Australian Government
Department of Health and Ageing

Communicable Diseases Intelligence

Volume 34 Number 2 Quarterly report June 2010

Annual reports

Peer-reviewed articles

Short reports

Quarterly reports

Errata

ISSN 0725-3141
ISSN 1445-4866 Online

Communicable Diseases Intelligence aims to diseminate information on the epidemiology and control of communicable diseases in Australia. *Communicable Diseases Intelligence* invites contributions dealing with any aspect of communicable disease epidemiology, surveillance or prevention and control in Australia. Submissions can be in the form of original articles, short reports, surveillance summaries, reviews or correspondence. Instructions for authors can be found in *Commun Dis Intell* 2009;33:87–89.

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

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This journal is indexed by *Index Medicus*, Medline and the Australasian Medical Index

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Printed by Union Offset, Canberra

Annual reports

Annual Report of the Australian Gonococcal Surveillance Programme, 2009

The Australian Gonococcal Surveillance Programme

Abstract

The Australian Gonococcal Surveillance Programme (AGSP) monitors antibiotic susceptibility testing of *Neisseria gonorrhoeae* isolated in all states and territories. In 2009 the *in vitro* susceptibility of 3,220 isolates of gonococci from public and private sector sources was determined by standardised methods. Varying antibiotic susceptibility patterns were again seen across jurisdictions and regions. Resistance to the penicillins nationally was 36% and, with the exception of the Northern Territory, ranged between 19% in Queensland and 52% in Victoria. Quinolone resistance, most at high minimal inhibitory concentration (MIC) levels, was 43% nationally (excepting the Northern Territory), and ranging from 30% in Queensland to 60% in Victoria. Decreased susceptibility to ceftriaxone (MIC 0.06 mg/L or more), was found nationally in 2% of isolates. Nationally, all isolates remained sensitive to spectinomycin. Azithromycin resistance surveillance was performed in New South Wales, Queensland, Western Australia, the Northern Territory and South Australia, and was found to be present in low numbers of gonococci with MIC values up to 16 mg/L. In larger urban centres the ratio of male to female cases was high, and rectal and pharyngeal isolates were common in men. In other centres and in rural Australia the male to female ratio of cases was lower, and most isolates were from the genital tract. *Commun Dis Intell* 2010;34(2):89–95.

Keywords: antimicrobial resistance; disease surveillance; gonococcal infection; *Neisseria gonorrhoeae*

Introduction

Gonorrhoeal infections continue to be a public health challenge globally, and effective antibiotic treatment is fundamental to disease control at the population level.[1] Around the world, the increasing prevalence of antimicrobial resistance (AMR) in *Neisseria gonorrhoeae,* and its impact on treatment outcome is a major concern.[1] Resistance to the penicillins, tetracyclines and macrolides has necessitated the widespread removal of these low cost, oral agents from standard treatment regimens. In urban Australia,[2] and in neighbouring countries, the emergence of high levels of resistance to fluoroquinolone antibiotics has compromised the efficacy of this antibiotic group at both the individual and population health level. This has resulted in widespread replacement with extended spectrum cephalosporin antibiotics as the recommended first line treatment for gonorrhoea in Australia and elsewhere.[3] Unusually, but importantly in Australia however, treatments based on the penicillins remain effective in many rural centres where extremely high disease rates persist.[2]

In large centres in urban Australia, AMR in *Neisseria gonorrhoeae* has long been influenced by the introduction of multi-resistant overseas strains.[2] There are an increasing number of reports from overseas sources[4,5] of treatment failures with orally administered extended spectrum cephalosporin. In Australia, oral, extended spectrum cephalosporin antibiotics are not available, therefore the injectable form (ceftriaxone) is recommended for use in high doses.[3] No treatment failures have yet been reported following ceftriaxone treatment of genital tract gonorrhoea; however there have been 2 instances of failure of treatment of pharyngeal gonorrhoea reported in Sydney[6] where elimination of intercurrent genital tract infection with the same organism was achieved. The gonococci involved both had raised minimal inhibitory concentrations (MICs) for ceftriaxone.

Strategies for treating and controlling gonorrhoea are based on single dose regimens effecting cure in a minimum of 95%, and the formulation of these regimens is reliant on data derived from continuous AMR monitoring of gonococcal isolates to the antibiotics in clinical use.[1,7] Recently, and following the reports of treatment failures with orally administered extended spectrum cephalosporins,[4,5] calls have been made internationally for enhanced surveillance of all forms of gonococcal AMR in order to optimise gonococcal antibiotic treatment.[8] Since 1981 the Australian Gonococcal Surveillance Programme (AGSP) has monitored the susceptibility of *N. gonorrhoeae* continuously, making it the longest, continually running national surveillance

system for gonococcal AMR.[9] The emergence and spread of penicillin and quinolone resistant gonococci in major cities in Australia has been well documented.[2]

This analysis of AMR in *N. gonorrhoeae* in Australia was derived from data collated by the AGSP during the 2009 calendar year. It provides information regarding the gonococcal isolates showing resistance to multiple antibiotics, including those with decreased susceptibility to ceftriaxone.[2,10]

Methods

Ongoing monitoring of AMR in gonococci in Australia is performed by the AGSP through a collaborative program conducted by reference laboratories in each state and territory. The AGSP is a component of the National Neisseria Network of Australia and comprises participating laboratories in each state and territory (see acknowledgements). This collaborative network of laboratories obtains isolates for examination from as wide a section of the community as possible, with both public and private sector laboratories referring isolates to regional testing centres. The increasing use of non-culture based methods of diagnosis has the potential to reduce the size of the sample of isolates available for testing. Details of the number of organisms examined are thus provided in order to indicate the AGSP sample size.

Gonococci, isolated in and referred to the participating laboratories, are examined for antibiotic susceptibility to the penicillins, quinolones, spectinomycin and third generation cephalosporins, and for high level resistance to the tetracyclines by a standardised methodology previously described.[9,11] The AGSP also conducts a program specific quality assurance (QA) program.[12]

Antibiotic sensitivity data from each jurisdiction are submitted quarterly to the coordinating laboratory, which collates the results and provides individual feedback to each participating laboratory. Additionally, the AGSP collects data on the gender of the patient and site of isolation of gonococcal strains. Where available, data on the geographic source of acquisition of antibiotic resistant isolates were included in the analyses.

Results

Number of isolates

There were 3,220 gonococcal isolates referred to, or else isolated in, AGSP laboratories in 2009, little changed overall from the 3,189 examined in 2008. The source and site of infection with these isolates are shown in the Table. Nine hundred and forty-nine gonococci (29.5% of the Australian total) were isolated in New South Wales, 786 (24.4%) in Victoria, 561 (17.4%) in Queensland, 387 (12%) in the Northern Territory and 318 (9.9%) in Western Australia, 170 (5.3%) in South Australia. There were a small number of isolates from the Australian Capital Territory (38; 1.2%) and Tasmania (11; 0.3%).

Isolate numbers increased from those reported in 2008 in New South Wales (from 857), Victoria (from 567), and the Australian Capital Territory (from 9). Conversely, there was a decrease in the number of isolates from South Australia (from 391), and Western Australia (from 410), but there was little change in Queensland (from 542), the Northern Territory (from 403) and Tasmania (from 13).

Table: Source and number of gonococcal isolates, Australia, 2009, by sex, site and region

		State or territory								
Gender	Site	ACT	NSW	NT	Qld	SA	Tas	Vic	WA	Aust
Male	Urethra	16	523	238	353	86	8	412	219	1,855
	Rectal	13	193	0	48	15	2	157	9	437
	Pharynx	7	101	2	28	19	1	105	7	270
	Other/NS	1	8	13	11	8	0	11	8	60
	Total	37	825	253	440	128	11	685	243	2,622
Female	Cervix	0	100	125	116	31	0	87	71	530
	Other/NS	1	24	9	5	9	0	14	4	66
	Total	1	124	134	121	40	0	101	75	596
Unknown*	Total	0	0	0	0	2	0	0	0	2
Total		38	949	387	561	170	11	786	318	3,220

* The site of isolation and sex of some infected patients was not known.

NS Not serotyped

Source of isolates

There were 2,622 isolates from men and 596 from women, with the male to female (M:F) ratio of 4.4:1; higher than the 3.7:1 ratio for 2008. The number of isolates from men increased from 2,509 in 2008, but the number of isolates from women decreased from 682. Isolates from females increased from 2008 in New South Wales (from 117) and Victoria (from 74), but decreased in Queensland (from 139), and Western Australia (from 106) and with a marked decline in South Australia (from 104). The number of isolates from Northern Territory was essentially unchanged (137 in 2008). The M:F ratios in each jurisdiction were much the same as those reported in 2008, and remained high in New South Wales (6.7:1) and Victoria (6.8:1), where strains were more often obtained from urban populations, than in Queensland (3.6:1), Western Australia (3.2:1), South Australia (3.2:1) and the Northern Territory (1.9:1), where there is a large non-urban component of gonococcal disease. Male rectal and pharyngeal isolates were most frequently found in Victoria (together, 38% of isolates obtained from men), New South Wales (36%) and South Australia (27%). Further, the total number of isolates was small in the Australian Capital Territory (38), but it is notable that 54% were rectal or pharyngeal.

For 126 of the isolates in the Table, the site is shown as 'other' or 'not stated'. Included in this total were 27 cases of disseminated gonococcal infection; 23 in men (0.9% of all infections), and 4 (0.7%) in women. From women, 23 gonococci were pharyngeal, and there were 9 rectal isolates. Although not all infected sites were identified, isolates from urine samples were regarded as genital tract isolates and most of the other unidentified isolates were probably from this source, although they were not specified.

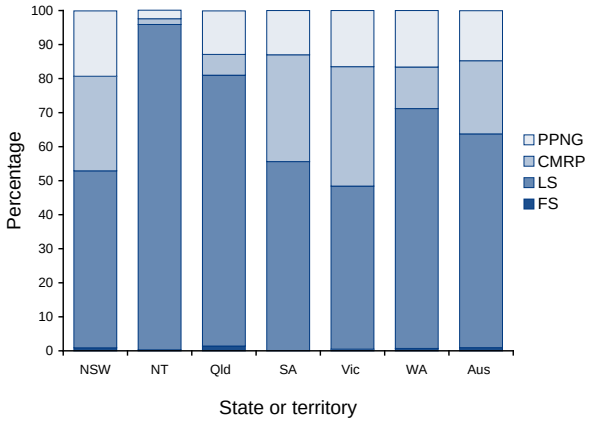
Antibiotic susceptibility patterns

Three thousand one hundred and fifty-seven (98%) gonococcal isolates in 2009 remained viable for susceptibility testing. These were examined by the AGSP reference laboratories for sensitivity to penicillin (representing this group of antibiotics), ceftriaxone (representing later generation cephalosporins), ciprofloxacin (representing quinolone antibiotics), spectinomycin; and for high level resistance to tetracycline (TRNG). As in past years, the patterns of gonococcal antibiotic susceptibility differed between the various states and territories. For this reason data are presented by region as well as aggregated for Australia as a whole.

Penicillins

The categorisation of gonococci isolated in Australia in 2009 by penicillin MIC is shown in Figure 1. Infections unlikely to respond to the penicillin group of antibiotics (penicillin, ampicillin, amoxycillin, with or without clavulanic acid) are those caused by gonococci shown as 'penicillinase producing' *N. gonorrhoeae* (PPNG) and 'relatively resistant'. Resistance in the PPNG group results from the production of beta-lactamase, and in those termed 'relatively resistant' by the aggregation of chromosomally controlled resistance mechanisms[1] – so called CMRP. Chromosomal resistance is defined by an MIC to penicillin of 1 mg/L or more.[1,11] (The MIC in mg/L is the least amount of antibiotic which inhibits *in vitro* growth under defined conditions). Infections with gonococci classified as fully sensitive (FS: MIC ≤ 0.03 mg/L) or less sensitive (LS: MIC 0.06–0.5 mg/L) would be expected to respond to standard penicillin treatments, although response to treatment may vary at different anatomical sites.

Figure 1: Penicillin resistance of gonococcal isolates, Australia, 2009, by region



FS	Fully sensitive to penicillin, MIC ≤ 0.03 mg/L
LS	Less sensitive to penicillin, MIC 0.06–0.5 mg/L
CMRP	Chromosomally mediated resistant to penicillin, MIC ≥ 1 mg/L
PPNG	Penicillinase producing *Neisseria gonorrhoeae*

Nationally, 1,145 (36%) gonococci were penicillin resistant by one or more mechanisms in 2009, a decrease in the proportion of isolates resistant to this group of antibiotics recorded in 2008 (44%) but similar to that of 2007 (38%). Of these 680 (22% of all isolates) were CMRP and 465 (15%) were PPNG, compared with 994 (32%) CMRP and 373 (12%) PPNG in 2008. The decrease in penicillin resistance nationally was predominantly due to decreased numbers of gonococci with chromosomally mediated resistance.

The proportion of penicillin resistance of all gonococcal isolates was highest in Victoria with 51.6%

(PPNG 16.5%, CMRP 35.1%), New South Wales 47.0% (PPNG 19.2%, CMRP 27.8%) and South Australia 44.4% (PPNG 13.0% and CMRP 31.4%), and although all proportions were lower than those reported in 2008, the most marked was from South Australia (from 73.2% in 2008). In Western Australia, the proportion at 28.8% (PPNG 16.6%, CMRP 12.2%) was essentially unchanged from 2008. In Queensland, the proportion of penicillin resistant gonococci again decreased, from 25% (PPNG 13.4%, CMRP 11.6%) in 2008 to 19.0% (PPNG 12.8%, CMRP 6.1%) in 2009, with the reduction being related to the proportion of CMRP isolates. Ten CMRP and 1 PPNG were identified in the Australian Capital Territory, while in Tasmania there were 3 CMRP and 2 PPNG. In the Northern Territory, there were 15 penicillin resistant gonococci, unchanged from 2008; 9 PPNG and 6 CMRP (2 from Darwin and 4 from Alice Springs) so representing a total of 4.2% of strains that were penicillin resistant in 2009 (3.9% in 2008, 4.1% in 2007, 4.6% in 2006).

Data on the country of acquisition were available for 72 (15.5%) of the infections with PPNG. Thirty-five (49%) of these were acquired locally and 37 (51%) were associated with overseas contact. These overseas contacts were principally in Western Pacific or South East Asian countries with those reported from Thailand (8), the Philippines (7) and Indonesia (5) the most numerous. Additionally, China, Vietnam, Cambodia and more widely The Netherlands and the United Kingdom were reported as countries of acquisition.

Ceftriaxone

From 2001 onwards, low numbers of isolates with raised ceftriaxone MICs have been found in Australia. This proportion has increased incrementally with data from recent years showing a rise from 0.6% in 2006; 0.8% in 2007 to 1.1% in 2008. In 2009, 64 (2.0%) gonococci were 'non-susceptible' to ceftriaxone with MICs in the range of 0.06–0.25 mg/L. Seventeen of these were present in Victoria (2.2% of isolates there); 16 in New South Wales (1.7%); 10 (1.8%) in Queensland; 9 (5.3%) in South Australia; nine in Western Australia (3.1%); 2 (5.3%) in the Australian Capital Territory; and 1 (0.3%) from the Northern Territory.

In Victoria in 2008, there were no gonococci with raised ceftriaxone MICs compared with 17 non-susceptible isolates in 2009. Sixteen of the 17 isolates were from the latter 2 quarters of the year.

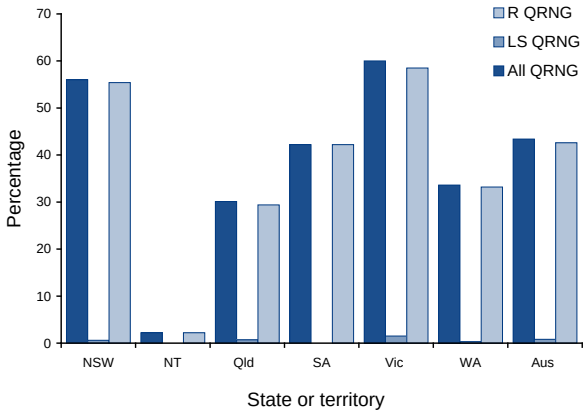
Spectinomycin

All isolates from all jurisdictions were again susceptible to this injectable antibiotic.

Quinolone antibiotics

Figure 2 shows the distribution of gonococci with altered susceptibility to quinolones nationally and by jurisdiction. Thus far, resistance to the quinolone antibiotics in *N. gonorrhoeae* is mediated only by chromosomal mechanisms so that incremental increases in MICs are observed. The AGSP uses ciprofloxacin as the representative quinolone and defines altered susceptibility as an MIC of 0.06 mg/L or more.[11] Treatment with currently recommended doses of 500 mg of ciprofloxacin is effective for strains with a lower level of resistance, viz. 0.06–0.5 mg/L, in about 90% of cases, but lower doses of the antibiotic will result in treatment failure more often. At higher levels of resistance i.e. an MIC of 1 mg/L or more, rates of treatment failure rise rapidly. At MIC levels of 4 mg/L or more, treatment failure, even with higher ciprofloxacin doses, approaches 100%.

Figure 2: Percentage of gonococcal isolates which were less sensitive to ciprofloxacin and all strains with altered quinolone susceptibility, Australia, 2009, by region



LS QRNG MIC 0.06–0.5 mg/L or with higher level ciprofloxacin resistance

R QRNG MIC 1 mg/L or more

Nationally in 2009, 1,370 (43.4%) gonococci examined had some level of resistance to quinolones (QRNG). A decrease from 1,685 (54%) detected in 2008; and 1,493, (49%) detected in 2007. Most of the QRNG found in 2009 (1,346 or 98.3%) had resistance at a higher level i.e. MICs ≥ 1 mg/L and many of these had MIC levels of the order of 8–64 mg/L. High proportions of QRNG were seen in Victoria where 469 (60.1%) of all isolates examined in this jurisdictions, were QRNG. The next highest rates were in New South Wales, 531 (56.0%); South Australia, 70 (41.4%); Western Australia, 99 (33.6%); and Queensland 167 (30.1%). In the Australian Capital Territory there were 21 (55.3%) QRNG

isolated, representing an increase from 2008 when 2 QRNG were identified. In other jurisdictions the number of QRNG remained low: Northern Territory 8; Tasmania 5.

Information on the country of acquisition of QRNG was available for 200 (14.6%) of the 1,370 cases. One hundred and thirty-six of these (68%) were acquired locally and 64 (32%) were acquired overseas from sources referred to under PPNG acquisition and with contacts additionally reported in Germany, Switzerland, Italy, South Africa, Sri Lanka, Hong Kong, the United States of America and Iran.

High level tetracycline resistance

The spread of high level tetracycline resistance in *N. gonorrhoeae* (TRNG) is examined as an epidemiological marker even though tetracyclines are not a recommended treatment for gonorrhoea and are rarely, if ever, used for treatment of gonorrhoea in Australia. Despite the lack of use of this antibiotic group, the proportion of TRNG detected continues to increase. In 2006, 12% of isolates were TRNG; increasing in 2007 (505 TRNG 16.6%) and again in 2008 (553 TRNG, 18%). In 2009, this increase continued with 650 (21%) TRNG detected.

TRNG were present in all jurisdictions, with the highest proportion in Western Australia (94 TRNG, 31%) and New South Wales (241 TRNG 25.4%). Lower proportions of TRNG were present in Victoria (148, 19.0%), Queensland (92, 16.6%) and South Australia (26, 15.4%). There were 47 (13.0% TRNG found in the Northern Territory, one in Tasmania and one in the Australian Capital Territory.

Discussion

The World Health Organization recommendations for standardised treatment regimens for gonorrhoea are based on data from epidemiological surveys of both the distribution and extent of AMR in gonococci.[1] AMR at a rate of 5% or more in gonococci sampled in a general population is the 'threshold' for removal of an antibiotic from treatment schedules and substitution with another, effective, agent.[1,13] Programs such as the AGSP seek to determine the proportion of AMR in gonococcal strains isolated in a defined patient population and relate these findings to the likely efficacy of current treatment schedules.[1,2,7,11,13] These strategies are dependant on quality AMR data, and the requirements for *in vitro* growth and AMR testing of the fastidious *N. gonorrhoeae* complicate this process. An important aspect of surveillance is to obtain and examine a sufficient and representative sample of isolates.[1,11,13] In 2009, the strains examined by the AGSP were sourced from both the public and private health sectors, constituting a comprehensive sample that meets these requirements, in spite of the increasing use of nucleic acid amplification testing for diagnosis of gonorrhoea in Australia. The AGSP distributes reference panels for use in internal quality control practice and for External Quality Assurance Schemes,[12,14] which are necessary for validation of gonococcal AMR data.

The overall number of gonococcal strains examined by the AGSP in 2009 (3,220) was essentially unchanged from 2008 (3,192), however there was a shift in proportions of the whole reported by jurisdiction with increased numbers from New South Wales, Victoria and the Australian Capital Territory and decreases from South Australia (391 in 2008 to 170 in 2009) and Western Australia (410 in 2008 to 318 in 2009).

In 2009, 36% of gonococci nationally were resistant to the penicillins, and 43% to the quinolone antibiotics. These proportions were reduced from those reported nationally in 2008 (penicillin resistance, 44%; quinolone resistance, 54%), where previously they have been increasing each year since 2003.[2] The decrease in penicillin resistance in 2009 is primarily accounted for by a reduction in CMRP rates, from 32% in 2008 to 22% in 2009. Aggregated data have shown that there is a predominant clone of CMRP coupled with high level quinolone resistance circulating with increasing frequency annually since 2003.[2,10] It is possible that the reduction in resistance to both penicillin and the quinolones in 2009 reflects a 'clonal shift' in gonococcal isolates.

In 2009, the level in Australia of gonococci isolates with high level tetracycline resistance was low but continues to rise annually despite low exposure to these antibiotics in Australia.[2] Evidence of the 'rural–urban divide',[2] in gonococcal resistance was maintained, (Figures 1 and 2) underscoring the necessity for disaggregated information rather than pooled national data to define treatment regimens appropriate for the various jurisdictions. Remote areas in some jurisdictions with high disease rates continue to be able to use penicillin based treatments, but effective use of this cheap and acceptable treatment is contingent on vigilant monitoring of resistance patterns.

Recent AGSP reports have drawn attention to the emergence and spread of gonococci in Australia that exhibit decreased susceptibility to the later generation cephalosporin antibiotics, also referred to as the extended spectrum cephalosporins (ESC). These gonococci have also been found in increasing numbers in the WHO Western Pacific Region.[15] In 'urban' Australia, the injectable agent ceftriaxone is now the standard treatment for gonorrhoea in public sector clinics, and is currently

given by intramuscular injection in a dose of 500 mg. This dose is higher than the 250 mg dose that is more commonly used throughout the Western Pacific Region,[3] however 500 mg is the smallest volume vial available in Australia. This decreased susceptibility to the ESC has been accompanied by an increasing number of reports of treatment failures with the orally administered members of this group.[3,4,16] This decreased susceptibility is quantified by the determination MIC. To date, there have been no substantiated reports of treatment failure in genital tract gonorrhoea following ceftriaxone therapy. In 2009, the number of strains with decreased susceptibility to the ESC in Australia was higher than 2008, reflecting the view that has been expressed that it is a matter of when, not if, the number of these strains will increase and that this will be accompanied by further MIC increases.

During 2009 there has been clarification of the mechanisms of resistance that are responsible for the MIC increases to ceftriaxone in gonococci. Attention has been paid particularly to the presence of 'mosaic' *penA* genes in gonococci with raised ESC MICs. *PenA* encodes penicillin binding protein 2 (PBP2), the major site of action of ceftriaxone and mosaic PBP2 are altered to reduce this activity. Additional gene polymorphisms that affect antibiotic access to the organism complement these PBP2 changes and further increase ESC MICs. Of recent interest has been an extension of a study from 2001 to 2005 on the dynamics of spread of mosaic PBP2-containing gonococci (mPBP2-GC) in Australia. This initial investigation suggested that mPBP2-GC found locally were also present in Hong Kong (where they were associated with treatment failure with an oral ESC, ceftibuten),[16] and also in Japan.[4] Continuing studies in 2007 and 2008 showed that the subtypes of the mPBP2-GC present in Australia had altered markedly and that these strains had increased as a proportion of all gonococci tested.[17]

Also of relevance have been local studies that showed other non-mosaic lesions in *penA* were also responsible for increases in ceftriaxone MICs similar to those found in mosaic PBP2 containing gonococci.[18] These lesions were single nucleotide polymorphisms that represented mutations occurring in the *penA* of *N. gonorrhoeae*. This contrasted with the mosaic *penA* alteration that results from acquisition of 'foreign' DNA by the gonococcus.[19] Despite these advances, not all the increases detected in ESC MIC levels can be explained by the molecular mechanisms described so far, and poses difficulties in developing reliable laboratory methods for the detection of ESC 'resistant' gonococci.

All gonococcal isolates tested in Australia in 2009, including those with altered cephalosporin susceptibility, were susceptible to spectinomycin. A low proportion of gonococci was also found to be resistant to azithromycin in 2009. Azithromycin has been suggested as a possible component of treatment for gonorrhoea that uses dual antibiotic treatment.[20] Resistance to azithromycin, widely used as an anti-chlamydial agent in conjunction with gonococcal treatment, has been reported with increasing frequency overseas. MIC levels in azithromycin resistant gonococci have reached very high levels in Europe, but these strains have not been detected in Australia.

The emergence and spread of antimicrobial resistance in *N. gonorrhoeae* is a global public health issue, and evolving problems of emergence and spread of resistance are complex and require attention to both disease control strategies and rational use of antibiotics.[8,21,22] Critically, both disease control strategies and the understanding of the global scope of AMR are informed by surveillance programs of AMR nationally and internationally. Continuing commitment and vigilance to surveillance of AMR in *N. gonorrhoeae* means that maintenance of culture-based systems will be required while this surveillance is still based on testing of gonococcal isolates.

Acknowledgements

The AGSP thanks the Australian Government Department of Health and Ageing for continued financial support and the many laboratories, private and public, throughout Australia for submission of isolates for testing.

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Surveillance of Creutzfeldt-Jakob disease in Australia: 2010 update

Genevieve M Klug, Alison Boyd, Amelia McGlade, Christiane Stehmann, Colin L Masters, Steven J Collins

Abstract

Surveillance of all human prion diseases in Australia has been the responsibility of the Australian National Creutzfeldt-Jakob Disease Registry (ANCJDR) on behalf of the Australian Government Department of Health and Ageing since the Registry's inception in October 1993. The ANCJDR was established in response to the identification of 4 CJD deaths in recipients of human-derived pituitary hormone. The initial brief was to perform focused surveillance for any further iatrogenic cases of CJD; however the scope of surveillance was soon expanded to include all cases of CJD occurring in Australia both prospectively and retrospectively to 1970. The activities of the ANCJDR have evolved from: routine surveillance responsibilities to detailed epidemiological analysis at both national and international levels; expert advice in relation to, and management of, infection control issues; and the provision of a number of tests to aid the diagnosis and classification of CJD in suspect cases. In this brief report, surveillance outcomes are examined with the inclusion of figures from the reporting period of 1 April 2009 to 31 March 2010 and the diagnostic services offered by the ANCJDR are outlined to provide a greater insight into this aspect of the Registry. *Commun Dis Intell* 2010;34(2):96–101.

Keywords: Australian National Creutzfeldt-Jakob Disease Registry, CJD, human-derived pituitary hormone treatment, transmissible spongiform encephalopathies

Introduction

Prion diseases, also known as transmissible spongiform encephalopathies (TSEs), occur in both humans and animals. They include Creutzfeldt-Jakob disease (CJD), Gerstmann Sträussler-Sheinker syndrome, fatal familial insomnia and variant CJD (vCJD) in humans and bovine spongiform encephalopathy in cattle, scrapie in sheep and chronic wasting disease in deer and elk. The disease manifests itself as a rapid, neurodegenerative illness and is invariably fatal. In humans, the aetiology of disease is unknown in most cases and is described as sporadic CJD. In the remaining minority of cases, disease is related to an iatrogenic exposure through medical intervention or an underlying genetic cause. The Australian National Creutzfeldt-Jakob Disease Registry (ANCJDR) investigates all cases of suspect human TSE in Australia and works actively to classify these cases according to the clinically validated criteria[1,2] as definite, probable and possible CJD cases. These classifications are based on neuropathological examination and clinical criteria.[1] A possible case classification is clinically suspected but diagnostically unsupported and therefore these cases are excluded from the following statistical analyses.

Australian National Creutzfeldt-Jakob Disease Registry surveillance update

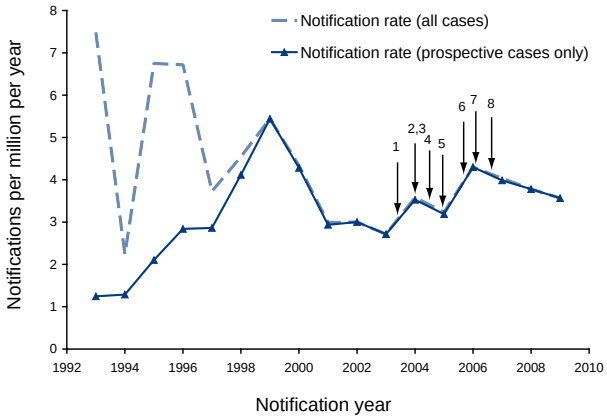
Notifications

From 1993 through to 31 March 2010, a total of 1,426 cases of suspect CJD have been notified to the ANCJDR, comprising of 309 notifications of case deaths prior to 1993 (retrospective cases) and 1,112 suspects notified prospectively. Since the last reporting period, a similar level of suspect case notification has been observed with 81 new cases referred for investigation (previous period – 90). Based on 1,090 prospective notifications for the complete calendar years between 1993 and 2009, the average annual number of notifications is 64 cases per year and the population-based referral rate is 3.2 suspect CJD cases per million per year. In the complete 2009 calendar year, the annual number of notifications has continued to be higher compared with pre-2006 levels (Figure 1). The reason for this is unclear although as speculated previously[3] it is likely to be related to increased referrals of cerebrospinal fluid (CSF) samples for 14-3-3 diagnostic testing. Of the 81 new notifications, 11 have been classified as definite CJD and nine have been removed from the registry, including seven excluded after neuropathological examination. For the remaining 61 incomplete cases, 22 are recorded as dead (11 with post-mortem examination pending) and 39 are still living. All incomplete cases are currently under investigation.

For all states and territories, the notification of prospective suspect cases has remained relatively stable compared with previous years (Figure 2), with the only exception being Tasmania where notifications have been in decline since 2006. A more recent trend is in New South Wales, where the lower number of notifications observed in 2008 has again been seen in 2009 with around 10 less

cases being notified for both years compared with the 2006–2007 period. The explanation for this does not rest with lower CSF referrals as the level of referrals for testing from New South Wales has remained consistently high over the last 3 years.

Figure 1: Annual notification rates of all suspect cases and prospective cases only, 1993 to 2009



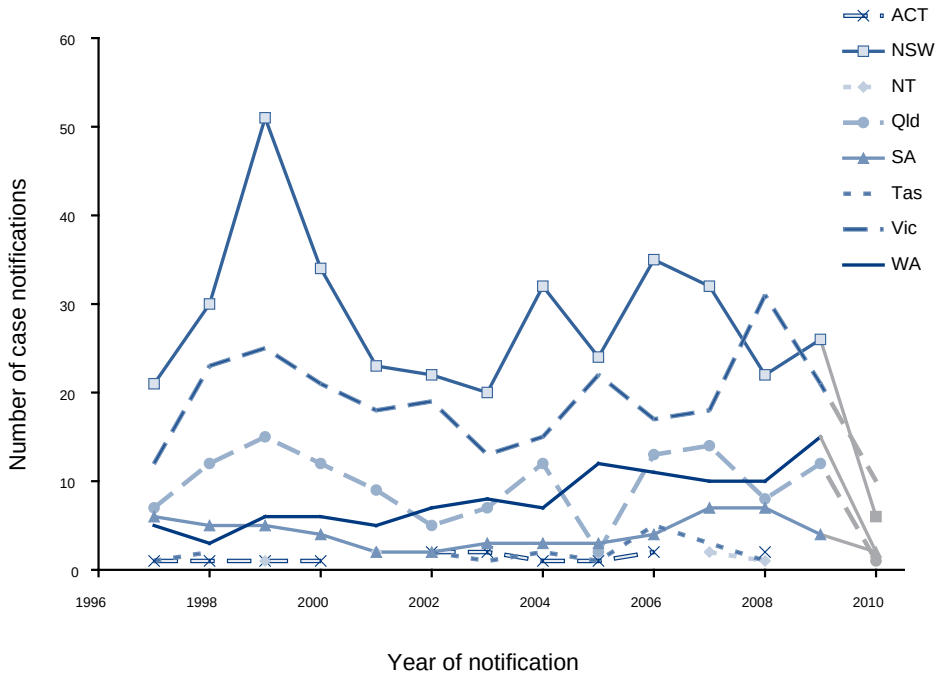
Numbers denote the point in time when Creutzfeldt-Jakob disease became notifiable in particular states and territories, 1 – Tasmania, 2 – Victoria, 3 – Western Australia, 4 – New South Wales, 5 – Northern Territory, 6 – Australian Capital Territory, 7 – Queensland, 8 – South Australia.

Case outcomes

Of the 1,426 notifications, a total of 560 suspect CJD cases have been excluded from the register with 46% of these having undergone neuropathological examination to provide an alternative diagnosis. The remaining 866 notifications are currently on the register and the large majority of these are definite and probable cases (629). A much smaller number (12) are possible cases (Table 1). Due to resource constraints, a concerning and ever-increasing number of incomplete cases grows annually and there are presently 224 incomplete cases that require investigation. This is an increase of 21% from the previous reporting period.

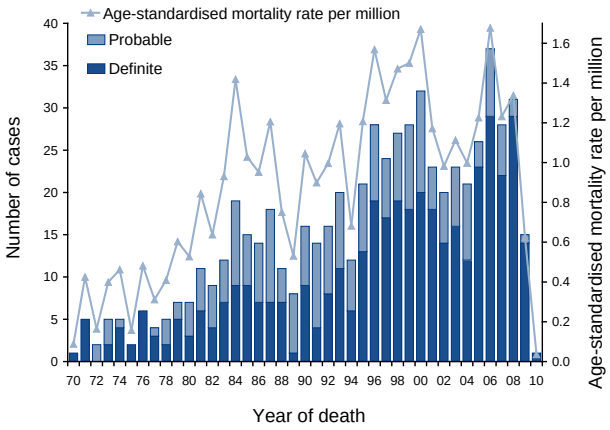
The number of definite and probable case deaths in 2009 is lower than expected; however, the current figure is provisional as several cases are awaiting case classification (Figure 3). The proportion of definite cases has increased since 2004 and this can perhaps be attributed to the ANCJDR's greater focus on assisting with autopsy planning and coordination. For the period 1993 to 2009, an average of 24.5 definite and probable CJD deaths per year has been recorded and the average age-adjusted mortality rate is 1.2 cases per million per year. These figures are consistent with previous long-term averages and with rates reported in other countries where similar surveillance systems are in

Figure 2: Prospective, suspect Creutzfeldt-Jakob disease case notifications to the Australian National Creutzfeldt-Jakob Disease Registry, 1997 to 2010, by state or territory



Grey lines denote findings for the incomplete 2010 surveillance year, which includes data to 31 March 2010 only.

Figure 3: Australian National Creutzfeldt-Jakob Disease Registry definite and probable cases 1970 to 2010,* number and age-standardised mortality rate



Age-standardised mortality rates were calculated using the Australian Bureau of Statistics 2000 estimated resident population for Australia.

* To 31 March 2010.

place.[4] This outcome provides a level of confidence that the ANCJDR is achieving and maintaining a high level of CJD surveillance in Australia.

By state and territory, fluctuations in the annual number of CJD cases have been observed, but this has resulted in only minor changes in the long-term average of the annual age-adjusted mortality rates calculated with current figures for the complete calendar years 1993–2009 (Table 2). The average mortality rate for most states and territories ranges from 0.9 in Northern Territory to 1.5 in Western Australia, and this aligns with the other national CJD rates of disease.[4] The only outlying figure is in Tasmania, which continues to have the lowest mortality in Australia with 0.7 deaths per million per year (Table 2). While this trend is of concern, an examination of the results for the more recent period of 2000–2009, does indicate that Tasmanian CJD incidence may be more consistent with other Australian states and territories. Between 1993 and 1999, there was only 1 confirmed case of CJD in Tasmania and while this skews the incidence negatively for the entire 1993–2009 period, it suggests that potential under-ascertainment of cases in this state occurred prior to 2000.

For the 629 definite and probable cases, mortality is greatest amongst the 65–69 year age group (4.9 deaths per million per year). Slightly more CJD cases are female (54%), which has been a consistent finding in Australia. While this trend is true for both sporadic (53% female) and familial (56% female) CJD groups, the small number of iatrogenic cases occur with equal gender proportions. For all Australian CJD cases, female mortality peaks in the 65–69 year age group with 5.6 deaths per million per year while men in the 70–74 year age group have the highest rate of CJD mortality (4.6 deaths per million per year).

With restriction of the data to the 1993–2010 prospective period of case ascertainment, which is considered a more comprehensive, active and co-ordinated surveillance period providing the most accurate epidemiological data, mortality peaks at a much higher level than the surveillance period of 1970–2010 (Figure 4). Peak mortality occurs in the 65–69 year age group for both genders with 6.7 and 9.0 deaths per million per year for males and females respectively. Overall, the rate in this age group is 7.9 deaths per million per year for this period, which is a 6-fold increased rate of disease compared with CJD deaths in all age groups. More generally, the age-specific mortality rates clearly demonstrate that age is a risk factor for CJD with the greatest risk for both genders being those of 65 years or greater (Figure 4). It must be noted, however, that

Table 1: Classification of cases by the Australian National Creutzfeldt-Jakob Disease Registry, 1 January 1970 to 31 March 2010

Classification	Sporadic	Familial	Iatrogenic	Variant CJD	Unclassified	Total
Definite	366	42	5*	0	0	413
Probable	203	10	4	0	0	217
Possible	11	0	1	0	0	12
Incomplete	0	0	0	0	224†	224
Total	580	52	10	0	224	866

* Includes 1 definite iatrogenic case who received pituitary hormone treatment in Australia but disease onset and death occurred while a resident of the United Kingdom. This case is not included in statistical analysis since morbidity and mortality did not occur within Australia.

† Includes 161 living cases.

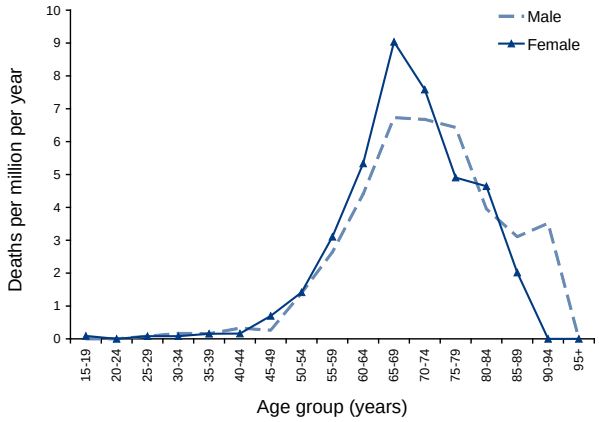
Table 2: Transmissible spongiform encephalopathy (TSE) deaths and mortality rate, by state or territory

State or territory	TSE cases by year of death											Total TSE deaths	Mean age-adjusted mortality rate (deaths/million/year)	
	00	01	02	03	04	05	06	07	08	09	10*		00–09†	93–09†
ACT			1		1		1		2			5	1.4	1.3
NSW	12	9	7	7	11	10	11	10	5	8		90	1.3	1.2
NT							2	1				3	1.0	0.9
Qld	7	3	3	3			7	2	4	2		31	0.8	1.0
SA	2			1	2		1	3	4	2		15	0.9	1.2
Tas			2			1	2					5	0.9	0.7
Vic	9	10	5	9	5	11	9	6	12	3	1	80	1.5	1.4
WA	2	1	2	3	2	4	4	6	4			28	1.3	1.5
Australia	32	23	20	23	21	26	37	28	31	15	1	257	1.2	1.2

* Provisional result for year to 31 March 2010.

† Includes all deaths occurring between the complete years 1 January 1993 or 1 January 2000 and 31 December 2009.

Figure 4: Age- and sex-specific mortality rates in all Creutzfeldt-Jakob disease cases 1993 to 2010



atypical cases do arise in both the younger and older age groups and the ANCJDR speculates that these groups have under-ascertainment, particularly those aged over 70 years.

The majority of Australian CJD cases have been classified as sporadic CJD (90.4%), whilst the remainder are familial (8.3%) and iatrogenic (1.3%) cases. Since the last reporting period, 20 new sporadic cases have been classified (16 definite, 4 probable) and 2 definite, familial cases. No cases of vCJD or further cases of iatrogenic cases have been identified. The inclusion of the 22 newly classified cases has not markedly altered the median illness duration or age at death of the 3 CJD aetiologies compared with previous reports. The median age at death occurs at 67 years in sporadic cases (males 66 years, females 67 years), 59 years in familial cases (males 51 years, females 62 years) and 42 years in iatrogenic cases (males 46.5 years, females 39 years). The median duration of illness is 4 months in sporadic cases (males 3 months, females 4 months), 6 months for familial cases (males 4.5 months, females 8 months) and 6.5 months for iatrogenic cases (males 2.5 months, females 9.5 months). Only 8% of all definite and probable CJD deaths occur under the age of 50 years and a third of these are attributable to iatrogenic or genetic CJD. The remaining cases have been investigated closely for the possibility of all forms of CJD including vCJD and after detailed follow-up, have been classified as sporadic cases.

Diagnostic functions of the Australian National Creutzfeldt-Jakob Disease Registry

One of the main operational functions of the ANCJDR is the provision of diagnostic tests to clinicians investigating a suspect case of CJD. Since 1997, the ANCJDR has offered a CSF test to detect 14-3-3 proteins in strongly clinically suspected cases in Australia and when requested, for cases in the Asia–Pacific region. The ANCJDR remains the only diagnostic laboratory offering this test in Australia. During the period from 1997 to 2010, 2,923 samples have been received and 2,570 (88%) of these have been tested. The remaining untested samples include those where testing is currently pending, unsuitable due to the sample, or where the ANCJDR has been advised by treating clinicians to not proceed with testing. An increasing number of annual referrals has been observed (Table 3) and currently the average number of CSF samples

Table 3: Referrals to the Australian National Creutzfeldt-Jakob Disease Registry for diagnostic testing 2000 to 31 March 2010*

Year	Brain tissue for immunohistochemical analysis†	Brain biopsy	Tonsil biopsy	Autopsy tissue‡ (Total annual CJD autopsies performed in Australia)	Genetic testing§	CSF testing	Total
2000	1	1		8 (34)	13	187	210
2001	4	3		19 (27)	27	209	262
2002	9			15 (26)	9	226	259
2003	2		1	14 (25)	6	237	260
2004	1			16 (22)	13	268	298
2005	1		1	21 (32)	22	276	321
2006	3	2	1	29 (36)	17	260	312
2007	1	1	2	28 (39)	13	349	394
2008		3	1	19 (39)	21	332	376
2009	1	1		28 (28)	14	335	379
2010	1	1	1	2 (8)	10	89	104
Total	24	12	7	199 (317)	165	2,768	3,175

* Referrals only. Numbers do not reflect the number of tested samples for the genetic and cerebrospinal fluid testing groups.

† Includes referrals of paraffin blocks and slide sets.

‡ Includes samples referred to the Australian National Creutzfeldt-Jakob Disease Registry annually.

§ Includes all referrals for PRNP testing and/or Codon 129 testing.

received per year is 218. The CSF test is a highly important surveillance mechanism as it is the most powerful notification tool for definite and probable CJD cases and more broadly, is the single most dominant initial notification source of all suspect cases to the Registry. Although the large majority of samples referred are not resolved as CJD, the greatest proportion of all prospective probable and definite cases have been initially referred through CSF referral. Since the test was offered in 1997, 66% of all definite and probable CJD cases have been initially notified to the ANCJDR via CSF referral. The benefit of ascertaining potential cases whilst clinical investigations are ongoing is that the Registry is able to effectively assist clinical teams in their investigations and further testing if required.

Other tests that are offered by the ANCJDR include genetic testing, to examine the prion protein gene (*PRNP*) for the presence of a mutation, tonsil biopsy testing for vCJD suspect cases and post-mortem brain-only examination. On rare occasions, brain biopsies are referred to the ANCJDR and these are tested as for tonsil biopsies with immunohistochemical and biochemical analytical techniques; however, it is to be emphasised that based on World Health Organization recommendations,[5] the ANCJDR advises against a brain biopsy examination due to the infection control implications involved with such a procedure and the possibility that biopsy testing is not as definitive as a whole brain examination. For these reasons, the ANCJDR advocates for whole brain examination after autopsy. The ANCJDR actively promotes the offered diagnostic tests to clinicians so that these options are available to all families should they wish to pursue these avenues of investigation. Table 3 outlines the number of cases referred to the ANCJDR for analysis of; brain specimens through biopsy or post-mortem; blood and DNA samples for genetic testing; and tonsil biopsies between 2000 and 2009. The Australian autopsy rate is notable with 63% of all suspect case deaths between 2000 and 2010 undergoing autopsy evaluation. This rate is also observed for the entire prospective period of 1993 to 2010.

In addition, the ANCJDR performs routine prion protein strain typing for molecular subtype classification in confirmed sporadic CJD cases, when frozen brain tissue is available for testing. Prion protein 'strains' correlate with phenotypic subtypes and thus these analyses provide a method to categorise the phenotypically heterogeneous group of sporadic CJD. Two sets of analyses are involved in this testing. Firstly, brain tissue is examined by Western blotting techniques to determine the glycoform profile of the protease-resistant prion protein, based on the size and abundance of the 2 glycosylated and 1 unglycosylated species of the protein. In addition, the Codon 129 genotype is determined by analysing DNA. A polymorphism at the Codon 129 site of the prion protein gene distinguishes cases into 3 different genotypes (MV, MM and VV). In conjunction, these tests provide a method by which sporadic

cases can be categorised into molecular subtypes that allow the ANCJDR to monitor potential geographical clustering of particular strains, and also provide a mechanism to assess sporadic cases for the possibility of vCJD and novel strain profiles.

Acknowledgements

The ANCJDR wishes to thank families, medical practitioners and associated staff for their generous support of Australian CJD surveillance. The ANCJDR also thanks Dr Handan Wand, Dr Matthew Law and Professor John Kaldor (National Centre in HIV Epidemiology and Clinical Research at the University of New South Wales) for their expert epidemiological and statistical support.

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Peer-reviewed articles

Pandemic Influenza H1N1 2009 in north Queensland – risk factors for admission in a region with a large Indigenous population

Patrick NA Harris, Rashmi Dixit, Fleur Francis, Petra G Buettner, Clinton Leahy, Bjorn Burgher, Angela Egan, Michelle Proud, Ruvinka Jayalath, Amrit Grewal, Robert E Norton

Abstract

This study describes the epidemiology of laboratory-confirmed pandemic influenza H1N1 within north Queensland, Australia. We collected data on all specimens tested for influenza (including H1N1) by polymerase chain reaction between May and August 2009 at Townsville Hospital. Patients requiring admission to hospital and a proportion of non-admitted patients had clinical characteristics recorded. Multi-variable logistic regression analysis was used to identify independent predictors for admission. Patients requiring admission were on average older, less likely to be of Aboriginal or Torres Strait Islander descent and more likely to be pregnant, female or suffer from diabetes mellitus. Oseltamivir provision was significantly higher within the Aboriginal or Torres Strait Islander patient population. However, when the relative sizes of the local Indigenous and non-Indigenous populations were considered, the relative risk of hospital admission for Indigenous people was found to be 7.9 (4.7–13.2) in comparison to non-Indigenous. *Commun Dis Intell* 2010;34(2):102–109.

Keywords: influenza A virus, H1N1 subtype; Indigenous health services; pregnancy; diabetes mellitus; Queensland

Introduction

A novel swine-origin influenza A virus (pandemic influenza H1N1 2009, herein referred to as pandemic H1N1) was first described from Mexico in April 2009.[1,2] This was associated with reports of patients requiring hospitalisation for pneumonia with an unexpected increase in mortality and a marked shift in age distribution to the 5–59 year age range. This contrasted to past epidemics of seasonal influenza whereby the greatest morbidity occurred in both those under 5 years of age and those older than 65 years.[3] By mid October 2009, more than 414,000 laboratory confirmed cases of pandemic H1N1 had been recorded worldwide and nearly 5,000 deaths reported to the World Health Organization, with these figures significantly under-representing the true totals.[4]

Early surveillance data from the pandemic in Australia indicated that the median age of patients tested for pandemic H1N1 in Western Australia was 22 years and in Victoria 21 years, again confirming a lower age distribution than that encountered in seasonal influenza epidemics.[5] However, it has been suggested that seasonal H1N1 and influenza B exhibit a tendency to infect those with a younger median age when compared with seasonal H3N2.[6] Attack rates of seasonal influenza in Australia may be as low as 1%.[7] Case fatality ratios have been modelled for seasonal influenza and range from 0.14% (attack rate of 10%) to 1.4% (attack rate of 1%).[5]

Obesity and various co-morbidities may also be risk factors for severe disease with pandemic H1N1. A small subgroup of patients requiring intensive care support was described during the early phase of the pandemic, with obesity appearing to be associated with poor outcome and death.[8] Similar studies from Australia have described the small but significant risk of respiratory failure in relatively young individuals with co-morbidities.[9] By October 2009, 183 deaths have been attributed to pandemic H1N1 in Australia with a median age of 53 years in confirmed cases who died, compared with 83 years for seasonal influenza in the period 2001–2008.[10] The burden on intensive care units in the region has also been substantial.[11]

Queensland, Australia, has a unique population mix with around 3.3% of the population being of Aboriginal and Torres Strait Islander origin, with figures of 7.2% seen in the district served by Townsville Hospital.[12] Indigenous Australians are over-represented statistically for a variety of co-morbid conditions, with an increasing contribution from chronic non-communicable disease.[13] Indigenous communities from several parts of the world appear to have been disproportionately affected by the pandemic H1N1 outbreak.[14,15] In particular, Indigenous Canadians appear to have experienced higher rates of severe H1N1.[16,17] This population is similar to the Australian Aboriginal population in that rates of chronic disease are between 1.5 and 6.9 times that of non-Indigenous Canadians.[16,18] In New Zealand, rates of notifications and hospital admissions for pandemic H1N1

are significantly higher in Maori and Pacific Islander groups compared with those of European or other ethnicities.[19]

Data are lacking on the real impact of laboratory-confirmed seasonal and pandemic H1N1 amongst Indigenous Australians. Anecdotal local clinical experience in north Queensland would suggest that this group is numerically over-represented in patients presenting with an influenza like illness (ILI). During the 1918 pandemic, Indigenous Australian populations were severely affected, with some remote communities reportedly experiencing high mortality rates.[20] Initial national estimates suggest that Indigenous Australians are approximately 10 times more likely than non-Indigenous Australians to be hospitalised with H1N1, and account for 20% of all influenza-related admissions during the initial months of the current pandemic.[10] One study estimated the relative risk for hospital admission, intensive care requirement and death as 6.6, 6.2 and 5.2 respectively for Indigenous Australians.[21]

The aim of this study was to prospectively look at all cases of influenza A (pandemic H1N1 and seasonal) confirmed at the Townsville Hospital laboratory between May and August 2009. The investigation aimed to compare admitted and non-admitted patients with confirmed H1N1 in order to identify risk factors for hospital admission, morbidity and mortality, particularly within the local Indigenous population.

Methods

This was a prospective study of all laboratory confirmed cases of influenza A who were tested at Pathology Queensland, Townsville Hospital, between April and August 2009. Ethics approval was granted by the Human Ethics Committee, Townsville Health Service District.

Location

Townsville Hospital is located within the tropical region of north Queensland. During the study period it remained the only local facility to offer on-site molecular diagnostic services for influenza. The laboratory receives specimens from hospitals and clinics over a large and diverse geographical area, covering a population of approximately 216,480.[22]

Subjects and design

All subjects who presented with an influenza-like illness between May and August 2009 and who subsequently had a respiratory specimen (nose and throat swab, endotracheal aspirate, bronchoscopic aspirate or nasopharyngeal aspirate) that tested positive for influenza A by nucleic acid amplification were included in the primary analysis. Subsequently, all admitted patients and a proportion of non-admitted H1N1 positive control patients were included in a comparative analysis. The number of non-admitted control subjects with clinical data collected was intended to match those admitted at a ratio of approximately 2:1. Data representing non-admitted patients with H1N1 were obtained from those tested and discharged from the Townsville emergency department, district hospitals (mainly Charters Towers and Palm Island) and a local general practitioner practice. Data could not be obtained for all non-admitted H1N1 positive patients, primarily as a result of the practical difficulties in accessing clinical notes from a diverse group of geographically isolated testing facilities.

Data collection

Demographic and laboratory data were collected on all patients tested. This included age, sex, indigenous status, need for admission and sub-typing of influenza A. Self-reported indigenous status is routinely collected at the time of registration in the laboratory database. For the comparative analysis, clinical data were ascertained by chart review of all admitted and a selection of non-admitted patients. The following variables were collected from both groups: pregnancy, the presence of co-morbid medical conditions (obesity, chronic lung, renal or cardiovascular disease, diabetes mellitus, malignancy or immunosuppression), commencement of antiviral agents, need for intensive care unit (ICU) admission and clinical outcome. Obesity was defined as a body mass index of above 30 kg/m^2. The term 'chronic lung disease' incorporated the diagnoses of asthma, chronic obstructive pulmonary disease and other conditions leading to significant respiratory compromise. Chronic renal failure was defined as an estimated glomerular filtration rate (GFR) of less than 60 mL per minute over a period of 3 months or longer. The definition of immunosuppression included the administration of long term systemic corticosteroids or immunosuppressive medications or HIV infection.

Laboratory diagnosis

Detection of influenza A was performed as previously described.[23] Briefly, this was a 5'-nuclease real-time polymerase chain reaction (RT-PCR) (WhSl-FluA-5N), which had been developed for the detection of influenza type A. This test utilised conserved primer and probe targets on the matrix protein genes of a broad range of influenza A subtypes, including avian influenza subtypes. Using this information, 2 primers and one 5'-nuclease probe were designed. By testing 10-fold dilutions of H1N1 and H3N2 strains, the detection limit of the WhSl-FluA-5N assay

was determined to be 1 TCID50 per millilitre for both viral types. Subsequent typing as pandemic H1N1 was performed as described by Whiley et al.[24] Two assays were used. These were H1-PCR and N1-PCR, targeting the novel influenza A (H1N1) virus haemagglutinin and neuraminidase genes, respectively.

Statistical analysis

Numerical variables were described using median values, inter-quartile ranges (IQR) and ranges, because their distribution was skewed. Only patients with complete clinical data recorded were included in the analysis. Patients admitted to hospital were compared with patients who were not admitted regarding patient characteristics using non-parametric Wilcoxon tests, Chi-square tests, Fisher's exact test, and Spearman rank correlation coefficient.

Multi-variable logistic regression analysis was used to identify independent predictors of being admitted to hospital. For this analysis, all variables were dummy coded. Age was categorised using the quartiles of the distribution. Stepwise forward and backward selection procedures were used. After a stable model was identified all remaining characteristics were considered as potential confounders. A characteristic was considered a confounder if the estimate changed by 5% or more. Results are presented as odds ratios and 95% confidence intervals. This analysis was repeated for female patients only. Throughout the analysis a significance level of 0.05 was assumed. Statistical analysis was conducted using SPSS for Windows, version 17.0 (SPSS Inc., Chicago, Illinois). We estimated the cumulative incidence of outcomes relating to hospital admissions, intensive care and death during the study period. To calculate the relative risk (RR) for both Indigenous and non-Indigenous groups we compared the cumulative incidence of the outcomes for each group with the same outcome in the total population minus the population at risk. The proportion of Indigenous people within the local population was estimated as 7.2% from Australian census data.[22]

Results

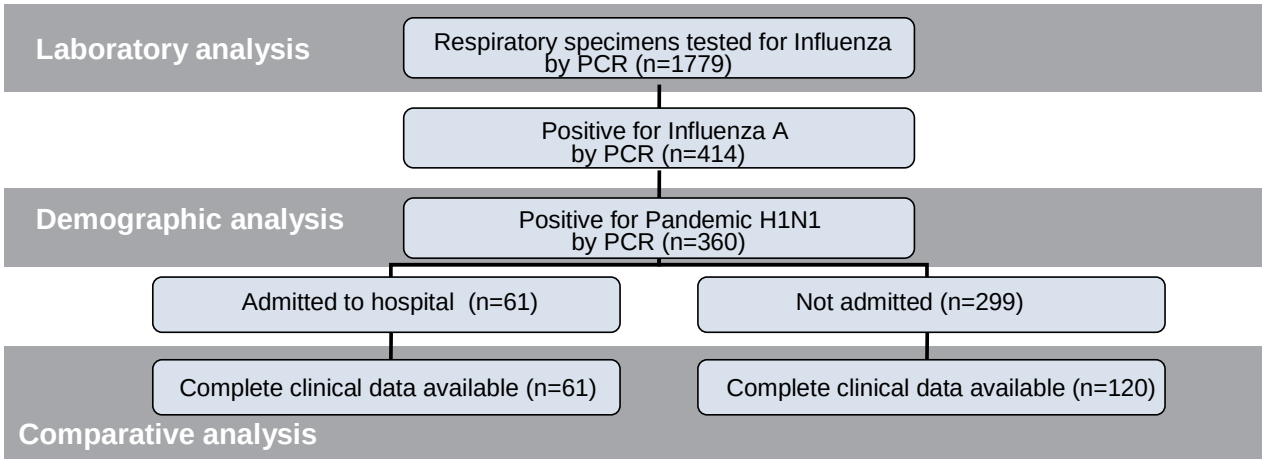
During May to August 2009, 1,779 respiratory specimens were tested for influenza A from subjects with an ILI. A total of 414 (23.3%) tested positive for influenza A by PCR. Of these, 360 (87%) were positive for pandemic H1N1. A total of 61 (17%) patients required admission to hospital with 5 deaths (1.4%). Complete clinical data were available for 181 (50.3%) H1N1 positive subjects (Figure 1).

Within the group of patients for which complete clinical data were available (n = 181), the median duration of stay in hospital was 3 days (IQR = 2 to 7 days; range 1 to 30 days). A majority (77.8%) of cases were Townsville residents, including 34 patients from Palm Island. Single cases came from Mackay, Ayr and Cairns, while 17 patients were resident in Charters Towers. The Townsville Hospital Emergency Department was the primary testing site in most cases (43.6%). The median age of recorded cases was 21 years (IQR = 9.5 to 39.5 years; range 0.2 to 90 years), 47.5% were male and 52.5% were Indigenous (Table 1).

Associations with being admitted to hospital

H1N1 positive cases admitted to hospital were on average older (median age 33 versus 15.5 years), less likely to be Indigenous (37.7% versus 60.3%), and more likely to have diabetes mellitus (24.6% versus 4.2%) compared with cases who were not admitted (Table 2). Multi-variable logistic regression analysis showed that patients with diabetes mellitus were 6.6 times more likely to be admitted to hospital than people without diabetes ($P = 0.005$) (Table 2). Patients of Indigenous descent were 0.3 times likely (that is, non-

Figure 1: Summary of study design and patient selection



Indigenous patients were 3.2 times more likely) to be admitted to hospital than non-Indigenous patients ($P = 0.003$).

Multi-variable regression analysis for female patients only, showed that pregnant women were 5.8 times more likely to be admitted to hospital compared with women who were not pregnant ($P = 0.007$) (Table 3). Women with diabetes mellitus were 9.1 times more likely to be admitted to hospital compared with women without diabetes ($P = 0.016$).

There was a linear trend towards higher admission rates for older patients (Spearman's correlation coefficient = 0.68; $P = 0.021$) (Figure 2). Of the 27 patients aged 5 years or younger, 33.3% were admitted to hospital; while of the 16 patients aged 41 to 50 years, 25% were admitted to hospital.

Indigenous patients

Indigenous subjects were significantly more likely than non-Indigenous, to have at least 1 significant clinical co-morbidity (74.4% vs. 53.8%; OR 2.501 (1.51-4.16); $P = 0.0003$), as might be expected for this population. This was particularly so with diabetes where 12% of H1N1 positive Indigenous subjects had diabetes compared with 3% of non-Indigenous (OR 4.23 (1.49-11.98); $P = 0.0045$).

Table 1: Basic characteristics overall and stratified by admission of 181 H1N1 positive cases

	Total (n=181)	Not admitted (n=120)	Admitted (n=61)	*P*-value
Median age (IQR); range (years)	21 (9.5, 39.5); range 0.2 to 90	15.5 (8.25, 26.75); range 0.75 to 61	33 (15.5, 53.0); range 0.2 to 90	*P*<0.001
% Male	47.5	46.7	49.2	*P*=0.749
% Aboriginal and Torres Strait Islander	52.5	60.3	37.7	*P*=0.004
% Townsville resident	77.8	78.2	77.0	*P*=0.866
% Females who were pregnant	15.1 (n=93)	8.1 (n=62)	29.0 (n=31)	*P*=0.013
Median gestation week of pregnant females (IQR); range (weeks)	33 (22.75, 37.25); range 12 to 40; (n=14)	32 (14, 36); range 12 to 38; (n=5)	36 (27, 38); range 22 to 40; (n=9)	*P*=0.298
% Diabetes mellitus	11.0	4.2	24.6	*P*<0.001
% Lung disease	29.8	27.5	34.4	*P*=0.336
% Renal disease	8.3	3.3	18.0	*P*=0.001
% Cardiac disease	14.4	8.3	26.2	*P*=0.001
% With malignancy	2.8	0.8	6.6	*P*=0.045
% Immunosuppressed	5.0	2.5	9.8	*P*=0.063
% Obese	16.1	10.0	28.3	*P*=0.002

Table 2: Independent factors associated with admission to hospital. Results of multi-variable logistic regression analysis based on 177* H1N1 positive cases

	Not admitted (n=116)	Admitted (n=61)	Odds-ratio (95% CI)†	*P*-value
Age	Continuous		1.02 (1.00, 1.04)	*P*=0.042
Being Aboriginal or Torres Strait Islander				
No	46	38	1	*P*=0.003
Yes	70	23	0.31 (0.14, 0.68)	
Diabetes mellitus				
No	111	46	1	
Yes	5	15	6.6 (1.8, 25.0)	*P*=0.005

* Four cases had missing values for ethnicity.

† 95% confidence interval; the model was adjusted for the confounding effects of gender.

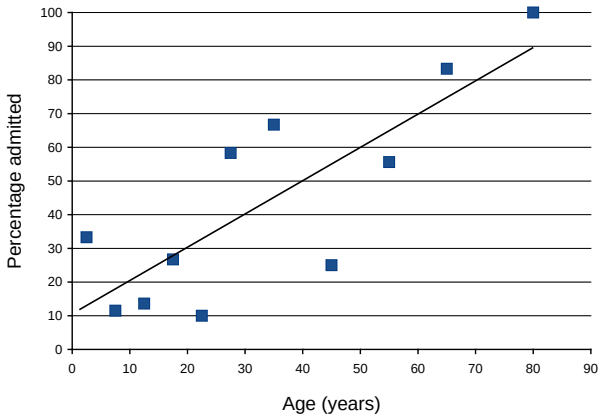
Table 3: Independent factors associated with admission to hospital. Results of multi-variable logistic regression analysis based on 93* female H1N1 positive cases

	Not admitted (n=62)	Admitted (n=31)	Odds ratio (95% CI)†	*P*-value
Being pregnant				
No	57	22	1	*P*=0.007
Yes	5	9	5.8 (1.6, 20.6)	
Diabetes mellitus				
No	59	23	1	*P*=0.016
Yes	3	8	9.1 (1.5, 55.0)	

* Two cases had missing values for being pregnant.

† 95% confidence interval; the model was adjusted for the confounding effects of age and ethnicity.

Figure 2: Association between age and percentage of cases admitted to hospital for pandemic H1N1



Of the 91 pregnant women with H1N1, 11 (12%) were Indigenous, of which four were admitted. Of the admissions to ICU, only two were Indigenous and neither of these died. There was a single death within the Indigenous group, in an elderly patient with multiple co-morbidities.

Neuraminidase inhibitor use

Oseltamivir was the only recorded anti-viral agent prescribed. Rates of oseltamivir provision were significantly greater in Indigenous patients. Seventy-nine per cent (n = 81) of non-Indigenous patients received antiviral drugs compared with 93.5% of those of Aboriginal and Torres Strait Island origin (n = 93) (OR 3.9 (1.4- 10.3); *P* = 0.005).

Outcome

Overall, 9 cases (5.0%) were treated in ICU and 8 cases (4.4%) required ventilation for a median time of 7 days (range 4–12 days). Five cases died, 2 cases required extracorporeal membrane oxygenation (ECMO), 1 case developed encephalitis, 1 pregnant woman lost her foetus but recovered herself, 2 pregnant women required a lower segment Caesarian section at term and 2 cases were still in ICU when data were retrieved. There was also one death from seasonal influenza A and *Staphylococcus aureus* pneumonia in a young non-Indigenous patient with a history of intravenous drug use.

Cumulative incidence of hospitalisation, intensive care admission and death for Indigenous populations

Estimations of the RR for hospital admission, intensive care and death for Indigenous Australians were 7.85 (4.7-13.2), 3.7 (0.8-17.8) and 3.24 (0.4-29.0) respectively in comparison with the non-Indigenous population. The wide confidence intervals for the latter 2 figures reflect the small numbers involved (Table 4).

Discussion

The current pandemic of H1N1 influenza was first reported to disproportionately affect Indigenous populations in Canada.[16] The Indigenous Australian population is similar in relation to the presence of co-morbidities such as diabetes, chronic renal, respiratory and cardiac disease. The study reported here, is the first to describe the impact of pandemic H1N1 influenza on the Indigenous population of north Queensland. The co-morbidities described conform to those expected throughout Indigenous populations in Australia. Despite making up approximately 7% of the local population, 34.7% (125/360) of all H1N1 positive specimens were from Indigenous subjects.

Patients admitted for pandemic H1N1 appeared less likely to be Indigenous in the comparative analysis. This finding appeared counter-intuitive given the well-described burden of co-morbidity within this population.[13] This result may reflect

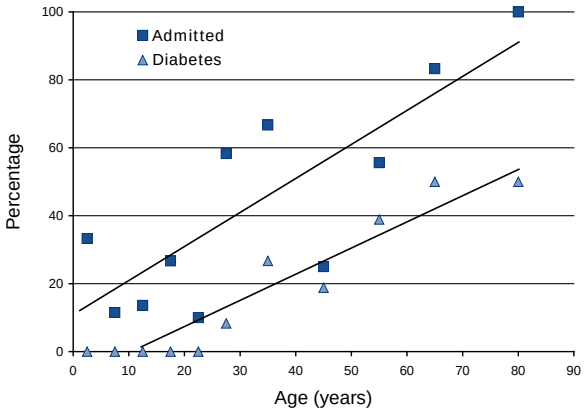
Table 4: Estimated relative risk of the cumulative incidence of hospitalisation, Intensive Care Unit (ICU) admission or death in relation to pandemic H1N1 for Aboriginal and Torres Strait Islander and non-Indigenous populations within the Townsville Health Services district

Outcome measure	Number	Population at risk	Rate per 100,000	RR	95% confidence interval
Hospitalisation total	61	216,480	28.2		
ICU admission total	9	216,480	4.2		
Death total	5	216,480	2.3		
Hospitalised Indigenous	23	15,500	148.4	7.85	4.7–13.2
Hospitalised non-Indigenous	38	200,980	18.9		
ICU Indigenous	2	15,500	12.9	3.70	0.8–17.8
ICU non-Indigenous	7	200,980	3.5		
Death Indigenous	1	15,500	6.5	3.24	0.4–29.0
Death non-Indigenous	4	200,980	2.0		

bias within the data whereby a greater proportion of Indigenous patients were represented within the non-admitted group. This could have resulted from the overuse of emergency services (the primary point of testing in most cases), the public health emphasis for testing in Indigenous populations (during the 'Protect' phase of the pandemic introduced locally on 22 June 2009[25]) and the predominance of clinical data available (and therefore inclusion in the study) from certain health services such as Palm Island. When the relative population sizes are taken into account a cumulative incidence of 148.4 per 100,000 for admission within the Aboriginal and Torres Strait Island group can be estimated and compared with a rate of 18.9 per 100,000 in the non-Aboriginal and Torres Strait Island group. These figures then translate into a significantly elevated relative risk of admission for Indigenous patients with H1N1. These results are broadly in agreement with previously published data analysed in a similar way.[21]

Despite the relatively young age of patients admitted in comparison with previous influenza seasons, increasing age remains a predictor of admission with a linear relationship seen in comparison with the proportion of those admitted. Equally, the under 5 year group were more likely to be admitted (33% admitted in this age range). In Australia, an average of 3,000 excess deaths a year may be attributable to influenza in people who are at least 50 years of age, with at least 85% of these occurring in people 65 years of age or older. Many of these would have underlying medical conditions.[26] Increasing age was associated with increasing co-morbid conditions and may account for the relationships seen (Figure 3). The presence of diabetes mellitus provided a strong predictor of requirement for admission. Diabetes has been described as occurring in up to 15% of hospitalised patients with H1N1 in the United States,[27] second

Figure 3: Relationships between age, diabetes mellitus and hospital admission for pandemic H1N1



only to asthma in frequency. Diabetes has long been thought to increase the risk of complications in seasonal influenza.[28] Whether this occurs as a product of diabetes itself, from increased risk of secondary bacterial infection, through co-existing cardiovascular mortality or via other factors remains to be clarified. There has been increasing awareness of the potential links between influenza and cardiovascular disease and cardiac death.[29]

Pregnancy was overrepresented in admitted patients with no significant difference being seen between Indigenous and non-Indigenous women. A total of 12 pregnant women required admission to hospital for H1N1 in this period (16% of all H1N1 admissions). Two of these subsequently required ICU management and, although neither died, one suffered a stillbirth. However, whether higher rates of admission in pregnancy reflect the presence of more severe respiratory disease or greater vigilance by clinicians (given the publicised concerns within this group) cannot be concluded from this study.

Whether older patients, those that were pregnant or those with diabetes developed more severe disease or were more likely to be admitted as a result of their co-morbid conditions alone is not clear. However, the pandemic response team within the hospital maintained a policy of encouraging medical staff to only admit patients with clear evidence of complicated disease (e.g. hypoxia, tachypnoea, abnormal chest signs, etc) rather than admitting due to the presence of co-morbidities alone. Patients not demonstrating these adverse clinical features were usually tested, started on oseltamivir and discharged with advice to return if symptoms deteriorated. As such, admission to hospital should remain a reasonable, although imperfect, surrogate marker of disease severity.

There were a total of 6 deaths in hospitalised patients attributable to influenza A during this period, of which five were confirmed H1N1 and one seasonal influenza A. Given that the number testing positive for H1N1 at our laboratory would grossly underestimate the total number infected in the community as a whole, the overall number of deaths and ICU admissions attributable to H1N1 was relatively low.

The early use of oseltamivir had initially been encouraged, on the basis of published papers, to reduce the duration of symptoms, transmissibility and possibly the likelihood of severe lower respiratory tract infection.[30] Oseltamivir use was significantly higher within the Aboriginal and Torres Strait Island group in comparison to non-Indigenous patients. Taken in conjunction with the relatively low rates of adverse outcomes in this group, the possibility is raised that widespread antiviral use ameliorated the anticipated impact of pandemic H1N1. However, this conclusion cannot be drawn with confidence from the data presented here.

Limitations of this study are acknowledged. Firstly only patients tested for influenza were included, but not all patients with ILI. With the progression of the pandemic, national and state protocols defined testing to be restricted to 'at risk' groups or those with severe disease manifestations. As such, numbers tested will greatly underestimate the true incidence of H1N1 within the community and 'at-risk groups' will be over-represented. However, we maintained a relatively liberal testing protocol during this time and continued to receive and process specimens from 'low-risk' individuals. Complete clinical data were not available for approximately 50% of non-admitted H1N1 positive patients. We attempted to obtain a representative sample of these, however, we cannot be certain that significant clinical differences exist between these included patients and those for whom data were not available. Furthermore this study only described the experience from a single centre, with relatively small numbers involved. Nonetheless, given these caveats, we believe that the findings presented here provide some insight into the effects of pandemic influenza H1N1 on Indigenous communities of north Queensland, especially given the paucity of accurate data in this area.

In summary, we describe the basic epidemiology of laboratory-confirmed pandemic H1N1 cases from north Queensland. Comparison with non-admitted patients with H1N1 suggested that those admitted were older, more likely to have diabetes mellitus and be non-Indigenous. Pregnancy also appeared to be strongly associated with probability of admission. However, the robustness of these findings are tempered by the possibility that significant ascertainment bias may exist within the data. Estimations of the cumulative incidence for H1N1 within the respective populations demonstrated that the relative risk for admission within the Aboriginal and Torres Strait Island group was higher (RR = 7.9 (4.7-13.2)) than for the non-Aboriginal and Torres Strait Island group. However, the overall numbers within the Aboriginal and Torres Strait Island group of ICU admission and death were small.

Acknowledgements

We declare no conflicts of interest or financial support. Many thanks to the laboratory staff at Pathology Queensland, Townsville Hospital, for the processing of large volumes of specimens and to the pandemic response team at Townsville Hospital who were instrumental in data collection.

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Evaluation of *Campylobacter* infection surveillance in Victoria

Nathan J Grills, Stacey L Rowe, Joy E Gregory, Rosemary A Lester, James E Fielding

Abstract

Campylobacter infection is a notifiable infectious disease in Victoria and with more than 6,000 cases notified annually, it is the second most commonly notified disease after chlamydia. The objectives of *Campylobacter* infection surveillance in Victoria are to monitor the epidemiology of *Campylobacter* infection, identify outbreaks, initiate control and prevention actions, educate the public in disease prevention, evaluate control and prevention measures, and plan services and priority setting. An evaluation of the system was undertaken to assess performance against its objectives, identify areas requiring improvement and inform a decision of whether *Campylobacter* infection should remain a notifiable infectious disease. The surveillance system was assessed on the attributes of data quality, timeliness, simplicity and acceptability using notifiable infectious diseases data and interviews with doctors who had failed to notify, and laboratory and public health staff. The evaluation found that the system collects core demographic data with high completeness that are appropriately reviewed, analysed and reported. In 2007, 12% of *Campylobacter* isolates were subtyped and only one to 3 outbreaks were identified annually from 2002 to 2007. Fifty-four per cent of cases were notified by doctors and 96% by laboratories, although nearly half of laboratory notifications were not received within the prescribed timeframe. Half of the surveyed non-notifying doctors thought that *Campylobacter* infection was not serious enough to warrant notification. The *Campylobacter* surveillance system is not fully satisfying its objectives. Investment in the further development of analytical methods, electronic notification and *Campylobacter* subtyping is required to improve simplicity, acceptability, timeliness and sensitivity. *Commun Dis Intell* 2010;34(2):110–115.

Keywords: *Campylobacter*, population surveillance, disease notification

Introduction

Infection with *Campylobacter* causes acute enteritis of mucopurulent and sometimes bloody diarrhoea, abdominal pain, fever, nausea, myalgia and headache.[1] Symptoms typically last for two to 5 days, but may continue for up to a week or longer. *Campylobacter* infection may be complicated by generalised sepsis, reactive arthritis and Guillian-Barré syndrome but is rarely fatal. *Campylobacter* infections are a common and significant public health issue in Australia. The notification rate of *Campylobacter* infections consistently exceeds 100 per 100,000 population,[2] and was estimated to have caused around 225,000 gastroenteritis cases annually between 2000 and 2004.[3] The morbidity and costs associated with *Campylobacter* infection are significant despite infections generally being self-limited, and it was associated with more than 3,000 hospitalisations in Australia in 2000.[4]

Campylobacter infection is notifiable in all Australian jurisdictions except New South Wales.[5] Under the *Victorian Health (Infectious Diseases) Regulations 2001,* medical practitioners and pathology services in Victoria are required to notify cases to the Victorian Department of Health within 5 days of diagnosis. The regulations also require pathology services to immediately notify the department of *Campylobacter* isolated or detected in food or water supplies. Furthermore, two or more related cases of suspected food or water-borne illness must be notified within 24 hours of diagnosis. A notified case is investigated if it meets any of the following criteria: is a food handler, health care worker or child care worker; is a child in a child care centre; is associated with one or more other cases; is a resident of a special care facility or institution; or if a suspected source of the case's illness has been reported.[6]

The objectives of *Campylobacter* surveillance in Victoria are: to monitor epidemiological trends of *Campylobacter* infection in the population; identify outbreaks and their possible or probable cause; initiate action to prevent, contain or minimise outbreaks and illness; take the opportunity to educate the public in disease prevention; evaluate control and prevention measures; and plan services and priority setting in the allocation of health resources.

Campylobacter infection is the second most commonly notified disease in Victoria (accounting for about 20% of the 2007 total) after chlamydia. There has been a generalised increase in the number of notified cases in the last 15 years from an average of approximately 2,220 cases annually between 1991 and 1993 to approximately 6,130 annually notified cases between 2004 and 2007.[6,7] Annual notification rates have increased by a similar magnitude over the same period.[2]

An evaluation of Victoria's *Campylobacter* infection surveillance was undertaken prior to a review of Victoria's public health regulations to: determine whether it was achieving its stated objectives; identify redundancies or specific areas requiring improvement; and potentially identify alternative methods for *Campylobacter* infection surveillance (such as in New South Wales for which it is only notifiable when implicated as the source of foodborne disease or gastroenteritis in an institution).[5]

Methods

The evaluation was conducted using the US Centers for Disease Control and Prevention's *Updated guidelines for evaluating public health surveillance systems*.[8] Here we report on the specific attributes of: data quality (assessed by completeness of core data fields and proportion of case isolates that are subtyped); timeliness (for laboratory notifications assessed by time in days between specimen collection date, date of result and notification receive date at the department and for doctor notifications assessed by time in days between the date of signature by the doctor and notification receive date); simplicity (assessed by method of notification); sensitivity (assessed by number of outbreaks identified from case investigation or epidemiological analysis); and acceptability (assessed by proportion of cases notified by doctors and survey of non-notifying doctors).

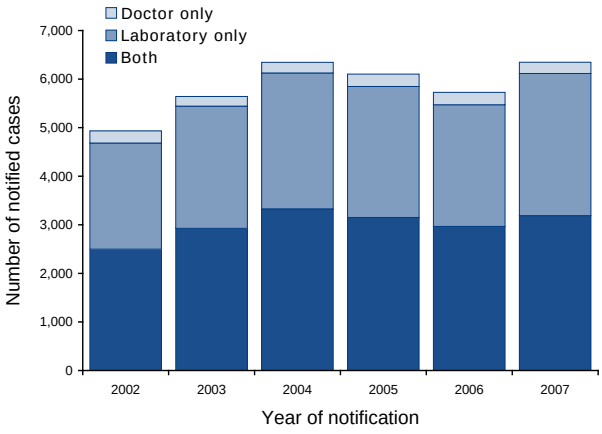
Semi-structured interviews about the system's performance against the attributes and perceptions about its usefulness were conducted with 20 key system users including laboratory staff, and head office and regional departmental public health staff that contribute to and operate the system. Records of all confirmed cases of *Campylobacter* infection with a notification receive date between 1 January 2002 and 31 December 2007 inclusive were extracted from the Victorian Government Department of Health Notifiable Infectious Diseases Surveillance database for descriptive analysis. For clarity and brevity, most figures in this paper present the most recent (2007) annual data. Using a telephone-administered survey, a sample of 30 doctors chosen randomly from a population of 270 doctors who failed to notify at least 1 case of *Campylobacter* infection in February 2008 (but were identified from a laboratory notification of the case) were asked about their awareness of *Campylobacter* infection as a notifiable disease, reasons for their failure to notify and the level of importance they would ascribe to various doctor notification improvement strategies. Survey responses were descriptively analysed using frequency tables.

EpiData software was used to collate and analyse doctors' survey data. Other descriptive analyses were conducted with Stata/IC version 10 and Microsoft Excel.

Results

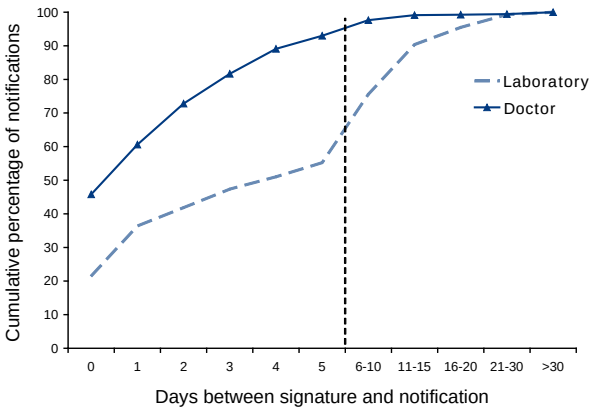
Completeness of the core data fields of date of birth, sex and residential postcode was in excess of 98% in each year from 2002–2007. However, two of the prescribed fields for medical practitioner notifications, indigenous status and occupation (which are not prescribed for laboratory notifications), were only completed for 40%–46% and 5%–14% of total cases respectively over the same time period. Of the total notified cases of *Campylobacter* infection, doctors failed to notify 44%–46% annually (Figure 1). Generally, 50%–52% of cases were notified by both a doctor and laboratory and 3%–5% by a doctor only.

Figure 1: Number of notified *Campylobacter* infection cases notified by doctors, laboratories or both, Victoria, 2002 to 2007



In 2007, 89% of laboratory notifications of *Campylobacter* infection were diagnosed within 5 days of specimen collection (median = 3 days); 55% of these notifications were received by the department within the prescribed 5 days from when the diagnosis was confirmed by the laboratory (Figure 2). However, there was significant inter-laboratory variation; 1 laboratory notified 98% of its diagnosed cases within 5 days but another only notified 3%. In comparison, nearly 93% of the *Campylobacter* infection notifications made by doctors in 2007 were within 5 days of the signature (diagnosis) date (Figure 2). The distribution of notification methods amongst laboratories and doctors reflected the time elapsed between diagnosis and notification, with nearly ¾ of laboratory notifications made by post compared with more than half of doctor notifications being made by the faster methods of fax or telephone (Table 1).

Figure 2: Days difference between diagnosis and notification of *Campylobacter* infection by laboratories and doctors, Victoria, 2007



One-third of the surveyed non-notifying doctors indicated the most important reason for not notifying cases of *Campylobacter* infection was that it was unnecessary because laboratories notify anyway (Table 2). A further 17% of doctors surveyed indicated their primary reason as being that *Campylobacter* infection is not important enough to warrant notification. Overall, half of the surveyed doctors indicated this as a reason for not notifying.

Table 1: Percentage of laboratory or doctor *Campylobacter* infection notifications, Victoria, 2007, by method

Notification method	Laboratories	Doctors
Post	72	44
Fax	26	48
Telephone	0	4
Web	0	3
Indirectly	2	1

Table 2: Most important reason reported by doctors for not notifying *Campylobacter* infection

Reason	Number	Per cent
Laboratory notifies anyway	10	33
Don't know	7	23
Not important	5	17
Too busy	4	13
System broke down	2	7
Other	2	7
Total	30	

Of factors that would encourage notification of *Campylobacter* infections, 23 of the 30 respondents noted 'a more simplified process' would encourage notification; 11 of these believed it the most important factor in increasing notifications. Cost and resource implications were acceptable to doctors, with 83% responding that 'lack of administrative and staffing support' was of little or no importance in their failure to notify.

Notifications of *Campylobacter* infection between 2002 and 2007 resulted in the identification of between one and 3 outbreaks or clusters of infection annually. In 2007, the 3 *Campylobacter* outbreaks identified were not identified from the analysis of *Campylobacter* infection notifications but through investigation of directly reported outbreaks of unspecified gastroenteritis that were subsequently found to be caused by *Campylobacter*. Although the incident case investigation system did allow some prevention and education activities in 2007 (Table 3), in general little prevention, containment or treatment activity results from notification data because the incident case is contacted subsequent to the period of *Campylobacter* infectivity.

Table 3: Outcomes of *Campylobacter* infection single incident investigations, Victoria, 2007

Outcome(s) of investigation	Number	Per cent
Exclusion from school/childcare/ work	133	10
Other cases identified	118	9
Possible source identified	285	21
Source confirmed	6	<1
Education completed	685	56
Outbreak identified	0	0
Total	1,227	

Interviews with surveillance system stakeholders quickly achieved methodological data saturation as similar issues were repeatedly identified, the most common of which were a cumbersome system for case referral and investigation, and the need for more effective feedback and dissemination to encourage more notification and better influence practice and policy. The *Campylobacter* surveillance system generates significant data that are disseminated through: regular descriptive surveillance reports to national, state and local government stakeholders; automatically (daily) generated summary data reports, and descriptive annual and quarterly reports that are posted on the web; and relevant

stakeholders being informed of an outbreak within 24 hours of its detection. However, interviews with users of the outputs of the system suggested that it is not being used to its potential for the evaluation of control and prevention measures or to influence planning, priority setting, policy, practice, research and public education.

The quality of the microbiological data available in surveillance was limited at the laboratory level. In 2007 only 12% of 6,350 *Campylobacter* isolates were speciated, of which approximately 10% were *C. jejuni* and 3 other *Campylobacter* spp. comprised the remainder (Table 4).

Table 4: Notified cases of *Campylobacter* infection, Victoria, 2007, by reported species

Species	Number	Per cent
Campylobacter not further specified	5,561	88
C. jejuni	498	8
C. jejuni jejuni	146	2
C. coli/jejuni	82	1
C. coli	41	<1
C. upsaliensis	16	<1
C. lari	6	<1
Total	6,350	

Discussion

This evaluation has found that Victoria's notifiable infectious diseases surveillance system for *Campylobacter* infection is only partially achieving its objectives. Whilst the system has a number of strengths, there are a number of improvements that could be made so that it is more effective.

Approximately half of notified cases of *Campylobacter* infection have a doctor notification. This, and results of the survey of doctors who failed to notify cases of *Campylobacter* infection, suggest that acceptability is an important weakness of the system although it should be noted that acceptability among notifying doctors was not assessed in the evaluation. Amongst the non-notifying practitioners surveyed, improved simplicity was identified as the single most important factor that would increase notification rates. Integration of electronic notification systems with practice software would improve simplicity, timeliness and quantity of doctor notifications of *Campylobacter* infection, as well as all other notifiable diseases. This is especially pertinent given the high volume of cases and that more than 90% of general practices in Australia use a clinical software package.[9] Doctors from the sample also commented that improving feedback would encourage a higher notification rate, although these need to go beyond automatically generated summary reports currently published. Other potential avenues for feedback and comparison of notification indicators include formal education sessions, information distribution through peak body organisations and other medical publications, as well as utilising improved technology to generate automatic reports to the practitioner about outcomes and investigations arising from—and thus the importance of—their notifications. Doctors should also be reminded that their notifications contain important information not provided by laboratories, such as indigenous status and risk factor data.

Results from the survey of doctors in this evaluation have informed the development of a strategy to improve medical practitioner notification rates of infectious diseases. With the support of General Practice Victoria, the strategy is focussing on: improving technology to enable notification, and; educational activities and resources that support notification.[10] A project for electronic notification of notifiable infectious diseases from pathology laboratories in Victoria to improve completeness, timeliness and overcome the practice of batching is also being developed. Imposing fines for failing to notify is an alternative method to improve notification rates and although the legislative framework allows for this, there is general agreement that this would be counterproductive in a cooperative surveillance system.

The *Campylobacter* infection surveillance system is not optimally achieving its objective of identifying outbreaks. Despite generally high completeness of core demographic data, a key barrier to achieving this objective is the low proportion of case isolates that are differentiated to species and subspecies level. This is in contrast to outbreaks of *Salmonella* infection that are identified and traced to sources through comprehensive laboratory sub-typing. If identifying outbreaks and their causes from *Campylobacter* infection surveillance is to remain an objective, then more systematic utilisation of existing and emerging technologies to subtype *Campylobacter* will be important.[11] Systematic serotyping, ribotyping and genotyping of case isolates identified from notifiable infectious diseases surveillance were used to differentiate 28% of 975 patient isolates of *Campylobacter* into 43 different clusters in Denmark[12] and 55% of 183 isolates into 29 clusters in Canada.[13] In Australia, genotyping has been used periodically to identify genotype-specific risk factors for *Campylobacter* infection and identify outbreaks or clusters of

infection.[14,15] Improved and systematic subtyping for *Campylobacter* will also make the surveillance much more effective in achieving its objectives of evaluating control and prevention measures, and planning services and priority setting in the allocation of health resources. For example, it could potentially be utilised to track the impact and effectiveness of the new Primary Production and Processing Standard for Poultry Meat, given that contaminated poultry meat is the greatest risk factor for *Campylobacter* infection in Australia.[15–17] However, the high volume of *Campylobacter* notifications means that subtyping all—or even a substantial proportion of—specimens would require significant finance and resource investment.

Sensitivity (at case level), representativeness and positive predictive value were not assessed in this evaluation. However, in Australia it has been estimated that notifiable diseases surveillance detects approximately 10% of all community cases of *Campylobacter* infection.[3] Although this is a low proportion of cases, it is difficult to determine whether or not it is representative of all cases. However, high sensitivity is not essential for a surveillance system to be representative, particularly if there are high volumes already notified. If biases amongst unidentified cases are generally consistent over time, then comparisons in *Campylobacter* infection notifications over time, person and place can still be made to monitor trends.

The low number of outbreaks detected suggests that the sensitivity of the system to detect outbreaks is poor. However, when compared with *Salmonella*, a much lower proportion of notified *Campylobacter* infection cases in Australia and the United Kingdom are associated with outbreaks. It has been suggested that the low frequency of reported *Campylobacter* outbreaks may be due to the wide distribution of source foods such as poultry and beef manifesting as disseminated community outbreaks, which are more difficult to detect than point source outbreaks (with which *Salmonella* spp. are commonly associated).[11] It therefore follows that enhancement of *Campylobacter* subtyping would improve the system's sensitivity to detect outbreaks.

In the interests of relevance to other jurisdictions, this paper has generally focused on the notification elements that feed into the *Campylobacter* infection surveillance system, rather than the subsequent investigation processes that are specific to the Victorian Government Department of Health. Briefly though, the evaluation noted that the system collects high quality data that are appropriately reviewed, analysed and reported. For example, in 2006 increased notifications of *Campylobacter* infection in the Barwon–South Western Region resulted in an investigation that linked the increase to changes in laboratory culturing methods. However, opportunities exist to make better use of algorithms and mapping technologies for geospatial analysis. Such technology could automatically check for associations of *Campylobacter* infections with, for example, population density, occupation, proximity to waterways and industry. Specific areas of improvement for the system were identified, particularly the timeliness and effectiveness of the referral process for case investigation and outbreak identification which have already been modified. The high volume of cases also periodically impacts the timeliness of data entry and thus the identification of outbreaks and emerging trends; adoption of electronic notification technology will dramatically improve this.

In summary, the *Campylobacter* infection surveillance system was found to be generating quality data for monitoring trends, and case investigation likely raises public awareness about *Campylobacter* to a limited extent. However, the system is threatened by a perceived lack of usefulness, unnecessary complexity, suboptimal timeliness, and a lack of acceptance of *Campylobacter* infection as a significant public health threat among a subset of doctors. Rectification of these issues for the system to meet its objectives requires investment in laboratory testing, more advanced analytical software and electronic notification technologies, the latter of which is in progress. The Victorian Department of Health regards *Campylobacter* infection as an infectious disease of public health importance, and improving the current system is preferred to another model such as that in New South Wales, where *Campylobacter* infection is not notifiable but which relies on direct reporting of gastroenteritis outbreaks to identify those caused by *Campylobacter*. Whilst the recommendations relate to *Campylobacter* infection surveillance, they have some cross-validity in terms of informing, more broadly, the approach to single incident investigations for other enteric diseases and surveillance for notifiable diseases in general.

Acknowledgements

The authors gratefully acknowledge the ongoing contribution of notifying medical practitioners and pathology services to notifiable infectious diseases surveillance in Victoria. We thank staff of the Communicable Disease Prevention and Control Unit's Enteric Disease Control Section, as well as Regional and Local Government environmental health officers for their hard work in undertaking *Campylobacter* infection surveillance and investigation. We also thank the medical practitioners who took part in the survey as part of this evaluation.

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Who gives pertussis to infants? Source of infection for laboratory confirmed cases less than 12 months of age during an epidemic, Sydney, 2009

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Abstract

An important approach to protecting infants against pertussis is to provide a booster vaccination to close contacts, however this strategy requires a good understanding of infection sources to be effective. The objective of this study was to identify the most important sources of transmission of pertussis infection to infants, regardless of hospitalisation status. Standardised interviews were conducted during routine follow-up calls with the parent or guardian of laboratory confirmed pertussis cases less than 12 months of age notified to 3 Sydney metropolitan public health units during a pertussis outbreak from January to May 2009. All contacts with a coughing illness or laboratory confirmed pertussis during the 3 weeks prior to onset of illness in the index case, were recorded. A source of infection could not be identified for 29 infants (31%) and a total of 86 known or suspected sources were identified for the other 66 infants. The most frequently identified sources were siblings (36%) and parents (24%), followed by other family members (21%), friends (13%), and settings outside the home such as medical centres (6%). Of 20 siblings aged 3 or 4 years, 16 (80%) were sources of infection, compared with 14 of the 44 (32%) other siblings less than 18 years of age. During this epidemic siblings were more important sources of infant infection than parents. Siblings aged 3 and 4 years of age were particularly important transmitters of pertussis infection to infants. Minimising pertussis infection in 3 and 4 year olds may be an important measure to prevent infant infection. *Commun Dis Intell* 2010;34(2):116–121.

Keywords: whooping cough, *Bordetella pertussis*, infants, source of infection, immunisation strategy

Introduction

A resurgence of reported pertussis over the last 2 decades has been documented in countries with established pertussis immunisation programs with high levels of coverage, including Australia.[1,2] Infection rates have primarily increased in those over 10 years of age, due to waning immunity, and in infants less than 5 months of age.[2,3] The current Australian immunisation schedule for pertussis consists of 3 primary doses of diphtheria-tetanus-acellular pertussis vaccine (DTPa) at 2, 4 and 6 months, followed by a booster at 4 years and a 2nd booster of adolescent formulation dTpa between 12 and 17 years of age.[4] Thus infants are not fully protected against pertussis infection for the first few months of life, during which the burden of morbidity and mortality is greatest.[5]

Parents are the most commonly identified source of transmission of pertussis to young infants, accounting for approximately half the identified sources across a range of studies in different countries, with siblings accounting for about another quarter (Table 1). To our knowledge only one of these studies[6] was not limited to only severe index infant pertussis cases that required hospitalisation or resulted in death.

The recent pertussis epidemic in Australia, and New South Wales in particular,[15] presented an opportunity to collect detailed information regarding the source of pertussis infection during a period of high community transmission. Given the importance of accurately determining the source of infection and the lack of reliable existing data sources in New South Wales, the current study was developed to systematically identify and record all possible sources of infection for laboratory confirmed cases less than 1 year of age and attribute a level of evidence to each potential source. It is also the first Australian study to include pertussis cases of varying severity, not just those requiring hospital admission. Mild cases also have an important role in sustaining high levels of transmission and we hypothesised that a careful assessment of all notified cases, regardless of hospitalisation status, may identify sources other than mothers as playing an important role in the transmission of pertussis to infants.

Methods

For the purpose of this study we included only pertussis cases less than 12 months of age confirmed by polymerase chain reaction (PCR) laboratory test. Under New South Wales protocols, source of

Table 1: Summary of studies investigating source of infant pertussis infection

Country	Year	Study population	n*	Parents† (%)	Siblings† (%)	Reference
England	1998–2000	Hospitalisations <5 months of age	33	42	27	7
Multinational‡	2003–2004	Hospitalisations <6 months of age	44	55	16	8
United States	1999–2002	Notifications <12 months of age	264	47	19	6
Australia	2001	Hospitalisations <12 months of age identified through APSU	72	53	23	9
Australia	1997–2006	Hospitalisations <12 months of age	26	52	45	10
Multinational§	2001–2004	Hospitalisations <12 months of age	24	50	17	11
United States	1990–1999	Deaths <12 months of age	46	52	41	12
Canada	1991–1997	Hospitalisations <24 months of age	431	20	53	13
France	1996–2006	Hospitalisations <6 months of age	892	55	25	14

* Number of index cases for which a source could be identified.

† Percentage of all identified sources (some index cases had more than one potential source identified in some studies whereas others only identified a 'most likely source' for each case).

‡ France, Germany, United States and Canada.

§ Brazil, Costa Rica, Germany, Singapore, Spain, Taiwan and Uruguay.

APSU Australian Paediatric Surveillance Unit

infection information is routinely collected from the parent or guardian of each case under 2 years of age. Unfortunately however, options for completing this field in the electronic Notifiable Diseases Database (NDD) are inconsistent with those provided on the paper data collection form, making data entry, extraction, and interpretation difficult.

An enhanced data collection form was developed to ensure systematic and careful collection of detailed source of infection information by public health unit (PHU) staff during routine pertussis follow-up calls to the infant's parent or guardian. A known or suspected source was defined as any person who came into contact with the case infant for greater than 1 hour in the 3 weeks prior to the onset of illness and who had a clinically consistent coughing illness (a coughing illness lasting two or more weeks; severe fits or bouts of coughing; vomiting after coughing or; 'whooping' sound during coughing) or laboratory evidence of pertussis infection. All potential sources of infection were recorded for each infant and age, sex, relationship to infant, and level of evidence of the source individual's infection (clinical symptoms, doctor diagnosed, laboratory confirmed) was collected. Further risk factors such as overseas travel or exposure through a health care worker were also ascertained.

PHUs within three of the 4 Area Health Services covering metropolitan Sydney participated in the study: Sydney South West (SSW); Sydney West (SW); and the Hornsby Office of Northern Sydney and Central Coast (NSCC). De-identified completed paper forms from participating PHUs were returned in weekly batches by secure fax to SSW, where they were assigned a study identification number and entered into an Epi Info database (Version 3.4.3, US Centers for Disease Control and Prevention, Atlanta, GA, USA). Data collection began in the last week of January 2009 and ceased in the first week of May. Data completeness for this case series was determined at the end of the study by extracting the total number of pertussis cases that met the study inclusion criteria from NDD and comparing this with the number for which the enhanced data collection form had been administered.

Results

Enhanced source of infection data was collected for a total of 95 laboratory confirmed cases notified to participating PHUs during the study period; 44 from SW, 41 from SSW, and 10 from NSCC. A total of 111 cases that met the study inclusion criteria were extracted from NDD at the conclusion of the study, therefore overall data completeness was 88%.

The median age of the cases was 3 months, with 47 males and 47 females (sex was not recorded for 1 case). The median household size, including the infant, was 5 persons (range 3–11). Twenty-four infants were too young to be vaccinated, 14 were not fully vaccinated for age, and immunisation status was unknown for a further two. Of the 56 infants who were reported to be fully immunised for age, 32 were between 2 and 5 months of age and would not have received the full 3 dose primary vaccine series. Thirty-five cases were hospitalised as a result of the

pertussis infection, all except two of which were less than 5 months of age, and hospitalisation status was recorded as unknown for a further 9 infants. The percentage of cases for which a source was identified and the percentage with siblings was similar between hospitalised and non-hospitalised cases (Table 2).

A source of infection could not be identified for 29 infants (31%) and a total of 86 known or suspected sources were identified for the other 66 infants. The most frequently identified sources were those who lived in the same household as the infant with siblings representing over double the proportion of infection sources (36%) compared with mothers (15%). Other family members (e.g. aunts, uncles, grandparents) (21%), and friends (13%) were also significant sources of infection. While a specific source individual could not be identified for 5 infants, two were potentially exposed in childcare, one attended a medical centre in which people were coughing, one was exposed in a hospital emergency department, and 1 infant most likely acquired the infection overseas. Only ¼ of the suspected source individuals were laboratory confirmed, with the majority (61%) being implicated on the basis of clinically consistent pertussis symptoms (Table 3).

Overall, 53 household sources of infection were identified (62%), and source of infection varied with age as shown in Figure 1. No clear pattern was evident, with household and non-household sources relatively evenly distributed by age. For infants that had siblings, they were the most common source, followed by infection sources that were unable to be identified. In non-sibling households, parents and other family members were most frequently identified, each contributing ⅓ of the infection sources (Table 4).

Of the 81 persons identified as potential sources of infection, 49 were children under 18 years of age. Exact age was recorded for 45 of these children of which almost half (22) were aged 3 or 4 years. Figure 2 shows the age distribution for the 30 source children who were siblings, combined with the age

Table 2: Hospitalisation status of infant pertussis cases, by age, whether a source was identified, and if the case had siblings

Infant age	Hospitalised		Not hospitalised		Unknown	
	n	%	n	%	n	%
<2 months	15	43	4	8	5	50
2–3 months	13	37	9	19	3	30
4–5 months	6	17	7	14	0	–
6–11 months	1	3	29	59	2	20
Source identified						
Yes	25	69	34	69	7	70
No	11	31	15	31	3	30
Siblings						
Yes	31	86	36	73	9	90
No	5	14	13	27	1	10

Table 3: Known or suspected sources of infection, by method of diagnosis

Source	Method of diagnosis				Total	Per cent
	Clinical symptoms	Doctor diagnosed	Laboratory confirmed	Not applicable*		
Mother	7	2	4	0	13	15
Father	7	0	1	0	8	9
Sibling	24	1	6	0	31	36
Other family	9	3	6	0	18	21
Friend	8	0	3	0	11	13
Other	0	0	0	5	5	6
Total	55	6	20	5	86	100

* Source location rather than specific individual identified, therefore the method of diagnosis of source is not applicable.

Table 4: Known or suspected sources of infection, by number of siblings in index case household

Source	No siblings		1 sibling		More than 1 sibling	
	n	%	n	%	n	%
Mother	6	20.0	2	6.5	5	9.3
Father	4	13.3	1	3.2	3	5.6
Sibling	N/A	N/A	10	32.3	21	38.9
Other family	10	33.3	5	16.1	3	5.6
Friend	3	10.0	3	9.7	5	9.3
Other setting	3	10.0	2	6.5	0	–
Source unknown	4	13.3	8	25.8	17	31.5

distribution of the other siblings in the household of the index cases who were not sources of infection (34 out of 35 for which age was recorded). While the infants in the study were more likely to have older siblings around 3 or 4 years of age, 16 of 20 (80%) siblings aged 3 or 4 years were sources of infection compared with 14 of the 44 (32%) other siblings aged less than 18 years.

Figure 1: Known or suspected sources of infection, by age of index infant case

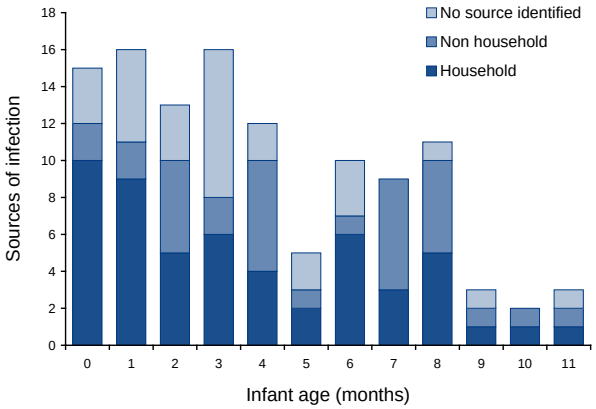
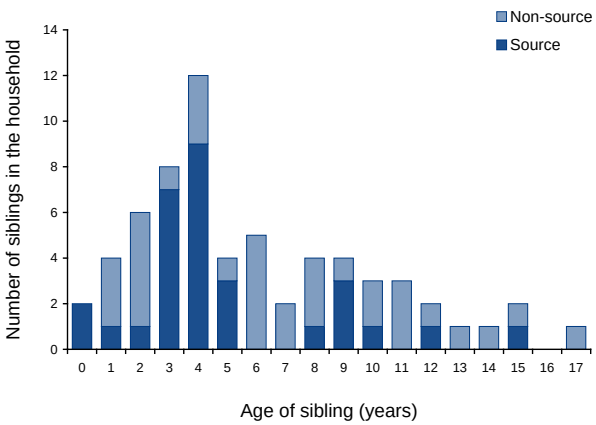


Figure 2: Age distribution of siblings less than 18 years of age resident in the index cases households



Discussion

Only 2 previous studies have investigated the source of infant pertussis infection in Australia. The first utilised the Australian Paediatric Surveillance Unit to identify 140 infants aged less than 12 months who were hospitalised for pertussis in 2001.[9] Contact with a person with a coughing illness compatible with pertussis was identified in 51% of cases. In the 72 cases where a source of infection was identified, a parent was identified as the source in 38 (53%) (mother 30, father 8) cases less than 24 weeks of age, but no parents were the source of infection for infants aged over 24 weeks. Siblings accounted for another 16 (22%) coughing contacts, with the remainder made up of grandparents, other relatives or non-family contacts. The second Australian study was a retrospective case series of 55 infants less than 12 months of age hospitalised at a tertiary paediatric hospital in Brisbane between 1997 and 2006 identified through hospital discharge coding and laboratory database.[10] A total of 31 potential sources were identified for 26 cases, of which 16 (52%) were parents and 14 (45%) were siblings. Of the 15 index cases where at least 1 parent was identified as the source of infection, 13 were under 3 months of age and all were aged under 4 months.

The present study is the first to investigate the source of infection among notified, PCR confirmed pertussis cases in Australia, including those not admitted to hospital. Importantly, interviews with parents or guardians were conducted shortly after notification and at the same time as public health investigation and follow-up to maximise the opportunity to recall coughing household members or visitors. Siblings were the most commonly identified source of infection for infants less than 12 months of age (36%

of all identified sources of infection). Parents were less frequently identified as the source of infection compared with the majority of previous studies (Table 1), with mothers representing a likely source of infection in only 15% of infants, compared with 42% and 26% in the two previous Australian studies.[9,10] Other non-household members and settings also made up a substantial proportion of infection sources, and even in households where the infant did not have siblings, only 1/3 of the infection sources were parents. The reason for the divergence observed in the present study is not entirely clear, but may reflect different transmission dynamics during the recent epidemic period, compared with studies that were conducted during periods of lower transmission. Based on the above findings, the cocoon strategy to selectively vaccinate household contacts of newborns would help to prevent some, but clearly not all, transmission to infants.

The finding that a high proportion of siblings aged 3 or 4 years were sources of infection identifies this group as an important reservoir for transmitting pertussis to infants during the outbreak. This could be a result of increased exposure to other children around this age, for example in child care settings. However, it may also be an indication that many children are not receiving the 4th dose of DTPa at 4 years of age on time. Furthermore, a dose of DTPa was previously recommended at 18 months of age, but was ceased in September 2003 due to the propensity for adverse reactions to result from this dose and it was thought the primary series provided sufficiently prolonged immunity until the booster dose at 4 years of age.[4] Elimination of the dose at 18 months may have resulted in waning immunity and an increased susceptibility to infection prior to receiving the 4th dose in the current schedule. It is of course not possible to determine this directly based on the results of the current study, but a review of the timing of the 4th dose may be required.

Infection risk also appeared to increase with household size and the number of older siblings present. The median household size in which the cases resided in this study was 5 persons. Data regarding the size and age structure limited to households with children are not reported in Australia, however the average size of households of OECD countries that do report such data are 2.7 and 3.9 for single parents and couples with children, respectively.[16] Therefore this study provides some evidence to support the particular importance that members of large households with newborn infants receive booster vaccinations.

This study was limited by the fact we were unable to identify a source of infection for 31% of the index cases. These cases may reflect a true unknown exposure, or have resulted from incomplete parental recall. However, as mentioned previously, this is a common issue in previous studies and in comparison the overall percentage of infants for which a source was identified was relatively high in the present study. This study was subject to recall bias as some parents may not have accurately remembered their infant's history of exposure to persons with a coughing illness, however, this would most likely have been minimal as interviews were conducted shortly after the infection was notified. Furthermore, only a minority of individuals suspected as sources of infection were laboratory confirmed, and the reliance on clinical symptoms to identify source cases may have missed subclinical cases or misclassified those with a coughing illness due to a pathogen other than *B. pertussis*.

The enhanced source of infection data collection form was not administered to all cases that met the criteria for inclusion in the study. However, the data completeness of 87.5% was greater than the 80% reported in the previous investigation into source of infection of notified cases in the United States of America.[6] Regardless, the review of the routinely collected data from SSW revealed that the cases not included in the study had a similar age, sex and infection source distribution to those that were. Therefore it is unlikely the exclusion of these cases would have introduced any systematic bias into the study. Finally, this study was small in comparison to some of the previous studies overseas, but similar in size to the 2 previous Australian studies. In contrast to these 2 studies, we collected data from cases with a broad range of severity over a short period of time, allowing a unique insight into pertussis transmission to infants during an epidemic. It should also be noted that PCR testing has replaced serology and culture confirmation due to its higher sensitivity, and confirmation by PCR was an inclusion criteria for the infants in this study. It not known what affect this had on the comparability to previous studies that included index cases that were confirmed by laboratory methods other than PCR testing, however there is no reason to assume it would have impacted the range of infection sources identified.

The recent pertussis epidemic in Australia, and New South Wales in particular, has underlined the necessity to reinforce control strategies, of which vaccination remains the most potent tool. The most significant shortcoming of the current pertussis immunisation schedule is that no protection is provided to infants less than 2 months of age. Universal adult vaccination would be an effective strategy to protect infants too young to be immunised, but very difficult to implement, and further safety and efficacy data are required before maternal and neonatal vaccination can be implemented.[17] Therefore the only available option at present to protect infants too young to be immunised themselves is

to encourage vaccination for those most likely to transmit infection, including not just parents, but siblings and other non-household contacts. Most importantly, our data highlight the role of siblings around 3 and 4 years of age as potential reservoirs of pertussis infection and reinforces the importance of both timely vaccination and the need to consider amending the immunisation schedule to minimise infection in this age group.

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Short reports

Serotype 6C invasive pneumococcal disease in Indigenous people in north Queensland

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A previously unrecognised serotype of *Streptococcus pneumoniae*, designated as serotype 6C, was first described in 2007.[1] Although classical serological procedures (i.e. Quellung reactions) do not distinguish this serotype from closely-related serotype 6A, it is now recognised that 6A and 6C are quite distinct and separate serotypes.[1,2]

The seven-valent pneumococcal conjugate vaccine (7vPCV) includes serotype 6B. No invasive pneumococcal disease (IPD) caused by this serotype has occurred in Indigenous people in north Queensland since 7vPCV was made freely available to Indigenous children in the latter part of 2001.[3] Although 7vPCV does not include serotype 6A, the experience in the United States of America (USA) indicates that the serotype 6B antigen included in 7vPCV provides cross-protection against serotype 6A IPD in children.[4,5] However, these studies were undertaken before serotype 6C was first recognised, and therefore serotype 6C cases may have been inadvertently included in what were then considered to have been serotype 6A IPD.

More recent studies, using methods to discriminate serotypes 6A and 6C, have revealed that although 7vPCV does indeed provide some cross-protection against serotype 6A, it does not provide protection against serotype 6C IPD.[6,7] Moreover, in the USA, the incidence of serotype 6C IPD has increased in recent years, with a concomitant increase in the proportion of serotype 6C to 6A cases of IPD.[6,8] This is probably a consequence of the widespread use of 7vPCV, as it is not only ineffective in preventing serotype 6C IPD, but it also appears to enable serotype 6C replacement carriage within the nasopharynx of young children.[7] The latter, in turn, increases the potential for transmission of serotype 6C pneumococci via respiratory droplets.

Although the number of cases was small, it initially seemed that serotype 6A had apparently become a 'prominent' cause of IPD in young Indigenous children in north Queensland following the introduction of 7vPCV.[3] However, because the Quellung reaction had been used to identify the infecting serotypes, there was no ability to distinguish any serotype 6C IPD that may have occurred and inadvertently identified as serotype 6A cases.

To determine the relevance that serotype 6C may have had since 7vPCV was introduced, all invasive serotype 6A pneumococci isolated in Queensland from 1997 to mid-2009 were re-examined using molecular methods.[2] From mid-2009 all serogroup 6 IPD isolates were routinely examined for serotype 6C using new specific antisera from Statens Serum Institut, Denmark and were also confirmed using the molecular methods.

Upon retesting, five of the IPD isolates from Indigenous people in north Queensland which were initially designated by the Quellung reaction as serotype 6A were correctly identified as serotype 6C. Three of these were in young Indigenous children included in the earlier report.[3] The earliest recognised invasive serotype 6C in an Indigenous person in north Queensland was isolated from an adult in August 2003; the earliest from an Indigenous child < 5 years of age was isolated in July 2004. A further 2 cases of serotype 6C IPD were identified through routine testing after mid-2009 (Table 1).

While the earlier results showed there were 6 serotype 6A cases in Indigenous children following the introduction of 7vPCV,[3] it is now apparent that half (3) of these were serotype 6C cases (Table 2). There is now no evidence that serotype 6A has become a more prominent cause of IPD in young Indigenous children in recent years; indeed it would seem that it is serotype 6C that has become more prominent.

A new 13-valent PCV includes serotype 6A but not 6C,[9] so that even with this vaccine, serotype 6C could become an even more prominent cause of IPD.

Table 1: Serogroup C invasive pneumococcal disease occurring in Indigenous people in north Queensland, 1999 to 2009

Serotype	1999–2001	2002–2004	2005–2007	2008–2009
6A	2	3	2	0
6B	7	0	0	0
6C	0	2	2	3

Table 2: Serotype 6A and 6C invasive pneumococcal disease occurring in Indigenous children less than 5 years of age in north Queensland, 1999 to 2007

	Serotype	1999–2001	2002–2004	2005–2007
Prior to testing for serotype 6C[3]	6A	1	3	3
Upon testing for serotype 6C	6A	1	2	1
	6C	0	1	2

Addendum

In 2010 (up to the end of May), there have been 3 cases of serotype 6C, but no serotype 6A cases of IPD in Indigenous people in north Queensland.

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An outbreak of *Salmonella* Litchfield on a car rally, Northern Territory, 2009

Polly Wallace, Martyn D Kirk, Sally A Munnoch, Jenine Gunn, Russell J Stafford, Paul M Kelly

Background

Salmonella Litchfield is a reasonably common serotype in northern Australia, but less so in southern Australia. From 2002 to 2008, non-human isolates of *S.* Litchfield were reported in 4 Australian jurisdictions and included isolates from animals (cats and dogs, farmed crocodiles and a dairy cow), foods (cucumber, millet, tree nuts, papaya and bar-ramundi) and an environmental survey of frogs, lizards and contents from vacuum cleaners around homes in Darwin (personal communication, National Enteric Pathogens Surveillance Scheme, Microbiological Diagnostic Unit, Public Health Laboratory, University of Melbourne, 14 May 2010).

A review of OzFoodNet outbreak surveillance data identified that 3 outbreaks of *S.* Litchfield have occurred in Australia since 2001. Transmission may have been foodborne in 2 instances; the 1st from contaminated papayas[1] (26 cases), and cucumbers[2] were suspected as the cause of the other. The 3rd outbreak of *S.* Litchfield was suspected to be water-borne (OzFoodNet Outbreak Register, unpublished data, 12 April 2010).

In June 2009, an outbreak of gastroenteritis occurred among participants on a Royal Flying Doctors Service (RFDS) (www.flyingdoctors.org.au) car trek in the Northern Territory. The 1st reported cases tested positive to *S.* Litchfield. The RFDS car trek is an annual event to raise funds for the organisation. Participants drive over 5,000 km across the outback in cars built prior to 1971. The trekkers contribute to the economies of the many small towns along the way. In 2009, the trek began in Grafton, New South Wales on 3 June and finished in Darwin on 14 June.

OzFoodNet conducted a multi-jurisdictional investigation to determine the source of the outbreak, the mode of transmission and to recommend appropriate public health actions in response to this outbreak.

Methods

A retrospective cohort study of RFDS trek participants was conducted. SurveyMonkey (an Internet-based survey tool) was used to create an online survey of participants based on the information provided by caterers and trek organisers. Participants provided demographic, clinical, food and other exposure information.

A case was defined as a person who developed diarrhoea (≥ 3 loose stools in 24 hours) and/or abdominal cramps and at least 1 other symptom of gastroenteritis during the trek and/or who had *S.* Litchfield isolated from a clinical specimen collected during June 2009. Other symptoms of gastroenteritis included lethargy, fever and vomiting.

All caterers who provided meals for trek participants were contacted by telephone and a standard questionnaire to determine what food items were served and to clarify food handling practices was used.

Isolates were genotyped using multi-locus variable-number tandem-repeat analysis[3] (MLVA) and pulsed-field gel electrophoresis[4] (PFGE). MLVA was performed by Queensland Health and PFGE was undertaken by the Western Australia Department of Health. The survey was available on-line for 10 days. Data analysis was performed using Stata V10.

Results

There were approximately 350 participants on the trek and contact details were provided for 82% (286/350) of participants. The response rate for the on-line survey was 50% (178/350) of all trekkers and 62% (178/286) for those participants who were able to be contacted. Seventy-six people met the case definition for *S.* Litchfield infection.

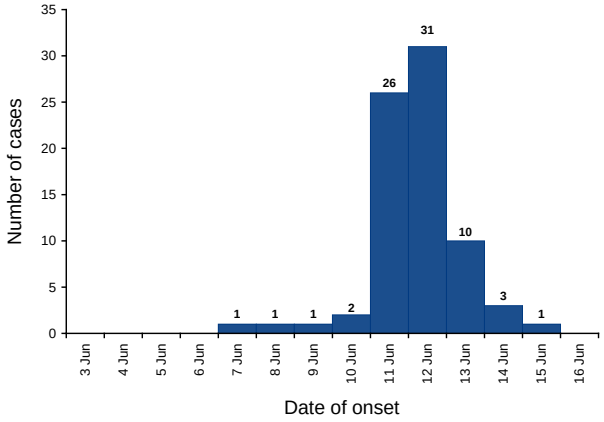
Completed surveys were received from around Australia (New South Wales 131; Victoria 15; Queensland 14; South Australia 6; Western Australia 4), Hong Kong (4), New Zealand (2) and the United Kingdom (2). The median age of respondents was 55 years (range 21–72 years) and almost all respondents were male (99% 176/178), reflecting who took part in this activity.

Among the 76 cases, 96% reported diarrhoea, 74% abdominal cramps, 74% lethargy, 55% fever and 12% vomiting. Fifty per cent of cases were ill for 6 days or more. The majority of cases became ill between 10 and 15 June 2009 (Figure). The sharp increase in the number of people becoming ill between 11 and 12 June indicates that participants were probably exposed to *S.* Litchfield in the previous 24 to 48 hours.

Univariate analysis of food exposures for preceding days showed a moderate association between illness

and consumption of barramundi fillets (attack rate 48%, relative risk 3.8, 95% CI 1.0–14.2, $P=0.007$) during dinner on 10 June. Consumption of barramundi fillets accounted for 91% (69/76) of cases. Several other food items were weakly associated with illness (including some fruits). It was not possible to calculate relative risks for some exposures, as all respondents reported eating the same foods.

Figure: Onset of illness, Royal Flying Doctor Service car trek, Grafton, New South Wales to Darwin, Northern Territory, 3 to 15 June 2009



The 5 outbreak associated case isolates of *S.* Litchfield were indistinguishable by PFGE and MLVA. The caterers were unable to provide a sample from the batch of barramundi fillet served to the trekkers. However, the seafood supplier was able to provide a sample of barramundi fillet from a different batch for testing. This sample returned a negative result for *S.* Litchfield.

Discussion with caterers providing foods for trek participants did not identify any major breaches in food safety. In addition, no ill food handlers were reported by caterers.

Discussion

This outbreak was difficult to investigate due to the remote location of the event and because our investigation began some time after the trek was completed. The geographical dispersion of participants to various states and countries immediately after the event necessitated a case finding method other than traditional methods, such as paper-based surveys or phone interviews. It was found that SurveyMonkey was a very effective tool in this investigation.

The only food exposure found to be significantly associated with illness was the pan fried barramundi served as the main ingredient of an evening meal. While 2 cases reported onsets of illness at 9.00 pm and 11.30 pm on the evening that barramundi was served, incubation periods would be considered short, but these cases could still indicate the evening meal may have been the cause of the outbreak. The 3 cases of gastrointestinal illness prior to 10 June 2009 did not provide specimens for testing, but may represent background levels of illness. However, it is difficult to exclude other meals and foods as potential vehicles of infection given that it was not possible to generate relative risks.

The barramundi sample collected as part of this investigation tested negative for *S.* Litchfield. This was not unexpected as it was not a sample from the same batch as that served on the trek. However, 2 strains of *S.* Litchfield were previously isolated from three different barramundi samples reported in 2007, 2008 and 2009 (personal communication, National Enteric Pathogens Surveillance Scheme, Microbiological Diagnostic Unit Public Health Laboratory, University of Melbourne, 14 May 2010). While the historical positive isolates from barramundi raised suspicions, it was not possible to identify the food vehicle responsible for this outbreak.

As part of this investigation, health departments also discussed food safety and hygiene with trek organisers and caterers. Food safety was difficult to assess due to the remote location of meal stops for the trek, but investigation team members were able to reinforce food and water safety with the event organisers.

This investigation highlights the effectiveness of collecting data on-line and that Internet-based surveys provide timely data collection for geographically dispersed cohorts. Previous investigations have shown Internet and email have successfully been used to collect public heath data in Australia[5–7] and overseas.[8]

On-line methods of data collection are likely be used more often as access to these technologies improves. However, this use of the Internet in gathering data from participants raised important issues of data privacy, ownership and storage, which need further discussion among public health agencies.

Acknowledgements

We would like to thank the trek organisers and participants for their help with this investigation. We acknowledge the assistance of the OzFoodNet multi-jurisdictional outbreak investigation team. We thank the following people for their technical input: Joan Powling (Microbiological Diagnostic Unit, Public Health Laboratory, University of Melbourne) who provided advice on *Salmonella* Litchfield, Lester Hiley (Public Health Reference Laboratory, Forensic and Scientific Services, Queensland Health, Brisbane) who conducted MLVA testing; Lyn O'Reilly (PathWest WA, Perth)

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Quarterly reports

OzFoodNet quarterly report, 1 January to 31 March 2010

The OzFoodNet Working Group

Introduction

The Australian Government Department of Health and Ageing established OzFoodNet in 2000 to collaborate nationally to investigate foodborne disease. OzFoodNet conducts studies on the burden of illness, co-ordinates national investigations into outbreaks of foodborne disease, develops nationally standardised protocols and tools for surveillance, identifies foods or commodities that may cause human illness and trains people to investigate foodborne illness. This quarterly report documents investigation of outbreaks of gastrointestinal illness and clusters of disease potentially related to food, occurring in Australia from 1 January to 31 March 2010.

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 months to finalise.

During the 1st quarter of 2010, OzFoodNet sites reported 306 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 illness. In total, these outbreaks affected 5,270 people, of whom 74 were hospitalised. There were 3 deaths reported during these outbreaks. The majority of outbreaks (71%, n = 216) were due to person-to-person transmission (Table 1).

Table 1: Mode of transmission for outbreaks of gastrointestinal illness reported by OzFoodNet, 1 January to 31 March 2010

Transmission mode	Number of outbreaks	Per cent of total
Foodborne and suspected foodborne	45	15
Person-to-person	216	71
Salmonella cluster	10	3
Other pathogen cluster	2	1
Unknown	33	11
Total	306	100*

* Percentages do not add up due to rounding.

Foodborne and suspected foodborne disease outbreaks

There were 45 outbreaks during this quarter where consumption of contaminated food was suspected or confirmed as the primary mode of transmission (Table 2). These outbreaks affected 271 people and resulted in 23 hospitalisations. There were no deaths reported during these outbreaks. This compares with a 5-year average of 34 foodborne outbreaks for the 1st quarter between 2005 and 2009 and 42 foodborne outbreaks during the 4th quarter of 2009.[1]

Salmonella was the aetiological agent for 22 outbreaks during this quarter, with *S.* Typhimurium being the most common serotype (n = 21). Of the remaining 23 outbreaks, three were due to *Clostridium perfringens*, two to ciguatera fish poisoning and one each to norovirus, suspected scombroid fish poisoning and *Campylobacter*. For 15 outbreaks, the aetiological agent was unknown or not specified.

Eighteen outbreaks (40%) reported in this quarter were associated with food prepared in restaurants, 9 (20%) were associated with private residences, 6 (13%) with aged care facilities, 4 (9%) with takeaway food outlets and two each (4%) with bakeries and commercial caterers. Single outbreaks (2%) were associated with commercially manufactured food, a national franchised fast food outlet and primary produce. In 1 outbreak the setting was unknown.

To investigate these outbreaks, sites conducted 7 cohort studies and collected descriptive case series data for 36 investigations, while for 2 outbreaks no individual patient data were collected. As evidence for the implicated food vehicle, investigators obtained both microbiological and analytic evidence for 2 outbreaks, relied on microbiological evidence in 3 outbreaks and analytical evidence alone for 1 outbreak. Descriptive evidence alone was obtained in 39 outbreaks.

The following jurisdictional summaries describe key outbreaks and public health actions that occurred in this quarter.

Australian Capital Territory

There was 1 reported outbreak of foodborne illness during the quarter.

Following a dinner party at a private residence, four of the 5 persons attending became unwell. Two cases were confirmed with *S.* Typhimurium phage type (PT) 170. A chocolate mousse was the suspected food vehicle.

In addition, 3 residents of the Australia Capital Territory with confirmed *S.* Typhimurium PT 9 infections, were linked to an interstate outbreak occurring at a hamburger restaurant (reported under Foodborne outbreaks, New South Wales).

New South Wales

There were 18 reported outbreaks of foodborne or suspected foodborne illness during the quarter.

Five people presented to an emergency department with symptoms of hypotension, rash, numbness and tingling, vomiting and diarrhoea, following the consumption of mahi-mahi fillets at 2 different restaurants in the same area. Mahi-mahi is a fish known to be associated with scombroid (histamine) fish poisoning and cases were treated with antihistamine. Both restaurants bought their fish from 1 supplier, who withdrew the suspected batch from sale.

Seven outbreaks of *S.* Typhimurium were investigated.

- Five of seven people who consumed a homemade seafood casserole with a raw egg mayonnaise dressing developed diarrhoea and/or vomiting. Four people were hospitalised and each case was positive for *S.* Typhimurium PT 170, multi-locus variable number of tandem repeats analysis (MLVA) 3-(9)10-7-15-523.* The farm from which the eggs used to prepare the mayonnaise were sourced was inspected and 50 samples (faecal matter, swabs, eggs) tested negative for *Salmonella*.

- An outbreak of *S.* Typhimurium MLVA type 3-(9)10-7-15-523 was reported amongst 7 people from a group of 100, who developed vomiting, diarrhoea, abdominal cramps and fever after consuming food at a wedding, prepared by a friend. Five people submitted stool samples, and all five were positive for *S.* Typhimurium, with MLVA patterns as follows: MLVA 3-10-7-15-523 (n = 3), MLVA 3-9-7-15-523 (n = 1) (1 MLVA typing outstanding). The food vehicle suspected to be the source of infection (eaten by only a few people at the function) was a tiramisu prepared with raw eggs. No environmental assessment was conducted.

- An outbreak of *S.* Typhimurium MLVA 3-9-7-13-523 occurred in a nursing home, where 2 residents developed diarrhoea due to *S.* Typhimurium infection. Both residents were on a pureed diet. No other residents in the facility were unwell and no source of infection could be identified.

- Four people from a family of five developed diarrhoea, vomiting and fever after consuming BBQ pork from an Asian takeaway. Three were admitted to hospital and their stool samples tested positive for *S.* Typhimurium MLVA type 3-11-10-9-523. Samples of raw pork, and swabs from the cutting board and the preparation bench were also positive for the *S.* Typhimurium (cutting board MLVA type 3-11-10-6-523, preparation bench MLVA type 3-11-10-9-523, and raw pork MLVA type pending). A sample of another batch of BBQ pork was negative for pathogens.

- Two of a family of four developed diarrhoea after eating a pork bun (the only common food eaten by both cases) at a Yum Cha restaurant. Stool specimens for both were positive for *S.* Typhimurium MLVA type 3-10-15-12-496. Environmental samples were all negative except for a swab taken from a table used to prepare raw pork, which was positive for *S.* Agona.

- Ten *S.* Typhimurium cases in December were investigated and four of these had identical MLVA type (3-14-8-12-523) while others had minor variations in MLVA type. This MLVA type is uncommon in New South Wales. The only link identified between three of the cases was the consumption of pork rolls: 2 cases consumed pork rolls from 1 bakery and 1 case from another bakery in a different part of Sydney. The bakery where two of the cases had eaten was inspected by the New South Wales Food Authority (NSWFA) and a number of issues were found regarding hygiene and cleanliness of the premises. The business was also producing raw egg mayonnaise and making other ingredients, such as pate, for the pork rolls on site. All food and environmental samples tested negative for Salmonella. The NSWFA issued a warning letter to the bakery on the use of raw egg foods along with an improvement notice.

- An outbreak of *S.* Typhimurium PT 9 was investigated amongst patrons of a takeaway food business in Albury. Illness was associated with the consumption of foods containing an aioli prepared with raw eggs. Interviews were con-

* Reported in Australian nomenclature used by the Institute of Clinical Pathology and Medical Research.

ducted with 206 people who ate at the outlet over a period of 6 days and 170 reported symptoms of diarrhoea and/or vomiting, fever, abdominal pain, myalgia and bloody stools, 102 were laboratory confirmed cases of *Salmonella,* with 87 of these typed as *S.* Typhimurium PT 9. This pathogen was also isolated from the aioli and from a swab taken from chopping boards. The egg farm that supplied the eggs used to prepare the aioli was inspected but no *Salmonella* was detected in environmental samples.

- During an investigation into an increase of *S.* Singapore notifications in the Hunter New England area, a small outbreak was identified amongst 5 people (three with laboratory confirmed *S.* Singapore infection) who had consumed food from a kiosk. Two cases consumed meals containing egg (salad and wrap). A traceback investigation identified that the eggs were supplied by an egg producer previously implicated in another *S.* Singapore outbreak affecting 3 people who dined at a common local restaurant.[1] An environmental investigation of the egg farm resulted in the identification of *S.* Singapore from swabs taken from the egg grading machine.

There were a further 9 reports of suspected foodborne outbreaks during the quarter that were of unknown aetiology. In New South Wales, foodborne outbreaks are often reported to the NSWFA Consumer Complaints Line by members of the public. This results in a large number of outbreaks affecting small numbers of people being referred to public health units. These outbreaks usually require limited epidemiological investigation and often the aetiology remains unknown.

Northern Territory

There were no reported outbreaks of foodborne or suspected foodborne illness during the quarter.

Queensland

There were eight reported outbreaks of foodborne or suspected foodborne illness during the quarter.

In January, 6 people reported gastrointestinal illness after a privately catered function attended by 30 to 40 people on New Year's Eve. One case (a 2-year-old female) presented to hospital with watery diarrhoea and vomiting. Norovirus genotype II was detected in a faecal specimen. Foods consumed at the gathering included salads, sausages, garlic eggs and deer meat that was cooked in a hāngi. Investigators were unable to determine the food vehicle and participants were unwilling to co-operate with the investigation.

In January, four of 5 people became ill after attending a restaurant where they consumed a range of chicken dishes with fried rice. No aetiological agent was identified.

Four outbreaks were due to *Salmonella* and all occurred in restaurant settings.

- Three cases of *S.* Typhimurium PT 170 (MLVA 1-13-3-21-3†) and 3 epidemiologically linked cases of infection were reported amongst people who had dined at a Brisbane café in January. All cases had consumed breakfast meals consisting of egg dishes only. The 3 laboratory-confirmed cases were unknown to each other and attended the café at separate times on the same day. No source of infection was identified.

- Three adults became ill after attending an Asian restaurant on the same day in January, all were confirmed to have been infected with *S.* Typhimurium PT 170 (MLVA profile 1-13-3-21-3). Meals consumed included fried rice, chicken and deep fried ice cream. *S.* Typhimurium with the same MLVA profile was detected in 2 samples of uncooked ice cream balls covered in an egg-based batter. *S.* Typhimurium was also detected in raw chicken samples collected from the restaurant. These cases were identified as part of a wider cluster investigation following an increase in *S.* Typhimurium (MLVA 1-13-3-21-3) cases in the Brisbane area.

- Four adults who attended an Asian restaurant over a 3-day period in January became ill and were confirmed to have been infected with *S.* Typhimurium PT 89 (MLVA 3-9-19-3-1). All 4 cases reported the consumption of lightly cooked egg-based calamari. *S.* Infantis was cultured in two of 6 pooled samples from 6 dozen eggs that were collected from the egg wholesaler that supplied the restaurant; however no *S.* Typhimurium was isolated. The implicated egg farm was audited by Safe Food QLD with further specimens (drag swabs, eggs, wash water etc.) collected. *S.* Typhimurium, *S.* Infantis, *S.* Mbandaka, *S.* subsp I and *S.* Orion were detected in drag swabs. *S.* Typhimurium was subtyped as MLVA 3-9-19-3-1; the same profile as the restaurant outbreak cases.

Nine cases of *S.* Typhimurium PT 89 (MLVA profile 1-5-5-2-3) were identified among people who had consumed sushi meals from a Brisbane sushi restaurant. The meals were purchased between 6 and 21 February 2010. One case was hospitalised. The food business was temporarily closed by the

† Reported in the European nomenclature used by Queensland Health Forensic and Scientific Services.

local council due to poor food hygiene and handling practices. Multiple food vehicles were associated with illness but no source of infection was identified. No *Salmonella* was detected in environmental or food samples despite extensive sampling.

Two outbreaks of ciguatera fish poisoning were reported.

- Four of 8 people became ill with ciguatera-like symptoms in January after consuming a Spanish mackerel soup. The incubation period was between four and 7 hours. All 4 cases were admitted to hospital with three of these cases treated in intensive care. The soup was made using a 2 kg mackerel fish head that was purchased from a local Brisbane fish market. Some of the remaining fish had been sold to the public; however Environmental Health were able to remove the left over fish from sale. Trace-back investigations identified that the mackerel was caught in a channel off Bribie Island.

- Six people became ill after consuming a home made fish curry using an unknown species of fish. Illness began between two and 12 hours after consuming the fish, with symptoms typical of ciguatera fish poisoning, including numbness or tingling of skin, diarrhoea, vomiting and reversed temperature sensation. The fish was purchased from a local seafood outlet in Brisbane but no trace-back could be conducted as the fish species was unknown.

South Australia

There were 2 reported outbreaks of foodborne or suspected foodborne illness during the quarter.

Following an increase in *S.* Typhimurium PT 9 notifications in January 2 separate outbreak investigations were conducted.

- Nine cases were associated with a local bakery. No specific food source was identified; however, an inspection of the food premises identified a number of issues with food handling in the bakery, which were addressed.

- Three cases of *S.* Typhimurium PT 9 were associated with a group function held at a private residence. Nine people attended the function and another ate leftovers. A cohort study was conducted to investigate the outbreak, but no specific food source could be identified.

Tasmania

Twenty-six of 43 people reported gastroenteritis after attending a 50th wedding anniversary luncheon at a restaurant in March. Consumption of a creamy chicken dish was the only food item significantly associated with illness (RR = 2.02, 95% CI 1.21 to 3.37). All faecal specimens (n = 5) and food samples tested negative for viral and bacterial pathogens and no aetiological agent could be identified. No food safety breaches were identified.

Victoria

There were 12 reported outbreaks of foodborne or suspected foodborne illness during the quarter.

Five were outbreaks of *Salmonella.*

- Six of the 14 guests at a party were confirmed with *S.* Typhimurium PT 170 and one was hospitalised. Home-made mayonnaise using raw eggs was used as a dressing for a crab salad and also as a dip. All confirmed cases ate either or both of these dishes. The host purchased the eggs used in the mayonnaise from a large supermarket chain, but could not recall the type or brand of eggs purchased.

- Analysis of surveillance data detected 3 cases of *S.* Typhimurium PT 9 associated with dining at a restaurant on a single day in January. Other groups booked at the restaurant for the same weekend were contacted, and a further 10 people reported illness with onsets from 1 to 2 days after their meal. Cases had consumed a degustation menu with approximately 10 courses. All cases had consumed an egg dish with the yolks served warm but uncooked. Eggs sampled from the restaurant tested negative for *Salmonella*. The eggs were supplied directly to the restaurant by an egg producer. All drag swabs and eggs collected from the farm also tested negative for *Salmonella*. The restaurant removed the egg dish from the menu.

- In February, analysis of surveillance data detected 2 cases of *S.* Typhimurium PT 9 linked to the same restaurant/café. Investigations revealed that 8 people from 4 separate groups who had eaten at the café on consecutive days also reported illness. Most cases had consumed dishes containing scrambled eggs, which the proprietor said were cooked for only 30 seconds. Eggs were supplied to the café by the same egg producer implicated in an outbreak of *S.* Typhimurium PT 9 in January (described above). Eggs sampled from the café and drag swabs and eggs collected from the farm all tested negative for *Salmonella*.

- A case confirmed with *Salmonella* was found to have attended a party held in a community hall in February, with 14 of approximately 90 guests reporting gastrointestinal symptoms

two to 4 days after the party. Five cases were subsequently confirmed with *S.* Typhimurium PT 141. The party was catered by a local butcher with a variety of salads, meats and desserts purchased from a bakery. The butcher was not registered with the council as a caterer. Interviews were conducted with 47 party guests, but analysis was inconclusive. *S.* Typhimurium PT 141 was detected in leftover pasta salad. Samples of raw meat and swabs of the preparation area at the home were negative for *Salmonella*.

- A general practitioner reported an outbreak of gastrointestinal illness amongst five of 6 family members. The cases developed diarrhoea and abdominal pain the day after consuming takeaway chicken and salad rolls and an egg and sausage meal at home. Stool specimens from all 5 cases tested positive for *S.* Typhimurium PT 135a. Chicken was sampled from the takeaway premises, and this was negative for *Salmonella*. The eggs consumed could not be identified as the family purchases a variety of brands of eggs from a major supermarket chain.

Three were outbreaks of *C. perfringens.*

- In February, a regional environmental health officer reported an outbreak of gastrointestinal illness in an aged care facility, with 8 of 60 residents ill with diarrhoea and/or vomiting. Meals were provided to the facility (and several other facilities) by a local hospital kitchen. No illness was reported in the other facilities. Of the 7 faecal specimens collected two were positive for *C. perfringens* enterotoxin. All ill residents had consumed roast beef the evening before onset of illness. The roast beef was cooked the day before service, cooled and then sliced. One-third of the residents consume vitamised foods. Before serving, the meat was reheated in a way that may have allowed bacteria to grow and foods were not subjected to a further heating step after vitamising. The council and the Food Safety unit addressed these issues with the facility.

- The Director of Nursing of an aged care facility reported gastrointestinal illness amongst 16 of 52 residents; all with onset of diarrhoea on the same day in March. Three faecal specimens were collected and all were positive for *C. perfringens* enterotoxin. It appears that foods (particularly meat dishes) were prepared a day before being served and cooled, stored refrigerated and then reheated prior to serving. Vitamising was conducted after the reheating step, and no further heating was conducted after this process step. Council was requested to address these issues with the facility, as well as the issue of incomplete food safety records.

- The clinical manager of an aged care facility reported gastrointestinal illness amongst 16 of 30 residents with onsets of diarrhoea clustered around one day in March. Three cases also had vomiting. Five of the 14 faecal specimens were positive for *C. perfringens* enterotoxin. Menus and food process details were requested, but insufficient information was provided to enable the source, or any issues with process steps, to be identified.

An outbreak of gastrointestinal illness was reported amongst 19 people from 35 who had attended a party in a private residence in January. Seven faecal specimens were tested and one was positive for *Shigella.* The remainder were negative for bacterial and viral pathogens. The *Shigella* case was the party host, who prepared all the food for the party. Several leftover foods were sampled and all were negative for bacterial pathogens.

An outbreak of diarrhoea and/or vomiting occurred amongst residents, visitors and staff of an aged care facility who had attended a lunch at the facility in January. Nine out of the 12 people attending the lunch reported illness. Four attendees agreed to be interviewed, with three providing faecal specimens. One faecal specimen tested positive for *Shigella* and another for *Plesiomonas*, while the 3rd was negative for bacterial and viral pathogens. Tuna sandwiches were served at the lunch and samples collected were negative for both *Shigella* and *Plesiomonas.*

An aged care facility reported an outbreak of gastroenteritis amongst 6 residents in February, with three of 5 specimens testing positive for *Campylobacter jejuni*. Onset of illness was over a 7-day period. Food histories for 5 symptomatic residents were examined along with menus provided by the facility, but the source of the outbreak could not be identified.

In February, an aged care facility manager notified illness in 4 of 107 residents with onset of diarrhoea on the same day. One faecal specimen was collected and this was negative for bacterial and viral pathogens. Several foods were sampled and all were negative for pathogens, however the presence of coliforms in some foods indicated post-process contamination. Council was requested to follow-up with the premises regarding issues with food process steps, particularly preparation of food the day before service, as well as cooling, cold storage and reheating steps.

Western Australia

There were 3 outbreaks reported during the quarter that were considered foodborne or suspected foodborne. One of these outbreaks occurred in December but was investigated in January.

Analysis of surveillance data in January revealed that 7 cases of gastrointestinal illness with onset dates over an 18-day period in December, had all eaten food from a metropolitan restaurant. Six cases were diagnosed with *S.* Typhimurium PT 170 (pulsed field gel electrophoresis [PFGE] type 0011). The other case had an illness consistent with *Salmonella* infection. Four cases had eaten scrambled eggs, two had pan fried fish and the last case could not recall what they had eaten. This restaurant was previously associated with an outbreak of the same strain of *Salmonella* in October 2009.[1] The eggs used by the restaurant in October were from the same farm as those used in December. No food was collected during the 2nd outbreak and environmental swabs of the food business were negative for *Salmonella*. In response to this 2nd outbreak the restaurant changed to a different egg supplier.

Twenty-five people became ill in January and at the beginning of February after eating food from a particular restaurant. Onset dates were spread over 21 days. Eighteen cases were diagnosed with *S.* Typhimurium PT 170 (PFGE type 0011) and the remaining 7 cases had an illness consistent with *Salmonella* infection. An aioli was consumed with a variety of foods by 22 of the cases and 2 cases had eaten Caesar salad. Both contained raw eggs. Samples of aioli and a red curry mayonnaise were positive for the outbreak strain but eggs and other sauces including Caesar salad dressing were negative for *Salmonella*. An environmental investigation showed that raw egg products were not stored adequately and batches were used over a long period of time. The eggs used by this restaurant were from the same egg farm that was implicated in 2 previous outbreaks of this *S.* Typhimurium strain in 2009[1] and 1 outbreak investigated in January 2010 (described above). Eggs and drag swabs from this farm were negative for *Salmonella*. In response to the outbreak, the restaurant started using pasteurised eggs for sauces and changed its egg supplier. Information on the risks of using raw eggs in mayonnaise and other products was posted on the Western Australia Department of Health web site and distributed to local government.

In March, an outbreak of gastrointestinal illness of unknown aetiology occurred amongst 11 of 12 tourists and their local guide on the day the group arrived from Japan. The group's driver and Japanese guide were not affected. There were no reports of prior illness among tourists or guides. Food eaten by the group included karaage chicken, rice balls and pickles purchased hot from a Japanese restaurant and served for lunch 12 hours later. No left over food was available for testing and no stool samples were collected. This outbreak was a suspected toxin-mediated illness resulting from inappropriate storage of food.

Multi-jurisdictional outbreak investigations

Hepatitis A

A previously reported multi-jurisdictional investigation into an outbreak of hepatitis A was stood down on 18 March 2010, with reports of new locally-acquired cases of hepatitis A decreasing from early November 2010. This outbreak led to a large increase in the number of hepatitis A cases reported in Australia in 2009, and the 2 peaks of the outbreak can clearly be seen on an epidemic curve (Figure.)

In the 2nd wave of the outbreak, from 29 June 2009 to 18 March 2010, there were 272 cases of hepatitis A in Australia that were known to have been locally acquired, with 119 of these cases reporting consumption of semi-dried tomatoes (44%). Nearly half of these cases (46%, 125/272) were known to have been hospitalised. The majority of cases (71%, 192/272) were from Victoria, where a 2nd case-control study was commenced in October 2009. Univariate analyses showed a significant association between consumption of semi-dried tomatoes and hepatitis A illness (OR = 10.3; 95% CI 4.7–22.7). There were 14 cases in Victoria who had close household contact with a confirmed case, and a further 8 cases who had casual contact with a confirmed case, indicating that at least 22 Victorian cases were likely due to secondary transmission.

Cluster investigations

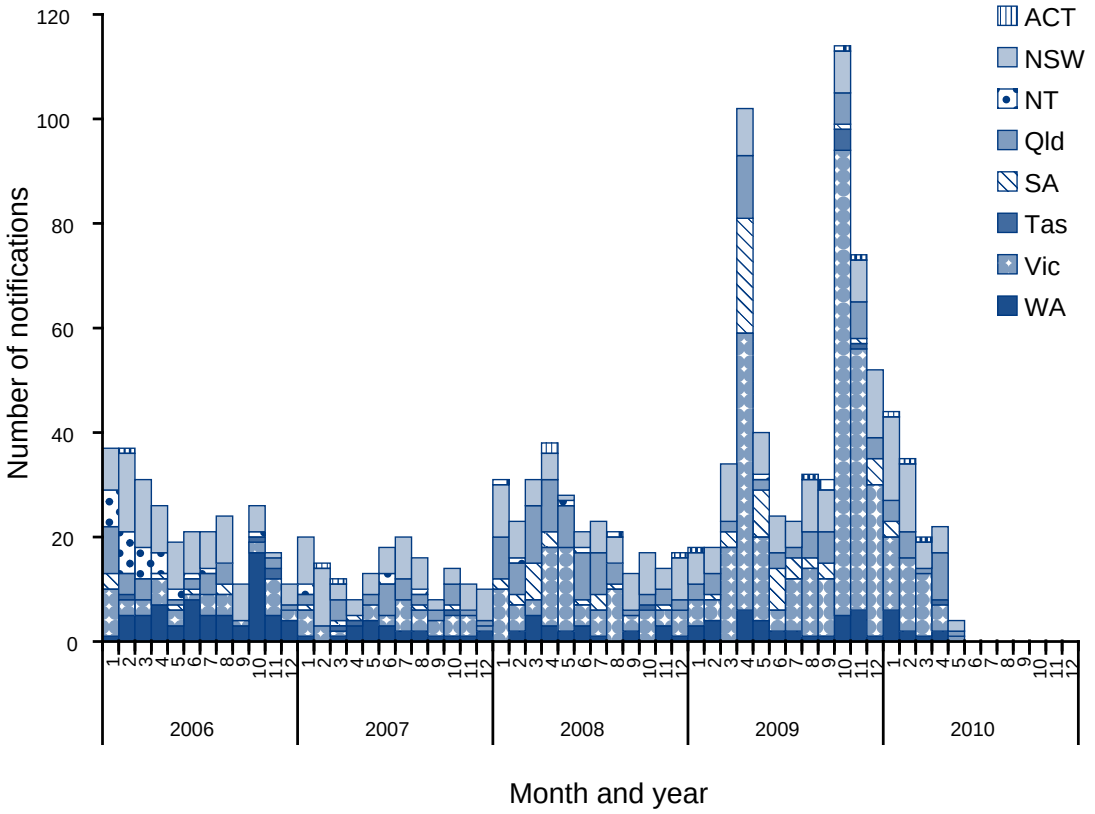
During the quarter, OzFoodNet sites conducted investigations into a number of clusters of infection for which no common food vehicle or source of infection could be identified. Clusters investigated included Shiga toxin producing *Escherichia coli* (STEC) serogroup O157 and shigellosis. A number of *Salmonella* serotypes were also investigated; *S.* Hvittingfoss, *S.* Infantis, *S.* Wangata, *S.* Potsdam, *S.* Montevideo, *S.* Bredeney and *S.* Saintpaul and *S.* Typhimurium (PT 9 and PT 135a).

Comments

The number of foodborne outbreaks reported during the quarter (n = 45) exceeded the average number during the same quarter over the past 5 years (n = 34). This increase in the number of foodborne outbreaks coincided with a general increase in the number of notifications of salmonellosis to the Nationally Notifiable Diseases Surveillance System (NNDSS), with 4,038 notifications of salmonellosis during the quarter compared with a mean of 3,106 notifications for the same period over the past 5 years.

Establishing relatedness between isolates of a particular pathogen using methods such as MLVA

Figure: Epidemic curve of hepatitis A infections, Australia, 1 January 2006 to 20 May 2010, Nationally Notifiable Diseases Surveillance System, by month and year of diagnosis



and PFGE is an essential component of foodborne disease surveillance. For inter-jurisdictional identification of clusters, the availability of a particular test across the jurisdictions, consistent methodology and nomenclature for the chosen method is essential. OzFoodNet continues to work with public health laboratory staff and the Public Health Laboratory Network of Australia towards achieving this. NNDSS and most jurisdictional notification systems in Australia have a limited capacity to store typing information beyond serotype and phage type. The capacity of these systems needs to be evaluated, and improved where possible.

Sharing information about the multi-jurisdictional outbreak of hepatitis A in Australia was vital to investigators finding the source of infection for outbreaks occurring overseas. The information provided through a notification under the WHO *International Health Regulations* (2005), via the WHO International Food Safety Authorities Network and the European Centre for Disease Control prompted the Euro virology network to compare sequences and identify a related cluster in the Netherlands. The sequences of the Australian outbreak strain and the cluster in the Netherlands were found to be identical.[2] The sequence of the hepatitis A virus from the outbreak in France was similar but not the same as the virus from The Netherlands and Australian outbreaks.[2] In an outbreak in France, investigators were alerted to the possibility of an epidemiological link with semi-dried tomatoes. Case-control studies identified semi-dried tomatoes as the source of infection in both countries.[2–3]

Outbreaks of foodborne disease associated with eggs are of continuing concern in Australia. During the quarter, 24% (11/45) of outbreaks of foodborne illness were suspected or confirmed to have been associated with the consumption of eggs. In particular, a high risk practice identified this quarter from the food service sector was the use of mayonnaise and aioli that has been prepared on one day for use over subsequent days.

During the quarter, OzFoodNet provided epidemiological support to the investigation of a non-microbial food incident—thyroid dysfunction associated with the consumption of a soy milk product that contained seaweed. In December 2009, clinicians at a New South Wales hospital investigated a case of neonatal hypothyroidism, which was suspected to be linked to the mother's high levels of consumption of a seaweed-enriched soy milk product. The hospital laboratory found that the soy milk product contained very high levels of iodine. An endocrinologist in New

South Wales subsequently reviewed adult cases of thyroid dysfunction (hyperthyroidism and hypothyroidism) in their practice, and found that there were 9 cases that were suspected to be associated with consumption of the same product, and the cluster was reported to local public health authorities. Further testing on the soy milk product was conducted at an accredited laboratory and confirmed the product contained very high levels of iodine, with tolerable daily iodine intake levels for an adult likely to have been exceeded with the consumption of as little as 30 mL per day.[4] The product was subsequently voluntarily recalled by the Australian importer. Between 23 December 2009 and 31 March 2010, 40 cases of thyroid dysfunction suspected to be associated with the consumption of the recalled soy milk product were reported to the national database.

OzFoodNet conducted an outbreak debrief of response to the multi-jurisdictional outbreak of listeriosis that occurred in 2009.[5] Several recommendations were made to improve co-ordination of outbreaks and surveillance for listeriosis. A key recommendation was to develop a surveillance plan for listeriosis, under which all *Listeria* isolates from human cases would be initially typed by molecular serotype and binary gene type and all epidemiological data would be collected in a national database. Weekly review of subtyping data would enable rapid identification of clusters and collecting all epidemiological data in a central database would facilitate rapid case–case analyses of potential clusters. Definitive sub-typing methods would be used to provide further evidence for the cluster.

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

Acknowledgements

OzFoodNet thanks the investigators in the public health units and state and territory departments of health, as well as public health laboratories and local government environmental health officers who provided data used in this report. We would also like to thank laboratories conducting serotyping, molecular typing and phage typing of *Salmonella* and other pathogens for their continuing work during this quarter.

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Table 2: Outbreaks of foodborne disease reported by OzFoodNet sites,* 1 January to 31 March 2010 (n = 45)

State	Month of outbreak	Setting prepared	Agent	Number affected	Hospitalised	Evidence	Responsible vehicles
ACT	Mar	Private residence	*S.* Typhimurium PT 170	4	0	D	Suspected chocolate mousse containing raw egg
NSW	Jan	Takeaway	*S.* Singapore	5	0	D	Suspected foods containing eggs (egg and salad wrap, egg salad)
	Jan	Bakery	*S.* Typhimurium	10	0	D	Suspected pork roll
	Jan	Restaurant	*S.* Typhimurium	2	1	D	Suspected pork buns
	Jan	Unknown	*S.* Typhimurium	2	0	D	Unknown pureed food
	Jan	Private residence	*S.* Typhimurium PT 170	5	4	D	Suspected mayonnaise prepared with raw eggs
	Jan	National franchised fast food	*S.* Typhimurium PT 9	Unknown	Unknown	AM	Aioli prepared with raw egg
	Jan	Restaurant	Suspected scombroid (histamine) fish poisoning	5	0	D	Mahi-mahi fish fillets
	Jan	Restaurant	Unknown	25	0	D	Unknown
	Jan	Takeaway	Unknown	3	0	D	Suspected assorted pizzas
	Feb	Restaurant	Unknown	3	0	D	Unknown
	Feb	Takeaway	*S.* Typhimurium	4	3	M	Barbecued pork
	Feb	Restaurant	Unknown	3	0	D	Suspected chicken or beef
	Feb	Restaurant	Unknown	4	0	D	Suspected lamb, beef and chicken skewers and an assortment of vegetables
	Feb	Restaurant	Unknown	4	0	D	Unknown
	Mar	Private residence	*S.* Typhimurium	9	1	D	Suspected tiramisu prepared with raw eggs
	Mar	Commercial caterer	Unknown	50	0	A	Suspected fruit kebabs
	Mar	Commercial manufactured food	Unknown	3	0	D	Suspected orange and mango fruit drink
	Mar	Takeaway	Unknown	3	1	D	Unknown
Qld	Jan	Primary produce	Ciguatera fish poisoning	6	0	D	Fish curry
	Jan	Private residence	Ciguatera fish poisoning	4	4	D	Mackerel soup
	Jan	Private residence	Norovirus	6	1	D	Unknown
	Jan	Restaurant	*S.* Typhimurium PT 170	6	1	D	Unknown
	Jan	Restaurant	*S.* Typhimurium PT 170	3	0	M	Deep fried ice cream
	Jan	Restaurant	*S.* Typhimurium PT 89	4	0	D	Unknown
	Jan	Restaurant	Unknown	4	0	D	Unknown
	Feb	Restaurant	*S.* Typhimurium PT 89	9	1	D	Unknown

Table 2: Outbreaks of foodborne disease reported by OzFoodNet sites,* 1 January to 31 March 2010 (n = 45), *continued*

State	Month of outbreak	Setting prepared	Agent	Number affected	Hospitalised	Evidence	Responsible vehicles
SA	Jan	Bakery	*S.* Typhimurium PT 9	9	0	D	Bakery products, no specific item identified
	Jan	Private residence	*S.* Typhimurium PT 9	6	0	D	Unknown
Tas	Mar	Restaurant	Unknown	26	0	AM	Unknown
Vic	Jan	Aged care	Not further specified	9	0	D	Unknown
	Jan	Private residence	Not further specified	19	0	D	Unknown
	Jan	Private residence	*S.* Typhimurium PT 170	12	1	D	Suspected eggs
	Jan	Restaurant	*S.* Typhimurium PT 9	13	1	D	Suspected eggs
	Feb	Aged care	*C. perfringens*	9	0	D	Unknown
	Feb	Commercial caterer	*S.* Typhimurium PT 141	14	1	D	Unknown
	Feb	Restaurant	*S.* Typhimurium PT 9	8	1	D	Suspected eggs
	Mar	Aged care	*C. perfringens*	17	0	D	Unknown
	Mar	Aged care	*C. perfringens*	16	0	D	Unknown
	Mar	Aged care	*Campylobacter*	5	0	D	Unknown
	Mar	Aged care	Not further specified	4	0	D	Unknown
	Mar	Private residence	*S.* Typhimurium PT 135a	5	0	D	Suspected chicken or eggs
WA	Dec	Restaurant	*S.* Typhimurium PT 170	7	1	D	Scrambled eggs
	Jan	Restaurant	*S.* Typhimurium PT 170	25	5	M	Aioli and Caesar salad
	Mar	Restaurant	Unknown	12	0	D	Karaage chicken and rice

The month of outbreak represents the month of onset of outbreak.

A Analytical epidemiological association between illness and one or more foods

D Descriptive evidence implicating the suspected vehicle or suggesting foodborne transmission

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

* No foodborne outbreaks were reported by the Northern Territory.

Communicable diseases surveillance

Tables

National Notifiable Diseases Surveillance System

A summary of diseases currently being reported by each jurisdiction is provided in Table 1. There were 47,235 notifications to the National Notifiable Diseases Surveillance System (NNDSS) with a notification received date between 1 January and 31 March 2010 (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 <2 years duration	All jurisdictions
Syphilis >2 years or unspecified duration	All jurisdictions except South Australia
Syphilis - congenital	All jurisdictions

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

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

* Notifiable in South Australia as of 1 May 2008.

† Flavivirus (NEC) replaced Arbovirus (NEC) from 1 January 2004. Arbovirus (NEC) replaced Flavivirus (NEC) from 2008.

NEC Not elsewhere classified.

Table 2: Notifications of diseases received by state and territory health authorities, 1 January to 31 March 2010, by date of diagnosis*

Disease	State or territory								Total 1st quarter 2010†	Total 4th quarter 2009	Total 1st quarter 2009	Last 5 years mean 1st quarter	Ratio‡	Year to date 2010	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	0.4	0.0	0	0.4
Hepatitis B (newly acquired)	1	7	0	18	6	1	22	0	55	51	40	64.0	0.9	55	64.0
Hepatitis B (unspecified)	20	784	44	267	95	19	499	203	1,931	1,781	1,840	1,649.0	1.2	1,931	1,649.0
Hepatitis C (newly acquired)	2	7	NN	0	10	1	44	0	64	90	69	91.4	0.7	64	91.4
Hepatitis C (unspecified)	56	1,168	35	713	106	55	534	278	2,945	2,650	2,809	2,958.0	1.0	2,945	2,958.0
Hepatitis D	0	1	0	3	0	0	0	0	4	6	10	9.8	0.4	4	9.8
Gastrointestinal diseases															
Botulism	0	NN	0	0	0	0	0	0	0	0	1	0.6	0.0	0	0.6
Campylobacteriosis§	110	0	38	1,272	306	210	1,650	572	4,158	4,203	4,079	4,295.2	1.0	4,158	4,295.2
Cryptosporidiosis	1	128	26	133	18	35	133	48	522	345	2,860	1,501.6	0.3	522	1,501.6
Haemolytic uraemic syndrome	0	2	0	0	0	0	1	0	3	4	3	5.4	0.6	3	5.4
Hepatitis A	3	34	0	10	3	0	40	9	99	239	70	77.8	1.3	99	77.8
Hepatitis E	1	3	0	1	0	0	4	0	9	1	16	12.4	0.7	9	12.4
Listeriosis	0	16	0	2	1	1	12	1	33	21	29	22.2	1.5	33	22.2
STEC, VTEC‖	0	4	0	23	12	0	4	4	47	58	53	30.6	1.5	47	30.6
Salmonellosis	69	1,345	151	1,098	163	87	729	361	4,003	2,411	3,393	3,106.4	1.3	4,003	3,106.4
Shigellosis	2	25	21	40	9	0	33	33	163	118	216	203.6	0.8	163	203.6
Typhoid	0	10	1	6	1	0	6	4	28	35	37	30.4	0.9	28	30.4
Quarantinable diseases															
Cholera	0	0	0	0	0	0	0	0	0	0	2	1.0	0.0	0	1.0
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	0.0

Table 2: Notifications of diseases received by state and territory health authorities, 1 January to 31 March 2010, by date of diagnosis,* *continued*

Disease	State or territory								Total 1st quarter 2010†	Total 4th quarter 2009	Total 1st quarter 2009	Last 5 years mean 1st quarter	Ratio‡	Year to date 2010	Last 5 years YTD mean
	ACT	NSW	NT	Qld	SA	Tas	Vic	WA							
Sexually transmissible infections															
Chlamydial infection¶	318	4,672	636	4,992	1,029	511	4,108	2,463	18,729	15,201	15,665	13,242.2	1.4	18,729	13,242.2
Donovanosis	0	0	0	0	0	0	0	0	0	0	0	1.6	0.0	0	1.6
Gonococcal infection	14	629	413	404	79	12	457	326	2,334	1,912	2,245	2,127.6	1.1	2,334	2,127.6
Syphilis (all)	7	338	31	105	8	6	202	41	738	646	726	692.4	1.1	738.0	692.4
Syphilis <2 years duration	4	79	9	56	8	2	65	23	246	258	337	268.6	0.9	246	268.6
Syphilis >2 years or unspecified duration	3	259	22	49	NDP	4	137	18	492	388	389	423.8	1.2	492	423.8
Syphilis - congenital	0	0	0	0	0	0	0	1	1	1	2	2.8	0.4	1	2.8
Vaccine preventable diseases															
Diphtheria	0	0	0	0	0	0	0	0	0	0	0	0.0	0.0	0	0.0
Haemophilus influenzae type b	0	0	1	2	0	0	0	0	3	2	8	4.8	0.6	3	4.8
Influenza (laboratory confirmed)	3	0	3	174	31	11	57	42	321	707	470	361.2	0.9	321	361.2
Measles	0	6	1	1	0	0	5	1	14	9	78	27.4	0.5	14	27.4
Mumps	0	6	0	8	0	0	2	2	18	30	57	67.6	0.3	18	67.6
Pertussis	54	1,316	52	1,523	1,202	62	984	143	5,336	6,959	8,569	3,155.4	1.7	5,336	3,155.4
Pneumococcal disease (invasive)	4	68	8	35	15	2	45	26	203	327	204	217.8	0.9	203	217.8
Poliomyelitis	0	0	0	0	0	0	0	0	0	0	0	0.0	0.0	0	0.0
Rubella	0	7	0	3	0	0	6	0	16	2	8	6.8	2.4	16	6.8
Rubella - congenital	0	0	0	0	0	0	0	0	0	0	0	0.0	0.0	0	0.0
Tetanus	0	0	0	0	0	0	1	0	1	0	3	1.4	0.7	1	1.4
Varicella zoster (chickenpox)	2	NN	10	1	79	0	17	44	153	272	349	231.0	0.7	153	231.0
Varicella zoster (shingles)	8	NN	30	5	250	2	147	172	614	651	676	376.0	1.6	614	376.0
Varicella zoster (unspecified)	18	NN	0	1,009	94	71	476	217	1,885	1,831	1,733	929.0	2.0	1,885	929.0
Vectorborne diseases															
Arbovirus infection (NEC)	0	0	0	2	0	0	1	0	3	4	14	10.8	0.3	3	10.8
Barmah Forest virus infection	1	81	41	266	11	0	13	36	449	293	569	588.4	0.8	449	588.4
Dengue virus infection	4	22	13	47	2	1	7	57	153	110	1,012	282.8	0.5	153	282.8
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	1	0	0	0	0	1	0	2	0.8	1.3	1	0.8
Malaria	0	16	4	41	3	1	16	16	97	95	135	194.2	0.5	97	194.2
Murray Valley encephalitis virus infection	0	0	0	0	0	0	0	0	0	0	2	1.0	0.0	0	1.0
Ross River virus infection	12	382	130	697	89	28	157	78	1,573	784	1,640	1,977.8	0.8	1,573	1,977.8

Table 2: Notifications of diseases received by state and territory health authorities, 1 January to 31 March 2010, by date of diagnosis,* *continued*

Disease	State or territory								Total 1st quarter 2010†	Total 4th quarter 2009	Total 1st quarter 2009	Last 5 years mean 1st quarter	Ratio‡	Year to date 2010	Last 5 years YTD mean
	ACT	NSW	NT	Qld	SA	Tas	Vic	WA							
Zoonoses															
Anthrax	0	1	0	0	0	0	0	0	1	0	0	0.4	2.5	1	0.4
Australian bat lyssavirus	0	0	0	0	0	0	0	0	0	0	0	0.0	0.0	0	0.0
Brucellosis	0	2	2	1	0	0	0	0	5	9	8	11.4	0.4	5	11.4
Leptospirosis	1	5	2	8	0	0	0	1	17	14	66	49.8	0.3	17	49.8
Lyssavirus (NEC)	0	0	0	0	0	0	0	0	0	0	0	0.0	0.0	0	0.0
Ornithosis	0	0	0	0	0	0	8	1	9	12	17	29.2	0.3	9	29.2
Q fever	0	35	0	26	2	0	2	2	67	69	95	100.4	0.7	67	100.4
Tularaemia	0	0	0	0	0	0	0	0	0	0	0	0.0	0.0	0	0.0
Other bacterial infections															
Legionellosis	1	20	0	6	3	0	17	8	55	64	66	78.4	0.7	55	78.4
Leprosy	0	0	0	0	0	0	0	0	0	2	1	3.0	0.0	0	3.0
Meningococcal infection**	0	17	0	8	7	2	5	5	44	63	47	56.4	0.8	44	56.4
Tuberculosis	0	106	5	83	19	2	90	26	331	454	299	274.6	1.2	331	274.6
Total	**712**	**11,263**	**1,698**	**13,034**	**3,653**	**1,120**	**10,534**	**5,221**	**47,235**	**42,536**	**50,293**	**39,329.5**	**1.2**	**47,235**	**39,329.45**

* Date of diagnosis = true onset date, or where not available, the earliest of (i) specimen date, (ii) notification date, or (iii) notification receive date. Hepatitis B and C unspecified were analysed by the notification receive date.

† 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.

‡ Ratio = ratio of current quarter total to the mean of last 5 years for the same quarter. Note: Ratios for varicella zoster (chickenpox), varicella zoster (shingles) and varicella zoster (unspecified) are based on 3 years of data.

§ Not reported for New South Wales where it is only notifiable as 'foodborne disease' or 'gastroenteritis in an institution'.

|| Infections with Shiga-like toxin (verotoxin) producing *Escherichia coli* (STEC/VTEC).

¶ Includes *Chlamydia trachomatis* identified from cervical, rectal, urine, urethral, throat and eye samples, except for South Australia, which reports only genital tract specimens; the Northern Territory and Queensland, which exclude ocular specimens; and Western Australia, which excludes ocular and perinatal infections.

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

NEC Not elsewhere classified.

NN Not notifiable.

NDP No data provided.

Table 3: Notification rates of diseases, 1 January to 31 March 2010, by state or territory. (Annualised rate per 100,000 population)

Disease*	State or territory								Aust
	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)	1.1	0.4	0.0	1.6	1.5	0.8	1.6	0.0	1.0
Hepatitis B (unspecified)	22.8	44.2	78.3	24.2	23.4	15.1	36.8	36.3	35.3
Hepatitis C (newly acquired)	2.3	0.4	0.0	NN	2.5	0.8	3.2	0.0	1.2
Hepatitis C (unspecified)	63.8	65.8	62.3	64.7	26.1	43.8	39.4	49.7	53.9
Hepatitis D	0.0	0.1	0.0	0.3	0.0	0.0	0.0	0.0	0.1
Gastrointestinal diseases									
Botulism	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
Campylobacteriosis†	125.3	NN	67.6	115.5	75.4	167.1	121.6	102.3	76.0
Cryptosporidiosis	1.1	7.2	46.3	12.1	4.4	27.9	9.8	8.6	9.5
Haemolytic uraemic syndrome	0.0	0.1	0.0	0.0	0.0	0.0	0.1	0.0	0.1
Hepatitis A	3.4	1.9	0.0	0.9	0.7	0.0	2.9	1.6	1.8
Hepatitis E	1.1	0.2	0.0	0.1	0.0	0.0	0.3	0.0	0.2
Listeriosis	0.0	0.9	0.0	0.2	0.2	0.8	0.9	0.2	0.6
STEC, VTEC‡	0.0	0.2	0.0	2.1	3.0	0.0	0.3	0.7	0.9
Salmonellosis	78.6	75.8	268.6	99.7	40.2	69.2	53.7	64.6	73.2
Shigellosis	2.3	1.4	37.4	3.6	2.2	0.0	2.4	5.9	3.0
Typhoid	0.0	0.6	1.8	0.5	0.2	0.0	0.4	0.7	0.5
Quarantinable diseases									
Cholera	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
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	0.0
Sexually transmissible infections									
Chlamydial infection§	362.2	263.2	1,131.4	453.1	253.6	406.7	302.7	440.4	342.5
Donovanosis	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
Gonococcal infection	15.9	35.4	734.7	36.7	19.5	9.5	33.7	58.3	42.7
Syphilis (all)	8.0	19.0	55.1	9.5	2.0	4.8	14.9	7.3	13.5
Syphilis <2 years duration	4.6	4.5	16.0	5.1	2.0	1.6	4.8	4.1	4.5
Syphilis >2 years or unspecified duration	3.4	14.6	39.1	4.4	NDP	3.2	10.1	3.2	9.0
Syphilis - congenital	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.2	0.0
Vaccine preventable diseases									
Diphtheria	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
Haemophilus influenzae type b	0.0	0.0	1.8	0.2	0.0	0.0	0.0	0.0	0.1
Influenza (laboratory confirmed)	3.4	0.0	5.3	15.8	7.6	8.8	4.2	7.5	5.9
Measles	0.0	0.3	1.8	0.1	0.0	0.0	0.4	0.2	0.3
Mumps	0.0	0.3	0.0	0.7	0.0	0.0	0.1	0.4	0.3
Pertussis	61.5	74.1	92.5	138.2	296.3	49.3	72.5	25.6	97.6
Pneumococcal disease (invasive)	4.6	3.8	14.2	3.2	3.7	1.6	3.3	4.6	3.7

Table 3: Notification rates of diseases, 1 January to 31 March 2010, by state or territory. (Annualised rate per 100,000 population), *continued*

Disease*	State or territory								Aust
	ACT	NSW	NT	Qld	SA	Tas	Vic	WA	
Vaccine preventable diseases, continued									
Poliomyelitis	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
Rubella	0.0	0.4	0.0	0.3	0.0	0.0	0.4	0.0	0.3
Rubella - congenital	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
Tetanus	0.0	0.0	0.0	0.0	0.0	0.0	0.1	0.0	0.0
Varicella zoster (chickenpox)	2.3	NN	17.8	0.1	19.5	0.0	1.3	7.9	2.8
Varicella zoster (shingles)	9.1	NN	53.4	0.5	61.6	1.6	10.8	30.8	11.2
Varicella zoster (unspecified)	20.5	NN	0.0	91.6	23.2	56.5	35.1	38.8	34.5
Vectorborne diseases									
Arbovirus infection (NEC)	0.0	0.0	0.0	0.2	0.0	0.0	0.1	0.0	0.1
Barmah Forest virus infection	1.1	4.6	72.9	24.1	2.7	0.0	1.0	6.4	8.2
Dengue virus infection	4.6	1.2	23.1	4.3	0.5	0.8	0.5	10.2	2.8
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	0.1	0.0	0.0	0.0	0.0	0.0
Malaria	0.0	0.9	7.1	3.7	0.7	0.8	1.2	2.9	1.8
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	13.7	21.5	231.3	63.3	21.9	22.3	11.6	13.9	28.8
Zoonoses									
Anthrax	0.0	0.1	0.0	0.0	0.0	0.0	0.0	0.0	0.0
Australian 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	3.6	0.1	0.0	0.0	0.0	0.0	0.1
Leptospirosis	1.1	0.3	3.6	0.7	0.0	0.0	0.0	0.2	0.3
Lyssavirus (NEC)	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
Ornithosis	0.0	0.0	0.0	0.0	0.0	0.0	0.6	0.2	0.2
Q fever	0.0	2.0	0.0	2.4	0.5	0.0	0.1	0.4	1.2
Tularaemia	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
Other bacterial infections									
Legionellosis	1.1	1.1	0.0	0.5	0.7	0.0	1.3	1.4	1.0
Leprosy	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
Meningococcal infection‖	0.0	1.0	0.0	0.7	1.7	1.6	0.4	0.9	0.8
Tuberculosis	0.0	6.0	8.9	7.5	4.7	1.6	6.6	4.6	6.1

* Rates are subject to retrospective revision.

† Not reported for New South Wales where it is only notifiable as 'foodborne disease' or 'gastroenteritis in an institution'.

‡ Infections with Shiga-like toxin (verotoxin) producing *Escherichia coli* (STEC/VTEC).

§ Includes *Chlamydia trachomatis* identified from cervical, rectal, urine, urethral, throat and eye samples, except for South Australia, which reports only genital tract specimens; the Northern Territory and Queensland, which exclude ocular specimens; and Western Australia, which excludes ocular and perinatal infections.

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

NN Not notifiable.

NEC Not elsewhere classified.

NDP No data provided.

Laboratory Serology and Virology Reporting Scheme

There were 9,585 reports received by the Laboratory Virology and Serology Reporting Scheme (LabVISE) in the reporting period, 1 January to 31 March 2010 (Tables 4 and 5).

Table 4: Laboratory Virology and Serology reports, 1 January to 31 March 2010 and total reports for the year,* by state or territory†

	State or territory								This period 2010	This period 2009	Year to date 2010	Year to date 2009
	ACT	NSW	NT	Qld	SA	Tas	Vic	WA				
Measles, mumps, rubella												
Measles virus	–	–	–	1	2	–	5	–	8	39	8	39
Mumps virus	–	–	–	2	3	–	1	–	6	13	6	13
Rubella virus	–	3	–	1	1	–	4	–	9	4	9	4
Hepatitis viruses												
Hepatitis A virus	–	2	–	11	2	–	1	–	16	8	16	8
Hepatitis D virus	–	–	–	1	2	–	1	–	4	7	4	7
Hepatitis E virus	–	–	–	1	–	–	–	–	1	3	1	3
Arboviruses												
Ross River virus	–	8	24	228	84	6	4	1	355	400	355	400
Barmah Forest virus	–	3	–	49	16	–	1	–	69	106	69	106
Flavivirus (unspecified)	1	9	–	42	–	–	7	–	59	149	59	149
Adenoviruses												
Adenovirus not typed/ pending	1	58	–	69	88	3	4	–	223	407	223	407
Herpesviruses												
Herpes virus type 6	–	–	–	–	–	–	1	–	1		1	
Cytomegalovirus	–	32	–	181	166	6	10	–	395	360	395	360
Varicella-zoster virus	–	23	–	543	240	3	2	–	811	752	811	752
Epstein-Barr virus	1	21	23	419	323	5	8	66	866	613	866	613
Other DNA viruses												
Parvovirus	–	2	–	9	52	–	10	–	73	66	73	66
Picornavirus family												
Rhinovirus (all types)	–	49	–	–	–	1	–	–	50	24	50	24
Enterovirus not typed/ pending	–	5	–	6	7	–	1	–	19	33	19	33
Picornavirus not typed	–	–	–	–	–	2	–	–	2	4	2	4
Ortho/paramyxoviruses												
Influenza A virus	–	21	–	61	56	–	10	–	148	86	148	86
Influenza B virus	1	3	–	7	12	–	–	–	23	35	23	35
Newcastle disease virus	–	1	–	–	–	–	–	–	1		1	
Parainfluenza virus type 1	–	39	–	29	2	–	–	–	70	3	70	3
Parainfluenza virus type 2	–	6	–	7	5	–	–	–	18	14	18	14
Parainfluenza virus type 3	–	9	–	4	20	–	–	–	33	44	33	44
Respiratory syncytial virus	–	93	–	118	34	–	–	–	245	251	245	251
Other RNA viruses												
HTLV-1	–	–	–	–	17	–	–	–	17	83	17	83
Rotavirus	–	9	–	–	7	–	–	–	16	63	16	63

Table 4: Laboratory Virology and Serology reports, 1 January to 31 March 2010 and total reports for the year,* by state or territory† *continued*

	State or territory								This period 2010	This period 2009	Year to date 2010	Year to date 2009
	ACT	NSW	NT	Qld	SA	Tas	Vic	WA				
Norwalk agent	–	31	–	–	271	3	–	–	305	16	305	16
Other												
Chlamydia trachomatis not typed	1	156	4	2,152	728	14	11	2	3,068	2,376	3,068	2,376
Chlamydia psittaci	–	–	–	–	–	1	7	–	8	23	8	23
Chlamydia spp typing pending	–	5	–	–	–	–	–	–	5	2	5	2
Chlamydia species	–	1	–	–	–	–	–	–	1	5	1	5
Mycoplasma pneumoniae	1	5	5	42	115	6	92	10	276	253	276	253
Coxiella burnetii (Q fever)	–	1	–	10	17	–	3	–	31	58	31	58
Rickettsia prowazeki	–	–	–	–	1	–	–	–	1	1	1	1
Rickettsia - spotted fever group	–	1	–	4	5	–	–	–	10	38	10	38
Streptococcus group A	–	12	1	154	–	–	10	–	177	178	177	178
Brucella species	–	–	–	1	–	–	2	–	3	4	3	4
Bordetella pertussis	–	55	–	521	948	1	48	–	1,573	1,362	1,573	1,362
Legionella pneumophila	–	1	–	1	2	–	–	–	4	6	4	6
Legionella longbeachae	–	–	–	–	2	–	3	–	5	3	5	3
Legionella species	1	2	–	5	–	–	2	–	10	5	10	5
Cryptococcus species	–	1	–	8	6	–	–	–	15	9	15	9
Leptospira species	–	1	–	10	3	–	–	1	15	18	15	18
Treponema pallidum	–	48	–	346	133	–	4	–	531	488	531	488
Entamoeba histolytica	–	–	–	3	–	–	–	–	3		3	
Toxoplasma gondii	–	–	–	–	5	–	–	–	5	7	5	7
Echinococcus granulosus	–	–	–	–	1	–	–	–	1	8	1	8
Total	**7**	**716**	**57**	**5,046**	**3,376**	**51**	**252**	**80**	**9,585**	**8,427**	**9,585**	**8,427**

* Data presented are for reports with reports dates in the current period.

† State or territory of postcode, if reported, otherwise state or territory of reporting laboratory.

– No data received this period.

Table 5: Laboratory Virology and Serology reports, 1 January to 31 March 2010,* by laboratory

State or territory	Laboratory	January 2010	February 2010	March 2010	Total
Australian Capital Territory	The Canberra Hospital	–	–	–	–
New South Wales	Institute of Clinical Pathology and Medical Research, Westmead	–	–	95	95
	New Children's Hospital, Westmead	60	50	103	213
	Repatriation General Hospital, Concord	–	–	–	–
	Royal Prince Alfred Hospital, Camperdown	–	–	–	–
	South West Area Pathology Service, Liverpool	41	59	61	161
Queensland	Queensland Medical Laboratory, West End	1,618	1,686	2,039	5,343
	Townsville General Hospital	–	–	–	–
South Australia	Institute of Medical and Veterinary Science, Adelaide	1,151	1,131	1,088	3,370
Tasmania	Northern Tasmanian Pathology Service, Launceston	14	16	15	45
	Royal Hobart Hospital, Hobart	–	–	–	–
Victoria	Australian Rickettsial Reference Laboratory	–	–	–	–
	Monash Medical Centre, Melbourne	–	–	–	–
	Royal Children's Hospital, Melbourne	–	–	66	66
	Victorian Infectious Diseases Reference Laboratory	68	49	47	164
Western Australia	PathWest Virology, Perth	–	–	–	–
	Princess Margaret Hospital, Perth	–	–	–	–
	Western Diagnostic Pathology	33	71	24	128
Total		2,985	3,062	3,538	9,585

* The complete list of laboratories reporting for the 12 months, January to December 2010, will appear in every report regardless of whether reports were received in this reporting period. Reports are not always received from all laboratories.

– No data received this period.

Additional reports

Australian childhood immunisation coverage

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 (formerly the Health Insurance Commission). The Register was established by transferring data on all children under the age of 7 years enrolled with Medicare to the ACIR.*

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 of age for the cohort born between 1 October and 31 December 2008, at 24 months of age for the cohort born between 1 October and 31 December 2007, and at 5 years of age for the cohort born between 1 October and 31 December 2004 according to the National Immunisation Program Schedule. However from March 2002 to December 2007, coverage for vaccines due at 4 years of age was assessed at the 6-year milestone age.

For information about the Australian Childhood Immunisation Register see Surveillance systems reported in CDI, *published in* Commun Dis Intell *2008;32:134–135 and for a full description of the methodology used by the Register see* Commun Dis Intell *1998;22:36-37.*

Commentary on the trends in ACIR data is provided by the National Centre for Immunisation Research and Surveillance of Vaccine Preventable Diseases (NCIRS). For further information please contact the NCIRS at telephone: +61 2 9845 1435, Email: brynleyh@chw.edu.au

'Fully immunised' at 12 months of age is defined as a child having a record on the ACIR of 3 doses of a diphtheria (D), tetanus (T) and pertussis-containing (P) vaccine, 3 doses of polio vaccine, 2 or 3 doses of PRP-OMP containing Haemophilus influenzae *type b (Hib) vaccine or 3 doses of any other* Haemophilus influenzae *type b (Hib) vaccine, 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, three doses of polio vaccine, 3 or 4 doses of PRP-OMP containing* Haemophilus influenzae *type b (Hib) vaccine or 4 doses of any other* Haemophilus influenzae *type b (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 5 years 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.*

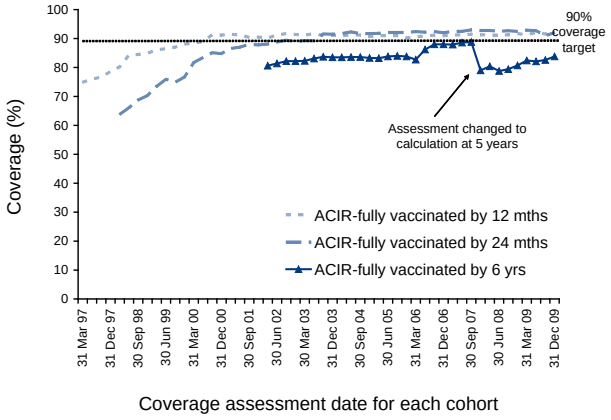
Immunisation coverage for 'fully immunised' at 12 months of age for Australia decreased slightly by 0.2 of a percentage point to 91.4% (Table 1). There were no important changes in coverage for any individual vaccines due at 12 months of age or by jurisdiction.

Immunisation coverage for 'fully immunised' at 24 months of age for Australia increased by 1 percentage point to 92.0 (Table 2). There were no important changes in coverage for any individual vaccines due at 24 months of age or by jurisdiction.

Immunisation coverage for 'fully immunised' at 5 years of age for Australia increased by 1.2 percentage points to sit currently at 83.8% (Table 3). However, 'fully immunised' coverage increased 2.1–2.2 percentage points in the Northern Territory and New South Wales and is now above 81% in all jurisdictions. These 2 jurisdictions also experienced similar increases in coverage for all individual vaccines due at 5 years of age.

Figure 1 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 6 years (5 years from March 2008), although coverage for vaccines due at 4 years decreases sig-

Figure 1: Trends in vaccination coverage, Australia, 1997 to 31 December 2009, by age cohorts



nificantly due to the change in assessment age from 6 to 5 years. It should also be noted that, currently, coverage for the vaccines added to the NIP since 2003 (varicella at 18 months, meningococcal C conjugate at 12 months and pneumococcal conjugate at 2, 4, and 6 months) are not included in the 12 or 24 months coverage data respectively.

Table 1. Percentage of children immunised at 1 year of age, preliminary results by disease and state or territory for the birth cohort 1 October to 31 December 2008; assessment date 31 March 2010

Vaccine	State or territory								Aust
	ACT	NSW	NT	Qld	SA	Tas	Vic	WA	
Total number of children	1,258	24,814	887	15,241	4,896	1,691	17,861	7,428	74,076
Diphtheria, tetanus, pertussis (%)	93.5	92.2	90.9	92.0	91.4	93.3	92.9	90.0	92.1
Poliomyelitis (%)	93.4	92.2	90.8	92.0	91.4	93.3	92.8	89.9	92.0
Haemophilus influenzae type b (%)	93.5	92.0	93.1	91.8	90.9	93.1	92.5	89.9	91.9
Hepatitis B (%)	92.8	91.9	91.0	91.7	90.8	93.0	92.2	89.6	91.6
Fully immunised (%)	92.8	91.7	89.4	91.5	90.6	93.0	92.0	89.2	91.4
Change in fully immunised since last quarter (%)	-0.4	-0.4	+1.4	-0.3	-0.7	+0.5	+0.1	-0.1	-0.2

Table 2. Percentage of children immunised at 2 years of age, preliminary results by disease and state or territory for the birth cohort 1 October to 31 December 2007; assessment date 31 March 2010*

Vaccine	State or territory								Aust
	ACT	NSW	NT	Qld	SA	Tas	Vic	WA	
Total number of children	1,205	24,383	927	15,078	4,924	1,616	18,090	7,678	73,901
Diphtheria, tetanus, pertussis (%)	95.9	95.1	94.4	94.1	94.5	94.9	95.6	93.3	94.8
Poliomyelitis (%)	95.9	95.0	94.4	94.1	94.5	94.9	95.6	93.2	94.8
Haemophilus influenzae type b (%)	95.9	95.2	93.3	93.5	93.2	95.1	94.8	92.9	94.4
Measles, mumps, rubella (%)	94.9	93.6	94.3	93.7	93.5	94.4	94.6	92.4	93.8
Hepatitis B (%)	95.4	94.6	94.3	93.6	93.8	94.7	94.7	92.8	94.2
Fully immunised (%)	93.9	92.3	92.0	91.6	91.5	93.5	92.6	89.9	92.0
Change in fully immunised since last quarter (%)	+0.7	+0.6	+1.3	+2.0	+2.0	+0.7	+0.9	-0.2	+1.0

* The 12 months age data for this cohort were published in *Commun Dis Intell* 2010;33(2):151.

Table 3. Percentage of children immunised at 5 years of age, preliminary results by disease and state or territory for the birth cohort 1 October to 31 December 2004; assessment date 31 March 2010

Vaccine	State or territory								Aust
	ACT	NSW	NT	Qld	SA	Tas	Vic	WA	
Total number of children	1,122	21,923	776	13,501	4,467	1,495	16,366	6,736	66,386
Diphtheria, tetanus, pertussis (%)	87.3	83.7	84.7	83.1	81.6	85.2	88.0	83.3	84.6
Poliomyelitis (%)	87.4	83.7	84.7	82.9	81.6	85.2	87.9	83.2	84.5
Measles, mumps, rubella (%)	86.7	83.5	84.4	82.7	81.4	84.8	87.6	82.8	84.2
Fully immunised (%)	86.3	83.0	83.3	82.2	81.0	84.4	87.3	82.3	83.8
Change in fully immunised since last quarter (%)	+0.8	+2.3	+2.1	-0.1	+0.5	-1.8	+1.1	+1.4	+1.2

Meningococcal surveillance

John Tapsall, The Prince of Wales Hospital, Randwick, NSW, 2031 for the Australian Meningococcal Surveillance Programme.

The reference laboratories of the Australian Meningococcal Surveillance Programme report data on the number of laboratory confirmed cases confirmed either by culture or by non-culture based techniques. Culture positive cases, where a Neisseria meningitidis *is grown from a normally sterile site or skin, and non-culture based diagnoses, derived from results of nucleic acid amplification assays and serological techniques, are defined as invasive meningococcal disease (IMD) according to Public Health Laboratory Network definitions. Data contained in 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 is contained in the annual reports of the Programme, published in Communicable Diseases Intelligence. For more information see* Commun Dis Intell *2009;33:82.*

Laboratory confirmed cases of invasive meningococcal disease for the period 1 January to 31 March 2010, are included in this issue of Communicable Diseases Intelligence *(Table 4).*

Table 4: Number of laboratory confirmed cases of invasive meningococcal disease, Australia, 1 January to 31 March 2010, by serogroup and state or territory

State or territory	Year	Serogroup													
		A		B		C		Y		W135		ND		All	
		Q1	YTD	Q1	YTD	Q1	YTD	Q1	YTD	Q1	YTD	Q1	YTD	Q1	YTD
Australian Capital Territory	10			0		0		0		0		0		0	
	09			0		0		0		0		0		0	
New South Wales	10			13		0		0		1		1		15	
	09			12		3		0		1		0		16	
Northern Territory	10			0		0		0		0		0		0	
	09			2		1		0		0		0		3	
Queensland	10			6		0		0		0		0		6	
	09			11		0		0		0		0		11	
South Australia	10			4		0		1		0		0		5	
	09			4		0		0		0		0		4	
Tasmania	10			1		0		0		0		1		2	
	09			0		0		0		0		0		0	
Victoria	10			3		0		1		1		0		5	
	09			5		1		0		0		2		8	
Western Australia	10			2		1		0		0		0		3	
	09			2		2		0		0		0		4	
Total	10			29		1		2		2		2		36	
	09	0		36		7		0		1		2		46	

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Australian Sentinel Practices Research Network

The Australian Sentinel Practices Research Network (ASPREN) is a national surveillance system that is owned and operated by the Royal Australian College of General Practitioners and directed through the Discipline of General Practice at the University of Adelaide.

The network consists of general practitioners who report presentations on a number of defined medical conditions each week. ASPREN was established in 1991 to provide a rapid monitoring scheme for infectious diseases that can alert public health officials of epidemics in their early stages as well as play a role in the evaluation of public health campaigns and research of conditions commonly seen in general practice. Electronic data collection was established in 2006 and currently, further development of ASPREN is in progress to create an automatic reporting system.

The list of conditions is reviewed annually by the ASPREN management committee and an annual report is published. In 2009, four conditions are being monitored. They include influenza-like (ILI) illness, gastroenteritis and varicella infections (chickenpox and shingles). Definitions of these conditions are described in Surveillance systems reported in CDI, *published in* Commun Dis Intell *2010;34:82–83.*

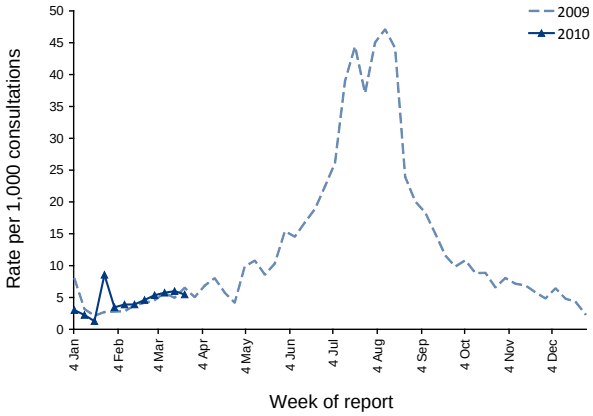
Data on influenza-like illness, gastroenteritis, chickenpox and shingles from 1 January to 31 March 2010 compared with 2009, are shown as the rate per 1,000 consultations in Figures 2, 3, 4 and 5, respectively.

Reporting period 1 January to 31 March 2010

Sentinel practices contributing to ASPREN were located in all jurisdictions other than the Northern Territory. A total of 98 general practitioners contributed data to ASPREN in the 1st quarter of 2010. Each week an average of 76 general practitioners provided information to ASPREN at an average of 7,334 (range 2,978 to 8,953) consultations per week and an average of 89 (range 29 to 120) notifications per week.

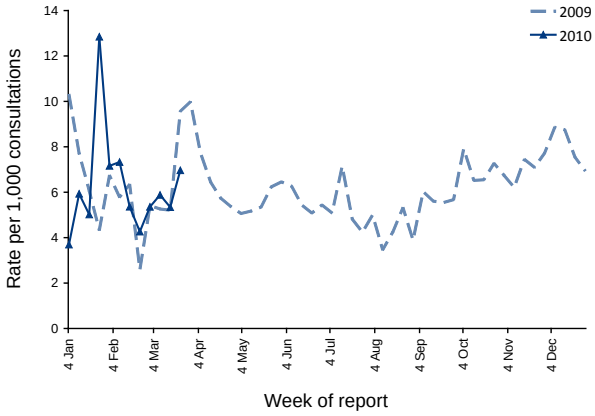
ILI rates reported from 1 January to 31 March 2010 were 1–9 cases per 1,000 consultations. The reported rates in January, February and March 2010 (1–9 cases per 1,000 consultations, 3–5 cases per 1,000 consultations and 5–6 cases per 1,000 consultations respectively) were relatively consistent compared with rates in the same reporting period in 2009 (2–8 cases per 1,000 consultations, 3–4 cases per 1,000 consultations and 5–7 cases per 1,000 consultations respectively) (Figure 2).

Figure 2: Consultation rates for influenza-like illness, ASPREN, 1 January 2009 to 31 March 2010, by week of report



During this reporting period, consultation rates for gastroenteritis ranged from 4 to 13 cases per 1,000 (Figure 3). This was slightly higher compared with the same reporting period in 2009 (3 to 10 cases per 1,000 consultations).

Figure 3: Consultation rates for gastroenteritis, ASPREN, 1 January 2009 to 31 March 2010, by week of report



Varicella infections were reported at a similar rate for the 1st quarter of 2010 compared with the same period in 2009. From 1 January to 31 March 2010, recorded rates for chickenpox were between 0 and 0.7 cases per 1,000 consultations (Figure 4).

In the 1st quarter of 2010, reported rates for shingles were between 0.3 and 1.9 cases per 1,000 consultations (Figure 5), slightly lower than the same reporting period in 2009 (0.3 to 2.6 cases per 1,000 consultations).

Figure 4: Consultation rates for chickenpox, ASPREN, 1 January 2009 to 31 March 2010, by week of report

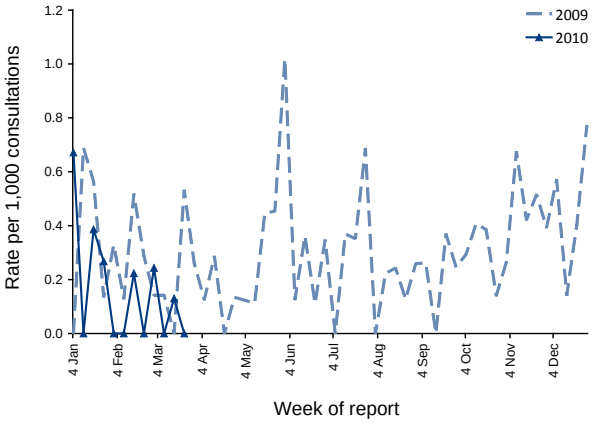
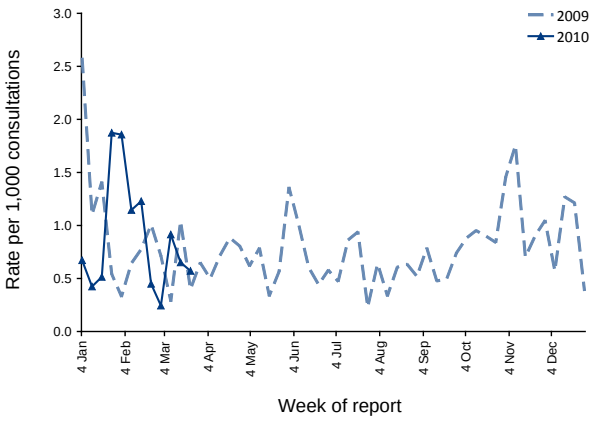


Figure 5: Consultation rates for shingles, ASPREN, 1 January 2009 to 31 March 2010, by week of report

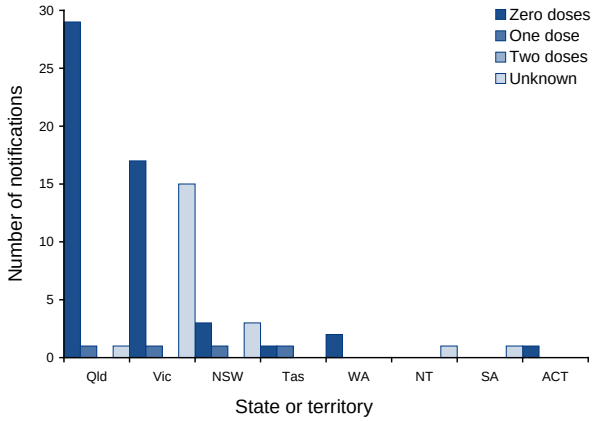


Errata

Measles status in Australia and outbreaks in the first quarter of 2009

The Figure 3 published in the report 'Measles status in Australia and outbreaks in the first quarter of 2009' (*Commun Dis Intell* 2009;33(2):225–231) was incorrect. The correct figure is reproduced below.

Figure 3: Notifications of measles, Australia, 1 January to 31 March 2009, by state or territory and number of vaccine doses



Communicable diseases surveillance: tables

The National Notifiable Diseases Surveillance System quarterly data published in the last issue of *Communicable Diseases Intelligence* contained errors in the total columns of Table 2. (*Commun Dis Intell* 2010;34(1):70–72). The following table contains the correct data.

Table 2: Notifications of diseases received by state and territory health authorities, 1 October to 31 December 2009, by date of diagnosis*

Disease	State or territory								Total 4th quarter 2009†	Total 3rd quarter 2009	Total 4th quarter 2008	Last 5 years mean 4th quarter	Ratio‡	Year to date 2009	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	1	0.2	0.0	0	0.6
Hepatitis B (newly acquired)	3	12	3	12	1	2	20	0	53	48	45	62.2	0.9	197	272.2
Hepatitis B (unspecified)	20	803	38	246	99	21	508	197	1,932	2,040	1,418	1,510.6	1.3	7,878	6,289.8
Hepatitis C (newly acquired)	2	11	1	NN	13	4	51	0	82	69	100	89.0	0.9	297	370.8
Hepatitis C (unspecified)	38	1,261	45	649	122	65	521	302	3,003	3,270	2,491	2,831.4	1.1	12,695	11,800.2
Hepatitis D	0	1	0	2	0	0	3	0	6	5	6	5.8	1.0	32	33.4
Gastrointestinal diseases															
Botulism	0	0	0	0	0	0	0	0	0	0	0	0.2	0.0	1	1.2
Campylobacteriosis§	94	NN	29	1,059	440	174	1,699	697	4,192	3,819	4,082	4,543.8	0.9	15,851	16,008.2
Cryptosporidiosis	5	110	28	40	14	29	90	27	343	259	423	589.0	0.6	4,599	2,577.2
Haemolytic uraemic syndrome	0	1	1	0	1	0	1	0	4	0	11	8.2	0.5	12	20.0
Hepatitis A	2	29	0	17	7	5	167	13	240	86	41	52.6	4.6	562	272.4
Hepatitis E	0	1	0	0	0	0	0	0	1	8	9	5.2	0.2	35	28.8
Listeriosis	1	5	0	2	0	1	9	3	21	22	8	14.0	1.5	91	59.8
STEC, VTEC‖	0	6	1	25	21	0	3	2	58	23	36	27.0	2.1	160	82.8
Salmonellosis	53	724	132	504	168	40	460	326	2,407	1,501	2,054	2,149.6	1.1	9,526	8,455.4
Shigellosis	3	25	20	29	11	0	12	19	119	124	199	155.8	0.8	629	643.4
Typhoid	0	16	0	2	1	0	14	2	35	25	19	15.6	2.2	116	79.0
Quarantinable diseases															
Cholera	0	0	0	0	0	0	0	0	0	1	4	1.8	0.0	4	3.8
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	0.0

Table 2: Notifications of diseases received by state and territory health authorities, 1 October to 31 December 2009, by date of diagnosis* *continued*

Disease	State or territory								Total 4th quarter 2009†	Total 3rd quarter 2009	Total 4th quarter 2008	Last 5 years mean 4th quarter	Ratio‡	Year to date 2009	Last 5 years YTD mean
	ACT	NSW	NT	Qld	SA	Tas	Vic	WA							
Sexually transmissible infections															
Chlamydial infection¶	226	3,716	470	4,052	850	352	3,378	2,149	15,193	15,124	13,966	11,548.4	1.3	62,677	47,062.6
Donovanosis	0	0	0	0	0	0	0	0	0	0	0	1.6	0.0	1	6.8
Gonococcal infection	6	496	349	381	61	1	343	278	1,915	1,699	1,807	1,856.2	1.0	8,094	7,828.6
Syphilis (all)	4	236	24	119	5	5	204	43	640	736	704	661.0	1.0	2,840	2,704.0
Syphilis <2 years duration	0	96	5	42	5	3	83	18	252	331	275	241.6	1.0	1,259	975.4
Syphilis >2 years or unspecified duration	4	140	19	77	NDP	2	121	25	388	405	429	419.4	0.9	1,581	1,728.6
Syphilis - congenital	0	0	1	0	0	0	0	0	1	1	2	2.4	0.4	4	11.2
Vaccine preventable diseases															
Diphtheria	0	0	0	0	0	0	0	0	0	0	0	0.0	0.0	0	0.0
Haemophilus influenzae type b	0	0	1	0	0	0	1	0	2	2	5	5.0	0.4	20	19.2
Influenza (laboratory confirmed)	8	0	24	275	177	11	70	135	700	34,824	1,521	767.0	0.9	47,718	5,897.8
Measles	0	7	0	0	0	0	1	1	9	8	1	6.2	1.5	104	51.4
Mumps	0	9	2	10	2	0	0	6	29	34	35	90.4	0.3	162	297.6
Pertussis	77	1,815	25	1,710	1,888	100	1,095	205	6,915	6,454	6,777	3,145.4	2.2	29,514	10,032.4
Pneumococcal disease (invasive)	7	97	19	63	33	2	80	26	327	625	353	349.6	0.9	1,564	1,734.8
Poliomyelitis	0	0	0	0	0	0	0	0	0	0	0	0.0	0.0	0	0.2
Rubella	0	0	0	1	0	0	0	1	2	7	9	7.6	0.3	25	38.2
Rubella - congenital	0	0	0	0	0	0	0	0	0	0	0	0.0	0.0	0	0.8
Tetanus	0	0	0	0	0	0	0	0	0	0	0	1.0	0.0	3	3.4
Varicella zoster (chickenpox)	1	NN	17	4	124	8	22	68	244	328	768	500.0	0.5	1,238	1,672.0
Varicella zoster (shingles)	7	NN	26	2	263	27	225	151	701	609	756	387.5	1.8	2,828	1,241.8
Varicella zoster (unspecified)	20	NN	2	1,001	77	25	431	214	1,770	1,680	1,381	873.5	2.0	6,693	3,097.8
Vectorborne diseases															
Arbovirus infection (NEC)	0	0	0	3	0	0	0	0	3	5	10	5.6	0.5	27	33.4
Barmah Forest virus infection	0	75	19	163	8	1	4	23	293	271	414	346.8	0.8	1,493	1,672.6
Dengue virus infection	6	17	5	42	1	0	9	30	110	87	193	74.0	1.5	1,401	322.8
Japanese encephalitis virus infection	0	0	0	0	0	0	0	0	0	0	0	0.0	0.0	0	0.4
Kunjin virus infection	0	0	0	0	0	0	0	0	0	0	0	0.6	0.0	2	2.4
Malaria	1	12	2	35	7	1	16	21	95	151	127	141.4	0.7	533	646.2
Murray Valley encephalitis virus infection	0	0	0	0	0	0	0	0	0	0	0	0.0	0.0	4	1.2
Ross River virus infection	0	128	71	358	94	4	16	107	778	833	914	728.2	1.1	4,777	4,425.2

Table 2: Notifications of diseases received by state and territory health authorities, 1 October to 31 December 2009, by date of diagnosis* *continued*

Disease	State or territory								Total 4th quarter 2009†	Total 3rd quarter 2009	Total 4th quarter 2008	Last 5 years mean 4th quarter	Ratio‡	Year to date 2009	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	0.4
Australian bat lyssavirus	0	0	0	0	0	0	0	0	0	0	0	0.0	0.0	0	0.0
Brucellosis	0	1	0	7	0	0	0	0	8	6	12	13.6	0.6	31	43.0
Leptospirosis	0	1	0	6	0	0	7	0	14	16	22	22.8	0.6	145	134.0
Lyssavirus (NEC)	0	0	0	0	0	0	0	0	0	0	0	0.0	0.0	0	0.0
Ornithosis	0	0	0	0	0	0	10	1	11	18	17	33.6	0.3	60	152.2
Q fever	0	26	0	27	7	0	5	0	65	66	88	98.8	0.7	308	406.6
Tularaemia	0	0	0	0	0	0	0	0	0	0	0	0.0	0.0	0	0.0
Other bacterial infections															
Legionellosis	1	19	0	7	14	0	14	8	63	74	70	84.0	0.8	305	313.0
Leprosy	0	0	0	2	0	0	0	0	2	0	3	1.8	1.1	3	9.4
Meningococcal infection**	0	20	1	19	3	0	14	6	63	86	63	78.2	0.8	259	340.8
Tuberculosis	13	162	5	73	13	2	159	35	462	340	351	331.4	1.4	1,325	1,142.2
Total	598	9,842	1,361	10,947	4,525	880	9,662	5,096	42,911	75,384	41,316	34,612.0	1.3	228,226	138,345

* Date of diagnosis = true onset date, or where not available, the earliest of (i) specimen date, (ii) notification date, or (iii) notification receive date. Hepatitis B and C unspecified were analysed by the notification receive date.

† 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.

‡ Ratio = ratio of current quarter total to the mean of last 5 years for the same quarter. Note: Ratios for varicella zoster (chickenpox), varicella zoster (shingles) and varicella zoster (unspecified) are based on 2 years of data.

§ Not reported for New South Wales where it is only notifiable as 'foodborne disease' or 'gastroenteritis in an institution'.

|| Infections with Shiga-like toxin (verotoxin) producing *Escherichia coli* (STEC/VTEC).

¶ Includes *Chlamydia trachomatis* identified from cervical, rectal, urine, urethral, throat and eye samples, except for South Australia, which reports only genital tract specimens; the Northern Territory and Queensland, which exclude ocular specimens; and Western Australia, which excludes ocular and perinatal infections.

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

NN Not notifiable.

NEC Not elsewhere classified.

NDP No data provided.