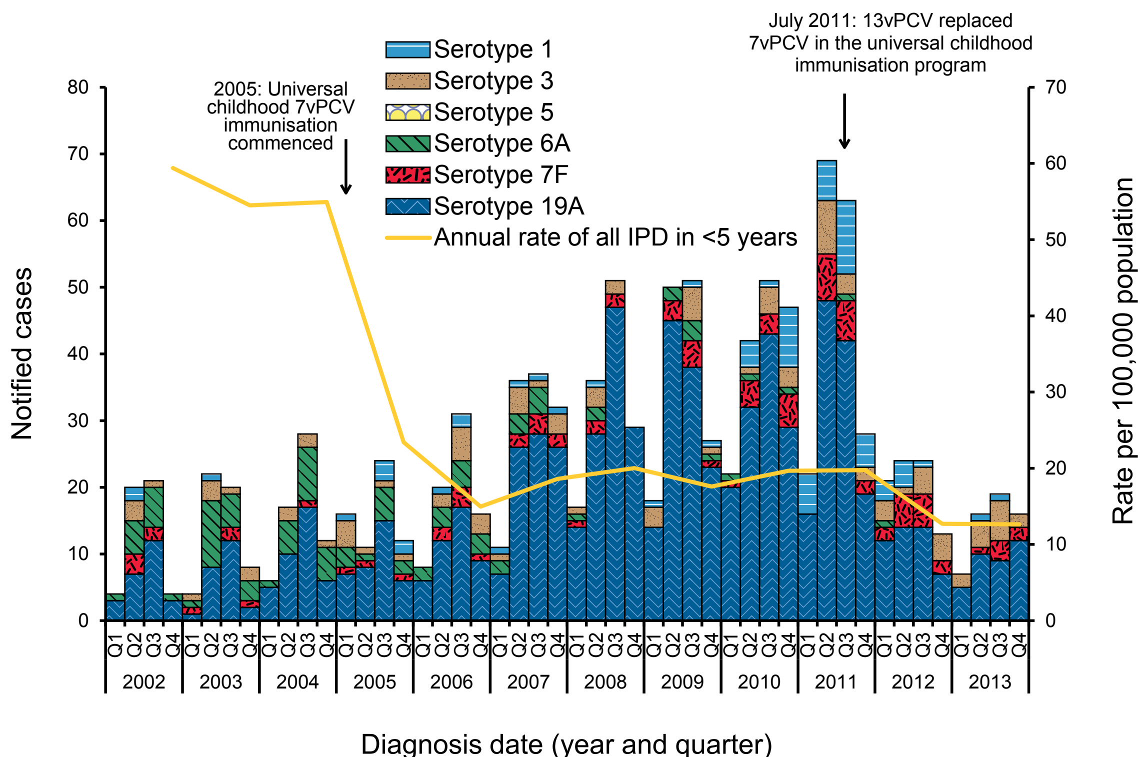
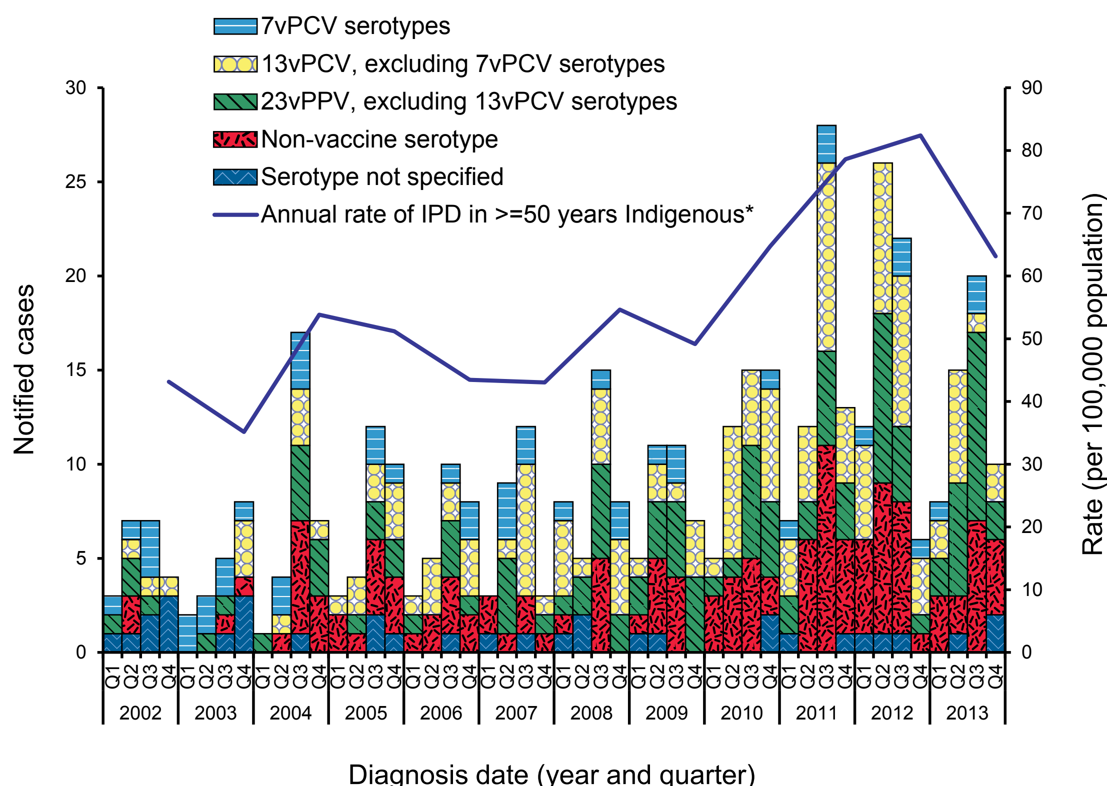


Figure 4: Notified cases and rates of invasive pneumococcal disease in Indigenous Australians aged 50 years or older, Australia, 2002 to 31 December 2013, by vaccine serotype group



* In 1999 immunisation with 23vPPV commenced for Indigenous Australians aged 50 years or over.

during the same period in 2012 (n=6). For 2010 to 2012, the annual rate of IPD in this group has tended to increase. An outbreak of disease caused by serotype 1 in Central Australia that commenced in late 2010 contributed, in part, to this increase.[1]

All but two of the cases notified in the 4th quarter of 2013 were reported with serotype information. Of these, 50% (n=4) were reported with disease due to serotypes targeted by the 23vPPV; the remainder reported disease due to a non-vaccine serotype (n=4).

Invasive pneumococcal disease in non-Indigenous Australians aged 65 years or older

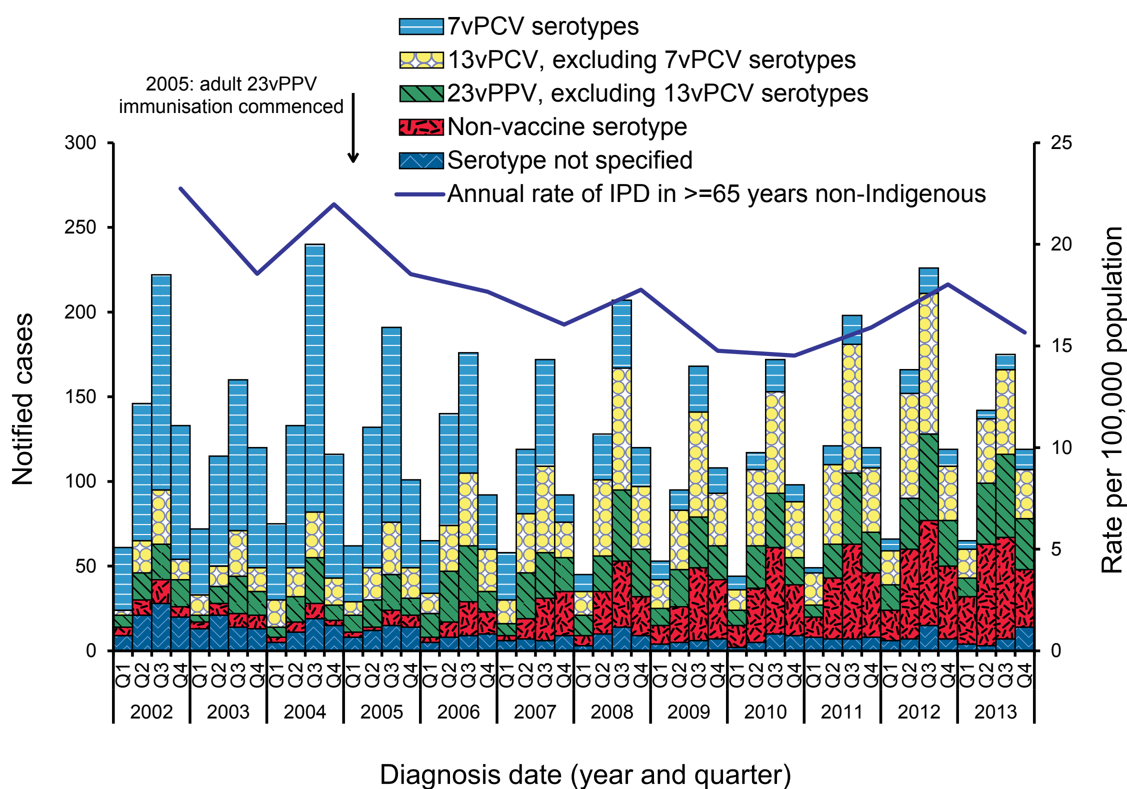
In the 4th quarter of 2013, 35% (n=119) of notified cases were reported as non-Indigenous Australians aged 65 years or over. This was a large decrease in the number of cases reported in the previous quarter (n=173) and was equal to the number reported during the same period of 2012 (n=119) (Figure 5). During 2013, the annual rate fell to 16 per 100,000 population, an 11% decrease from the rate of 2012 (18 per 100,000 population).

The majority (88%, n=105) of cases reported in this quarter were reported with serotype information. Of these cases, 68% (n=71) were reported with a serotype targeted by the 23vPPV. While the burden of disease in this age group has remained relatively stable, the profile of serotypes causing disease has changed over time. Disease due to serotypes targeted by the 7vPCV has reduced substantially in this age group and is likely due to herd immunity impacts from the childhood immunisation program.

Conclusion

The number of notified cases of IPD in the 4th quarter of 2013 was a 39% decrease on the previous quarter. Nationally, the pattern of disease has not changed from the 3rd quarter this year. Specifically, disease due to the serotypes targeted by the 13vPCV has continued to decline since the 13vPCV replaced the 7vPCV in the childhood immunisation program from July 2011. In addition, IPD associated with non-vaccine serotypes has decreased in all groups targeted for IPD vaccination. The overall rising trend in the rate of notified cases of IPD in Indigenous Australians aged 50 years or older continued, whereas disease rates

Figure 5: Notified cases and rates of invasive pneumococcal disease in non-Indigenous Australians aged 65 years or older, Australia, 2002 to 31 December 2013, by vaccine serotype group



in non-Indigenous Australians aged 65 years or older remained relatively stable despite an expansion of serotypes causing disease.

Acknowledgements

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Reference

1. Centre for Disease Control Northern Territory. Comments on notifications. *Northern Territory Disease Control Bulletin* 2012; 19(1): 29.

Surveillance summaries

Surveillance systems reported in *Communicable Diseases Intelligence*, 2014

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

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

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

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

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

This report describes the major features of the surveillance schemes for which CDI publishes regular reports.

Other surveillance schemes for which CDI publishes annual reports but are not described here include tuberculosis notifications (*Commun Dis Intell* 2014;38(1):E3–E15), the Australian Mycobacterium Reference Laboratory Network (*Commun Dis Intell* 2013;37(1):E40–E46), and the Australian Rotavirus Surveillance Program (*Commun Dis Intell* 2014;38(4):E37–E43).

Arbovirus and malaria surveillance

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

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

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

Further details are provided in the NAMAC annual report (*Commun Dis Intell* 2013;37(1):E1–E20).

Australian Childhood Immunisation Register

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

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

Medicare Australia coverage reports for the 3 milestones are published in CDI each quarter. Coverage estimates are provided for each state and territory and Australia as a whole and for each individual vaccine assessed at each milestone. Changes in 'fully immunised' coverage from the previous quarter are also included in the tables.

A commentary on ACIR immunisation coverage estimates is included with the tables in each issue and graphs are used to provide trends in immunisation coverage.

An immunisation coverage report is also published in CDI on an annual basis and provides more detailed data on immunisation coverage for all recommended vaccines by age group which are funded by the Immunise Australia Program, timeliness of immunisation, small area coverage estimates and data on conscientious objection to immunisation.

Australian Gonococcal Surveillance Programme

The Australian Gonococcal Surveillance Programme (AGSP) is a continuing program to monitor antimicrobial resistance in *Neisseria gonorrhoeae* and includes the reference laboratories in all states and territories. These laboratories report data on sensitivity to an agreed core group of antimicrobial agents on a quarterly basis and provide an expanded analysis as an annual report in CDI

(*Commun Dis Intell* 2013;37(3):E233–E239). The antibiotics that are currently routinely surveyed are the penicillins, ceftriaxone, ciprofloxacin and spectinomycin, all of which are administered as single dose regimens. A major purpose of the AGSP is to help define standard protocols for antibiotic treatment of gonococcal infection. When *in vitro* resistance to a recommended agent is demonstrated in 5% or more of isolates, it is usual to reconsider the inclusion of that agent in current treatment schedules. Additional data are also provided on other antibiotics from time to time. At present, all laboratories also test isolates for the presence of high level resistance to the tetracyclines and intermittent surveys of azithromycin resistance are conducted. Comparability of data is achieved by means of a standardised system of minimal inhibitory concentration (MIC) testing and a program-specific quality assurance process.

Australian Meningococcal Surveillance Programme

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

Data are reported annually and quarterly in CDI. Data in the quarterly reports are restricted to a description of the number of cases per jurisdiction, and serogroup where known. A full analysis of laboratory-confirmed cases of IMD, including phenotyping and antibiotic susceptibility data are published annually (*Commun Dis Intell* 2013;37(3):E224–E232).

Australian Paediatric Surveillance Unit

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

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

The activities of the APSU are funded in part by the Australian Government Department of Health, and the NHMRC Practitioner Fellowship No: 1021480 (E Elliott). The Faculty of Medicine, The University of Sydney, and the Royal Australasian College of Physicians, Division of Paediatrics and Child Health, and the Kids Research Institute, Sydney Children's Hospitals Network provide in-kind support. APSU publishes an annual report (*Commun Dis Intell* 2013;37(4):E394–E397). For further information please contact the APSU Director, Professor Elizabeth Elliott on telephone: +61 2 9845 3005, facsimile +61 2 9845 3082, email: apsu@chw.edu.au; Internet: http://www.apsu.org.au

Australian National Creutzfeldt-Jakob Disease Registry

Surveillance for Creutzfeldt-Jakob disease (CJD) in Australia is conducted through the Australian National Creutzfeldt-Jakob Disease Registry (ANCJDR). CJD is listed as a notifiable disease in all Australian states and territories. The ANCJDR is under contract to the Commonwealth to identify and investigate all suspect cases of transmissible spongiform encephalopathy in Australia. An annual update is published in CDI (*Commun Dis Intell* 2013;37(2):E115–E120).

Australian Sentinel Practice Research Network

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

The list of conditions is reviewed annually by the ASPREN management committee and an annual report is published. In 2014, 4 conditions are being monitored; all of which are related to communicable diseases. These are influenza like illness (ILI), gastroenteritis, chickenpox and shingles.

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

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

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

Influenza-like illness – record once only per patient

Must have the following: fever, cough and fatigue.

Gastroenteritis – record once only per patient

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

Chickenpox – record once only per patient

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

Shingles – record once only per patient

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

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

HIV surveillance

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

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

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

An annual surveillance report, *HIV, Viral Hepatitis and Sexually Transmissible Infections in Australia Annual Surveillance Report* has been published by the Kirby Institute since 1997. The Annual Surveillance Report, available through the Kirby Institute web site (http://www.kirby.unsw.edu.au), provides a comprehensive analysis and interpretation of surveillance data on HIV, viral hepatitis and sexually transmissible infections in Australia. The report *Bloodborne viral and sexually transmitted infections in Aboriginal and Torres Strait Islander*

people: Surveillance and Evaluation Report has been published from 2007, as an accompanying document to the annual surveillance report. The Surveillance and Evaluation Report provides detailed analysis and interpretation of the occurrence of these infections in Aboriginal and Torres Strait Islander communities in Australia.

Invasive Pneumococcal Disease Surveillance Program

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

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

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

IPD data are reported annually (*Commun Dis Intell* 2012;36(2):E151–E165) and quarterly in CDI. These reports include analysis notification and laboratory data collected through the NNDSS.

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

National Influenza Surveillance Scheme

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

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

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

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

National Notifiable Diseases Surveillance System

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

More than 60 communicable diseases agreed upon nationally are reported to NNDSS, although not all are notifiable in each jurisdiction. Data are sent electronically from states and territories daily (business days only in some jurisdictions). The system is complemented by other surveillance systems, which provide information on various diseases, including three that are not reported to NNDSS (HIV, and the classical and variant forms of CJD).

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

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

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

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

OzFoodNet: enhanced foodborne disease surveillance

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

OzFoodNet reports quarterly on investigations of outbreaks and clusters of gastroenteritis potentially related to food. Annual reports have been produced and published in CDI since 2001 with the most recent being the 2010 annual report (*Commun Dis Intell* 2012;36(3):E213–E241). Data are reported from all Australian jurisdictions.

References

1. Last JM. A dictionary of epidemiology. New York: Oxford University Press, 1988.
2. Hall R. Notifiable diseases surveillance, 1917 to 1991. *Commun Dis Intell* 1993;226–236. Available from: http://www.health.gov.au/internet/main/publishing.nsf/Content/cda-pubs-annlrpt-oz_dis19_91.htm Accessed March 2012.

Administration

Communicable Diseases Intelligence instructions for authors

Communicable Diseases Intelligence (CDI) is published quarterly (March, June, September and December) by the Health Emergency Management Branch, Office of Health Protection, Australian Government Department of Health.

The aim of CDI is to disseminate information on the epidemiology of communicable disease in Australia, including surveillance, prevention and control.

The objectives of CDI are to:

- report on surveillance of communicable diseases of relevance to Australia;
- publish other articles relevant to communicable disease epidemiology in Australia; and
- provide information on other activities relevant to the surveillance, prevention and control of communicable disease in Australia.

CDI invites contributions dealing with any aspect of communicable disease epidemiology, surveillance, prevention or control in Australia. Submissions can be in the form of original articles, short reports, surveillance summaries or letters to the Editor.

CDI publishes guidelines and position papers from the Communicable Diseases Network Australia and its expert sub-committees and may invite guest editorials and review articles on occasion.

Manuscripts for submission

Manuscripts submitted to CDI must be offered exclusively to the journal. All manuscripts should be accompanied by a covering letter that should include:

- confirmation that the manuscript content (in part or in full) has not been submitted or published elsewhere; and
- whether the manuscript is being submitted as an article, short report, surveillance summary, outbreak report or case report.

In addition, manuscripts should include a title page that should contain the following information:

- title (e.g. Prof, Dr, Ms, Miss, Mrs, Mr), full name including middle initial, position held, and institution at the time the article was produced, of each author;
- name of corresponding author, including current postal address, telephone, and email; and
- word count of the main text and of the abstract.

On receipt of a manuscript, authors will be sent a brief acknowledgement. Accepted manuscripts are edited for style and clarity and final proofs are returned to the corresponding author for checking prior to publication.

Authorship

Authorship should be based on substantial contribution to the article. Each author should have participated sufficiently to take public responsibility for the article. Others contributing to the work should be recognised in the acknowledgements.

Types of manuscript

Original articles

The text of articles must be structured to contain an abstract, introduction, methods, results, discussion, acknowledgements and references. Manuscripts submitted as articles must be 3,000 words or less and will be peer-reviewed.

Original articles may be submitted at any time and will be included in an issue once their review and revision has been completed. Articles may be published ahead of the scheduled issue, in the 'early release' format.

Systematic reviews submitted to CDI will be expected to conform to the Preferred Reporting Items for Systematic Reviews and Meta-Analyses (PRISMA) guidelines (http://www.prisma-statement.org/).

Letters to the Editor

The editorial team welcome comments on articles published in CDI in the form of letters to the Editor. Letters should normally be less than 500 words, include no more than a single chart and less than six references.

Short reports

Short reports may be submitted for peer review or for publication without peer review, depending on the content. Articles of particular relevance for rapid dissemination (such as timely outbreak reports) may be fast-tracked for early release prior to the next issue of CDI. Please discuss your requirements with the editorial team. Short reports may include an abstract. Types of short reports include:

Surveillance summaries

A report of 1,000 words or less that briefly reports on changes in the local epidemiology of a communicable disease, changes in surveillance systems, or new interventions, such as introducing vaccination in an at-risk group. Surveillance summaries should provide a brief description of the setting and a discussion of the significance of the events, changes or interventions.

Case reports

Brief reports of 500 to 1,000 words on cases of communicable disease will be considered based on their public health significance. Authors must note the instructions on the protection of patient's right to privacy (refer to the Ethics committee approvals and patients' right to privacy below). Some discussion of the significance of the case for communicable disease control should be included.

Outbreak reports

Reports of communicable disease outbreaks of 500 to 1,000 words will be considered for publication based on their public health significance. Reports should include details of the investigation, including results of interventions and the significance of the outbreak for public health practice. More comprehensive reports on outbreaks should be submitted as articles.

An outbreak report may be structured as below (the subheadings can be adjusted to suit), or may be unstructured if very brief.

Most outbreak reports will present only the descriptive epidemiology of the outbreak, with suspected risk factors for infection. The findings of any analytic study would usually be presented in an article at a later date, though authors may choose to present preliminary analyses from analytic studies.

Suggested structure

Abstract
A very brief unstructured abstract should be included.

Background and methods
Including initial detection of the outbreak, case finding and interview techniques, study design and any statistical methods.

Description of outbreak
Case definition, number of cases, number laboratory confirmed, symptoms. Time, place and person, epidemic curve.

A maximum of 2 tables and/or figures is suggested.

Laboratory, trace back and environmental investigations
Details of the proportion of laboratory confirmation of cases.

Public health response
A very brief description of any actions taken to prevent further cases may be included.

Discussion
Including the significance of the outbreak for public health practice.

References
A maximum of 20 references is suggested.

Peer review process

Articles provisionally accepted for publication will undergo a peer review process and articles may be rejected without peer review. Short reports may be submitted for peer review, or may be reviewed at the discretion of the Editor. Articles will be subject to review by two experts in the field and short reports by one or two reviewers (if any).

When submitting your manuscript, you may specify reviewers who are qualified to referee the work, who are not close colleagues and who would not have a conflict of interest. Suggestions regarding reviewers will be considered, however, the Editor has the final decision as to who to invite to review a particular article.

Authors may be asked to revise articles as a result of the review process before the final decision about publication is made by the Editor. Revised articles are to be returned with a covering letter addressing each comment made by each reviewer.

Annual reports and quarterly reports are not subject to peer review.

Document preparation

Articles and reports must be written in clear, comprehensible English. Authors should pay particu-

lar attention to the style guides, web accessibility requirements and table and figure formatting requirements provided on these pages.

Articles are only accepted in electronic form, in Microsoft Word and Microsoft Excel. Graphics may be provided in a range of other formats (see section below on illustrations). In addition:

- Arial font is preferred but if not available use Times New Roman.
- Abstracts should not exceed 250 words. Do not cite references in abstracts.
- Structured abstracts are acceptable.
- Include up to 10 keywords.
- Avoid too many abbreviations.

Manuscripts should be submitted with a one or two sentence summary of the article.

Tables

Tables and table headings should be located within the body of the manuscript and all tables should be referred to within the results section.

Information in tables should not be duplicated in the text.

Headings should be brief.

Simplify the information as much as possible, keeping the number of columns to a minimum and avoid merged cells as much as possible.

Separate rows or columns are to be used for each information type (e.g. percentage and number should be in separate columns rather than having one in parentheses in the same column).

If abbreviations are used these should be explained in a footnote.

Footnotes should use the following symbols in sequence:

* † ‡ § || ¶ ** †† ‡‡

Do not use blank rows or blank columns for spacing.

A short summary of each table should be included to satisfy government accessibility requirements (refer to Web accessibility requirements).

Figures and illustrations

Figures and illustrations, including headings, should be provided in the body of the manuscript and should be referred to within the results section. They should also be provided as a separate file.

Examples of each of the following can be found in the on-line version of Instructions to authors (http://www.health.gov.au/internet/wcms/publishing.nsf/Content/cda-pubs-cdi-auth_inst.htm)

A long text description should be included to satisfy government accessibility requirements (refer to Web accessibility requirements).

Figures

Use Microsoft Excel.

Each figure should be created as a separate worksheet rather than as an object in the datasheet (use the 'as new sheet' option for chart location).

The numerical data used to create each figure must be included on a separate worksheet (see example on the Department of Health web site).

Worksheets should be appropriately titled to distinguish each graph (e.g. Figure 1, Figure 2; Figure 1 data, Figure 2 data).

Do not include the graph heading on the Excel worksheet.

Graphs should be formatted to CDI requirements as much as possible. These requirements are available on the Health web site (http://www.health.gov.au/internet/main/publishing.nsf/Content/cda-pubs-cdi-auth_excel_fig.htm).

Photographs

Photographs may be submitted if required.

Photos need to be at least 300 dpi.

Electronic copies should be saved in Adobe Photoshop, or similar graphic software in one of the following graphic formats (in preferential order):

- PSD
- TIFF
- EPS
- JPEG (JPG).

Illustrations

Illustrations or flow charts can be included if required.

Images should preferably be at least 300 dpi.

Electronic copies of computer-generated illustrations should preferably be saved in a vector image program such as Adobe Illustrator or other simi-

lar graphic but charts created in either Word or PowerPoint are acceptable. Use a sans serif font for figures (e.g. Arial). Symbols, lettering and numbering should be clear and large enough to be legible when reduced in size.

Maps

Maps created by mapping programs such as MapInfo or ArcGIS should be saved at 300 dpi and in one of the following graphic formats (in preferential order) to allow editing of font size and colours:

- AI
- EMF

If this is not possible the following graphic formats should be used (in preferential order):

- TIFF
- EPS
- GIF.

Other images

Other images may be submitted in one of the following graphic formats (in preferential order):

- PSD
- TIFF
- EPS, or
- GIF.

Authors should aim for maximum levels of contrast between shaded areas. Use a sans serif font for text. Symbols, lettering and numbering should be clear and large enough to be legible when reduced in size.

Web accessibility requirements

The Australian Government is required to meet level AA of the Web Content Accessibility Guidelines version 2.0 (WCAG 2.0). These guidelines include the need for alternate methods of presenting the information depicted in images—including figures and maps—for readers with vision impairment and other disabilities using text readers. Complex tables also present challenges for text readers.

Articles and reports should be submitted with:

- a short summary of any tables
- a long text description of any figures;
- a long text description of any maps, flowcharts, or other images. For thermal maps showing disease rates by statistical location, a data table may be a preferred alternative.

Keep in mind that the description should be sufficient for a sight impaired person to understand what the information image is trying to convey.

Samples of descriptors for tables and figures can be found here on the Health web site (http://www.health.gov.au/internet/main/publishing.nsf/Content/cda-pubs-cdi-auth_web.htm).

Further information about WCAG 2.0 is available from the Australian Government Information Management Office (http://agimo.gov.au/)

References

References should be identified consecutively in the text using the Vancouver reference style. Any punctuation should precede the reference indicators.

Abbreviate journal names as in the PubMed journal database (http://www.ncbi.nlm.nih.gov/entrez/query.fcgi?db=journals) (e.g. *Commun Dis Intell*). Include the surnames and initials of all authors (or only the first six authors, et al, if there are more than six). Cite the first and last page numbers in full, and specify the type of reference (e.g. letter, editorial).

Examples of the Vancouver reference style are available on the Health web site. See also the International Committee of Medical Journal Editors Uniform requirements for manuscripts submitted to biomedical journals *Ann Intern Med* 1997;1126:36–47 (http://www.nlm.nih.gov/bsd/uniform_requirements.html).

Cite personal communications and unpublished papers in the text, not in the reference list, with the exception of material that has been accepted for publication (in press). Obtain written permission from people cited, and include their title, position and affiliation.

The accuracy of references is the responsibility of authors.

Ethics committee approvals and patients' rights to privacy

All investigations on human subjects must include a statement that the subjects gave their written informed consent, unless data collection was covered by public health legislation or similar studies have been considered by a relevant ethics committee and a decision made that its approval was not required. The name of the ethics committee that

gave approval for the study should be included in the text. Alternatively, if approval is not required a statement to this effect should appear in the manuscript.

Ethical approval and patient consent may also be required for case reports. Identifying details about patients should be omitted if they are not essential, but data should never be altered or falsified in an attempt to attain anonymity.

Copyright

All authors are asked to transfer copyright to the Commonwealth before publication. A copyright form will be sent to the corresponding author. All authors are required to sign the copyright release. The Commonwealth copyright will be rescinded if the article is not accepted for publication.

Submission of manuscripts

Manuscripts should be provided electronically by email to: cdi.editor@health.gov.au

Please contact the editorial team at cdi.editor@health.gov.au if you require any further information.

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