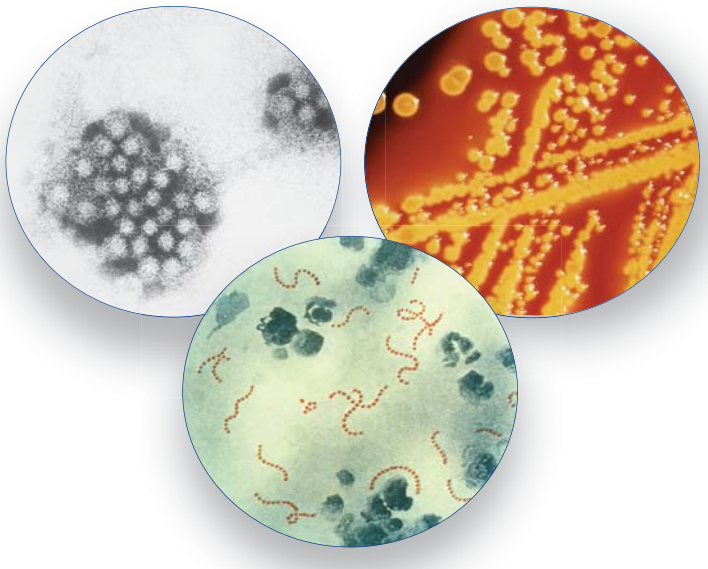
Australian Government
Department of Health and Ageing

Communicable Diseases Intelligence



Quarterly report

Volume 29
Issue no 4
2005

Communicable Diseases Intelligence

Quarterly report

Volume 29

Issue no 4

2005

ISBN 0725-3141

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* 2005;29:95–97.

Communicable Diseases Intelligence contributes to the work of the Communicable Diseases Network Australia (http://www.health.gov.au/internet/wcms/publishing.nsf/Content/cda-cdna-index.htm)

Website

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

Subscriptions and contacts

Communicable Diseases Intelligence is produced every quarter by:
Surveillance Section
Biosecurity and Disease Control Branch
Australian Government Department of Health and Ageing
GPO Box 9848, (MDP 6)
CANBERRA ACT 2601;
Telephone: +61 2 6289 8245
Facsimile: +61 2 6289 7791
Email: cdi.editor@health.gov.au

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

Disclaimer

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

Front cover: Images were sourced from the Centers for Disease Control and Prevention Public Health Image Library, courtesy of the Centers for Disease Control and Prevention, Atlanta, Georgia.
Clockwise from top left: An electron micrograph of norovirus; *Escherichia coli* bacteria; photomicrograph of *Streptococcus pyogenes* bacteria.

Printed by Union Offset, Canberra

Contents

Cont'd next page

Contents, *cont.*

Communicable Diseases Network Australia: National Arbovirus and Malaria Advisory Committee annual report 2004–05

Conan Liu,[1] Annette K Broom,[2] Nina Kurucz,[3] Peter I Whelan[3]

Abstract

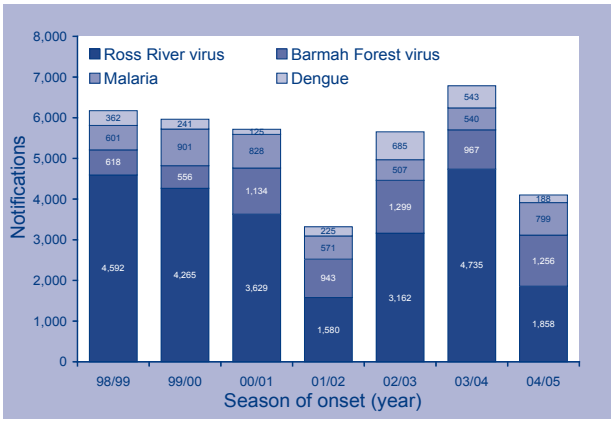
This report describes the epidemiology of mosquito-borne disease in Australia for the mosquito-borne disease season 1 July 2004 to 30 June 2005. Ross River virus (RRV) infections (45%), Barmah Forest virus (BFV) infections (30%) and malaria (19%) were the most common mosquito-borne diseases reported in 2004–05. The Northern Territory had the highest rate of RRV notifications and the peak notification rate (in February 2005) was 54 per cent less than the previous season. The Northern Territory also reported the highest BFV notification rate this season, peaking in April 2005, which was the second highest reported BFV notification rate since 1998. National RRV and BFV notification rates were highest in the 45–49 year age group. There were 799 notifications of malaria in 2004–05 of which none were reported as locally acquired. This was the third highest reporting season for malaria notifications since 1998. In contrast to previous years in which *Plasmodium vivax* was the predominant species, *Plasmodium falciparum* was reported as the infecting species in 57 per cent of the malaria notifications and *Plasmodium vivax* for 34 per cent of cases. Children in the 5–9 year age group had the highest number of cases compared to previous years in which the peak number of cases tended to be in young adult age groups. There were four cases of Kunjin virus (KUNV) and two cases of Murray Valley encephalitis virus (MVEV) reported in 2004–05. Sentinel chicken surveillance data for flaviviruses and sentinel pig surveillance data for Japanese encephalitis virus are reported. There were 188 notifications of dengue virus infection (DENV) in 2004–05, of which 46 per cent (n=86) were reported as having been acquired overseas. Dengue serotype 4 was the most frequently reported type, accounting for 32 per cent of cases (n=60). *Commun Dis Intell* 2005;29:341–357.

Notifications

Historical data from 1998 are also shown for comparison. Data were extracted by onset date from the National Notifiable Diseases Surveillance System (NNDSS) on 4 August 2005. During this reporting period, there were 4,127 notifications of mosquito-borne diseases (MBD) reported in Australia. Ross River virus infections accounted for 45 per cent (n=1,858) of these notifications (Appendix 1).

Figure 1 shows that the next two highest contributors to MBD notifications in 2004–05 were Barmah Forest virus infections (30% of total, n=1,256) and malaria (19%, n=799). Overall, total MBD notifications during 2004–05 were much lower than the 2002–03 and 2003–04 seasons, and were similar to the 2001–02 season.

Figure 1. Notifications of select mosquito-borne diseases, Australia, 1998 to 2005, by season of onset



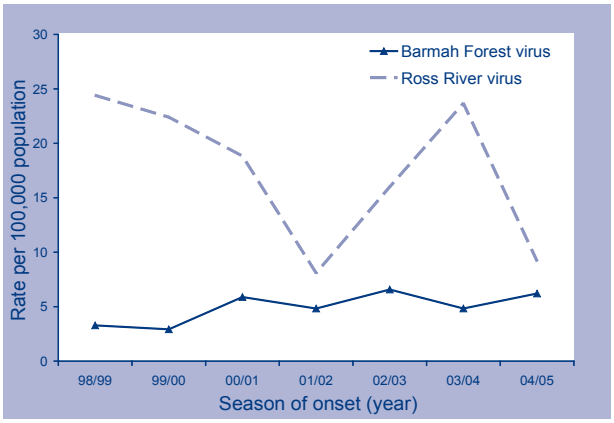
1. Surveillance Section, Australian Government Department of Health and Ageing, Canberra, Australian Capital Territory
2. Arbovirus Surveillance and Research Laboratory, Department of Microbiology, The University of Western Australia, Western Australia
3. Medical Entomology Branch, Communicable Disease Control, Northern Territory Department of Health and Community Services, Northern Territory

Corresponding author: Mr Conan Liu, Surveillance Section, Australian Government Department of Health, GPO Box 9848 (MDP 6), Canberra ACT 2601. Telephone: +61 02 6289 8564. Facsimile: +61 2 6289 7791. Email: conan.liu@health.gov.au

The national notification rates for RRV and BFV in 2004–05 were 9.2 cases per 100,000 population and 6.2 cases per 100,000 population, respectively (Appendix 1).

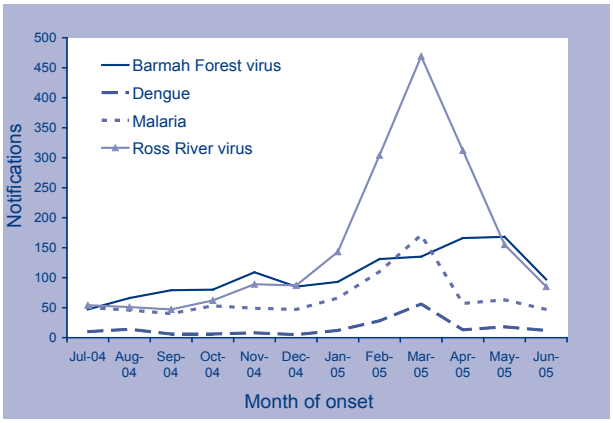
Figure 2 shows that there was a reduction in the rate of notifications for RRV infections in the 2004–05 season compared to last season and similar to the rate reported in 2001–02. The notification rate for BFV has remained relatively stable over the last six seasons.

Figure 2. Notification rate of select mosquito-borne diseases, Australia, 1998 to 2005, by season of onset



During the 2004–05 MBD season (1 July 2004 to 30 June 2005), the highest number of notifications for RRV, malaria and DENV were received in March (Appendix 2, Figure 3). BFV notifications rose steadily from July 2004 to reach the highest number of notifications for the season in April-May 2005.

Figure 3. Notifications of select mosquito-borne diseases, Australia, 1 July 2004 to 30 June 2005, by month of onset



Alphaviruses

During 2004–05, the Northern Territory had the highest rates of BFV notifications (21.4 cases per 100,000 population) and RRV notifications (89.1 cases per 100,000 population) (Table 1). Queensland reported the second highest notification rate for BFV (17.2 cases per 100,000 population) and RRV (25.9 cases per 100,000 population).

Ross River virus infection

The number of notifications of RRV infection began to rise in the Northern Territory during October 2004, peaking in February 2005. High rates of infection were reported, between two and eighteen times above the national rate (Figure 4). The Northern Territory peak notification rate in February 2005 (328.7 per 100,000 population) was 54 per cent less than the previous season, but similar to the 1998–99, 1999–00 and 2002–03 seasons.

Table 1. Number and crude annual rate of Barmah Forest virus and Ross River virus notifications, Australia, 1 July 2004 to 30 June 2005, by jurisdiction

Jurisdiction	Barmah Forest virus		Ross River virus	
	Notifications	Rate (per 100,000)	Notifications	Rate (per 100,000)
ACT	2	0.6	5	1.5
NSW	432	6.4	430	6.4
NT	43	21.4	179	89.1
Qld	675	17.2	1,014	25.9
SA	18	1.2	49	3.2
Tas	0	0.0	5	1.0
Vic	22	0.4	35	0.7
WA	64	3.2	141	7.1
Australia	1,256	6.2	1,858	9.2

Figure 4. Annualised notification rates for Ross River virus infection, select jurisdictions, July 1998 to June 2005, by month and season of onset

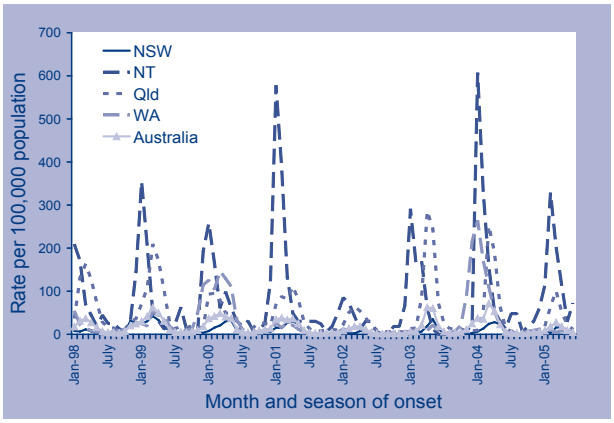
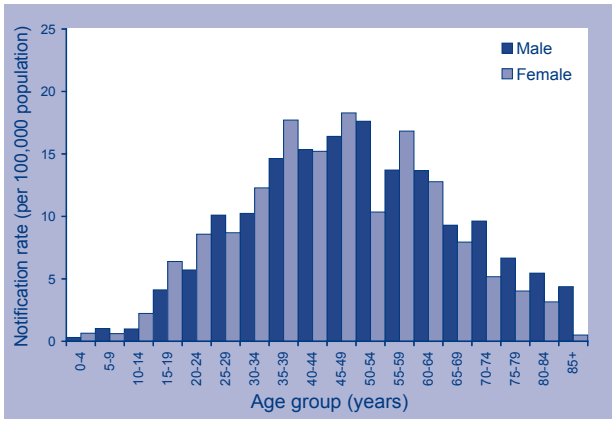


Figure 5. Notification rates for Ross River virus infections, Australia, 1 July 2004 to 30 June 2005, by age group and sex



Queensland reported a similar pattern but lower rates of RRV infection, with increases beginning in December 2004, peaking in March 2005 and subsiding in June 2005. Queensland's peak RRV rate was a 35–40 per cent decrease compared to the peak seasonal rates in April 2003, and March 2004.

In contrast to last season, the rate of RRV notifications in Western Australia remained around the national average throughout the reporting period.

The rate of national notifications for RRV was highest in the 45–49 age group (Figure 5). Females in the 45–49 and the 35–39 age groups, had the highest national notification rates (18.3 cases per 100,000 population and 17.7 cases per 100,000 population respectively, Appendix 3). The highest male national notification rate was reported in the 50–54 age group (17.6 cases per 100,000 population).

In general, state and territories showed similar age and sex distribution patterns, noting that small population sizes in older age groups in some jurisdictions can affect reporting rates (as small changes in the numerator lead to large changes in the overall rate).

Queensland, Western Australia and the Northern Territory reported higher age-specific RRV notification rates in females than in males (Appendix 3). New South Wales reported the highest notification rates in the 45–49 year age group (Figure 6), with males having higher notification rates than females in the same age group (14.8 and 11.7 cases per 100,000 population). The sex ratio for this age cohort was 99.3 males per 100 females.

In Queensland, the highest notification rates were reported in females in the 35–39 year age group (58 cases per 100,000 population) and in males in the 40–44 year age group (49.2 cases per 100,000 population) (Figure 7).

Figure 6. Notification rate for Ross River virus infections, New South Wales, 1 July 2004 to 30 June 2005, by age group and sex

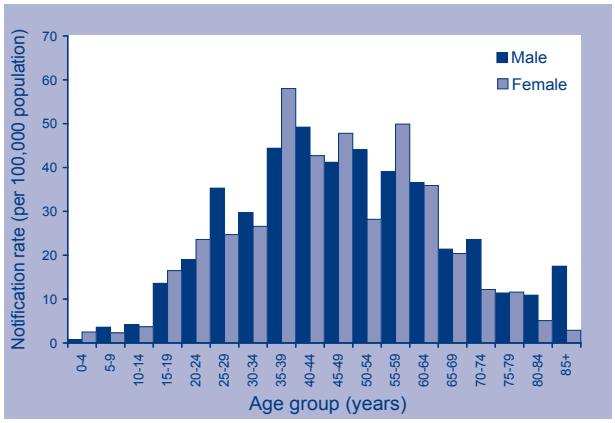
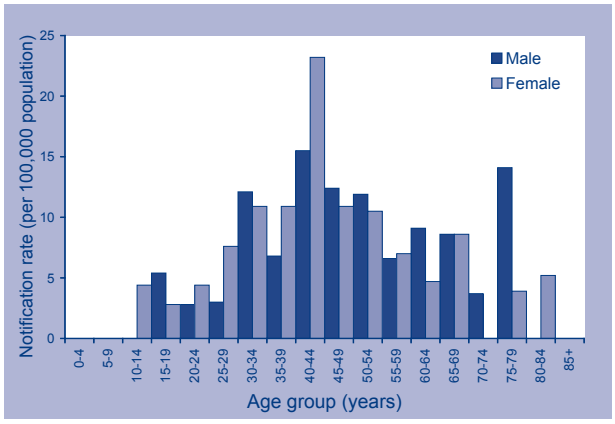


Figure 7. Notification rate for Ross River virus infections, Queensland, 1 July 2004 to 30 June 2005, by age group and sex



In Western Australia, the highest notification rates were reported from the 40–44 year age group for both sexes (males 15.5 cases per 100,000; females 23.2 cases per 100,000 population) (Figure 8). The Northern Territory reported the highest female notification rate in the 45–49 year age group (161.8 cases per 100,000 population) while the highest male notification rates (161.8 cases per 100,000 population) were reported in the same 40–44 year age group as Queensland and Western Australia (Figure 9). The overall higher notification rates in females were particularly striking given that the sex ratio in the Northern Territory was 111 males per 100 females.

Figure 8. Notification rate for Ross River virus infections, Western Australia, 1 July 2004 to 30 June 2005, by age group and sex

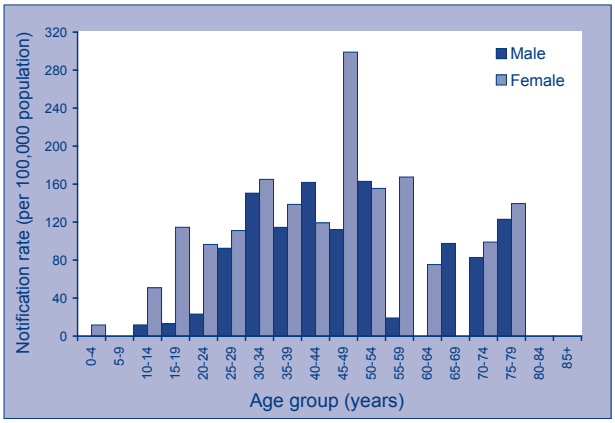
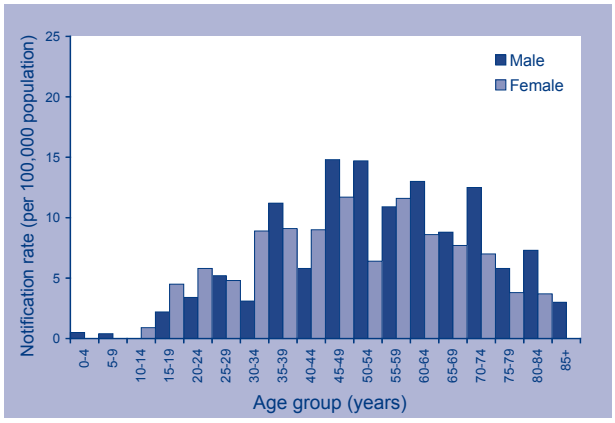


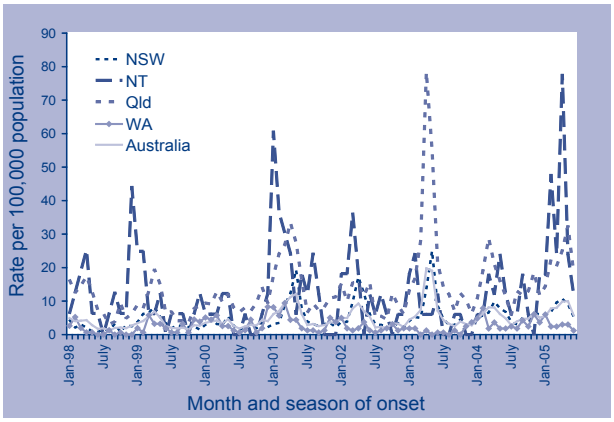
Figure 9. Notification rate for Ross River virus infections, Northern Territory, 1 July 2004 to 30 June 2005, by age group and sex



Barmah Forest virus infections

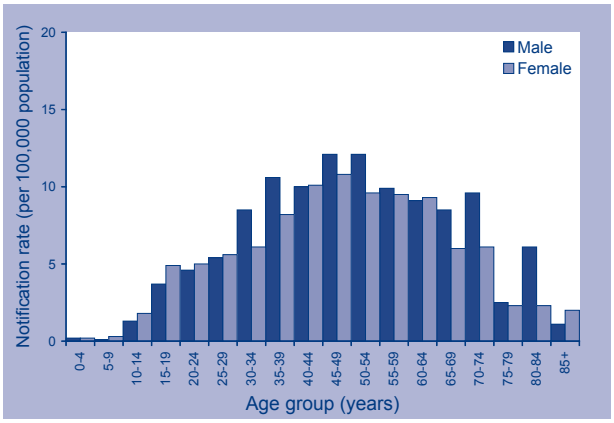
Queensland reported the highest number of BFV notifications (n=675) for this period whereas the Northern Territory had the highest crude annual rate of BFV infection (21.4 cases per 100,000 population) (Table 1). The monthly annualised BFV notification rate for the Northern Territory increased gradually from August 2004 and peaked in April 2005 (Table 1, Figure 10), to a rate of 77.7 cases per 100,000 population. This was the second highest reported BFV notification rate since 1998, with Queensland reporting the highest monthly annualised BFV notification rate in April 2003 (78.4 cases per 100,000 population).

Figure 10. Annualised notification rates for Barmah Forest virus infections, select jurisdictions, July 1998 to June 2005, by month and season of onset



The age group and sex distribution of BFV cases is similar to that of RRV with the majority reported in adults. The national rate of notifications for BFV was highest amongst the 45–49 year age group (11.4 cases per 100,000 population), with males and females in this age group having the highest national age specific rates (Figure 11, Appendix 4). High rates of national BFV notifications were also recorded for the 50–54 year age group in males (12.1 cases per 100,000 population).

Figure 11. Notification rate for Barmah Forest virus infections, Australia, 1 July 2004 to 30 June 2005, by age group and sex



New South Wales, Queensland and Western Australia reported the highest age-specific BFV notification rates as being in males (Figures 12, 13, and 14, Appendix 4). In Western Australia, the highest notification rate was reported in males in the 70–74 year age group while in the Northern Territory, the highest rate was observed in both males and females (Figure 15).

Figure 12. Notification rate for Barmah Forest virus infections, New South Wales, 1 July 2004 to 30 June 2005, by age group and sex

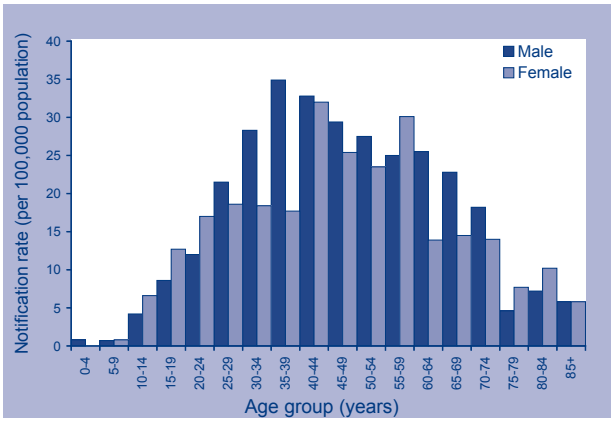
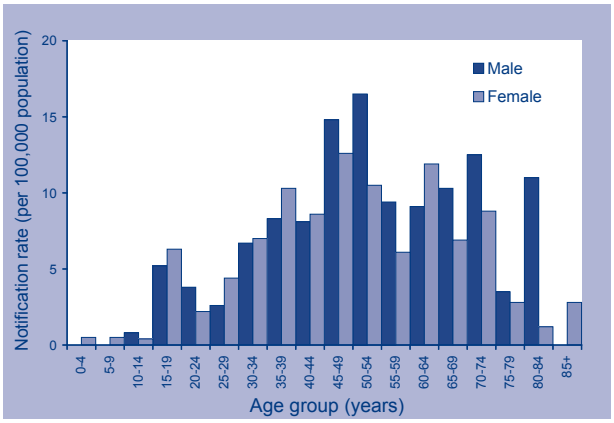


Figure 13. Notification rate for Barmah Forest virus infections, Queensland, 1 July 2004 to 30 June 2005, by age group and sex



Flaviviruses

The Sentinel Chicken Surveillance Programme is a network involving New South Wales, Queensland, the Northern Territory, Victoria and Western Australia, and is designed to detect flavivirus activity (including the endemic arboviruses MVEV and KUNV).[1]

Figure 14. Notification rate for Barmah Forest virus infections, Western Australia, 1 July 2004 to 30 June 2005, by age group and sex

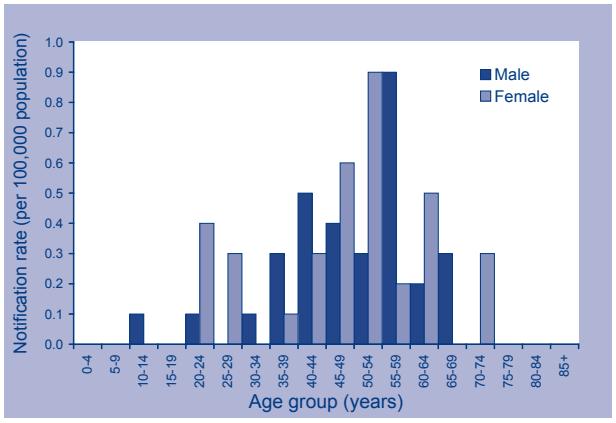
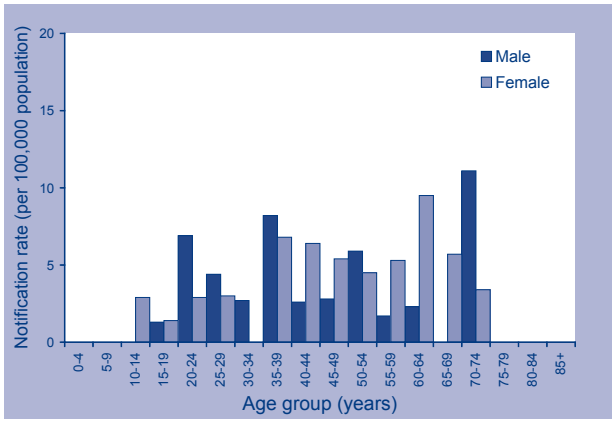


Figure 15. Notification rate for Barmah Forest virus infections, Northern Territory, 1 July 2004 to 30 June 2005, by age group and sex



Northern Territory

The sentinel chicken program in the Northern Territory commenced in January 1992 and replaced an earlier program run by the Australian Quarantine and Inspection Service (AQIS). Sentinel chicken flocks in the Northern Territory are maintained, bled and analysed for flavivirus in a combined program between the Northern Territory Department of Health and Community Services, the University of Western Australia, the Health Department of Western Australia, the Northern Territory Department of Business Industry and Resource Development (DBIRD), and volunteers.

The Map shows the sentinel chicken flocks are presently at Darwin urban (Leanyer), Darwin rural (Howard Springs), Adelaide River (Coastal Plains Research Station), Kakadu (Jabiru), Katherine, Nhulunbuy, Tennant Creek and Alice Springs (Ilparpa and Arid Zone research station). DBIRD officers or volunteers usually bleed flocks once a month and the samples are sent to the University of Western Australia for specific testing for MVEV and KUNV. Sometimes for operational reasons, chickens are not bled during a scheduled month and hence seroconversion shown in the next bleed could have occurred in the previous month. When chickens from a flock show new antibodies to MVEV during a prime risk period, a media warning is issued for the region for the risk period. These warnings advise residents of the need to take added precautions to avoid mosquito bites.

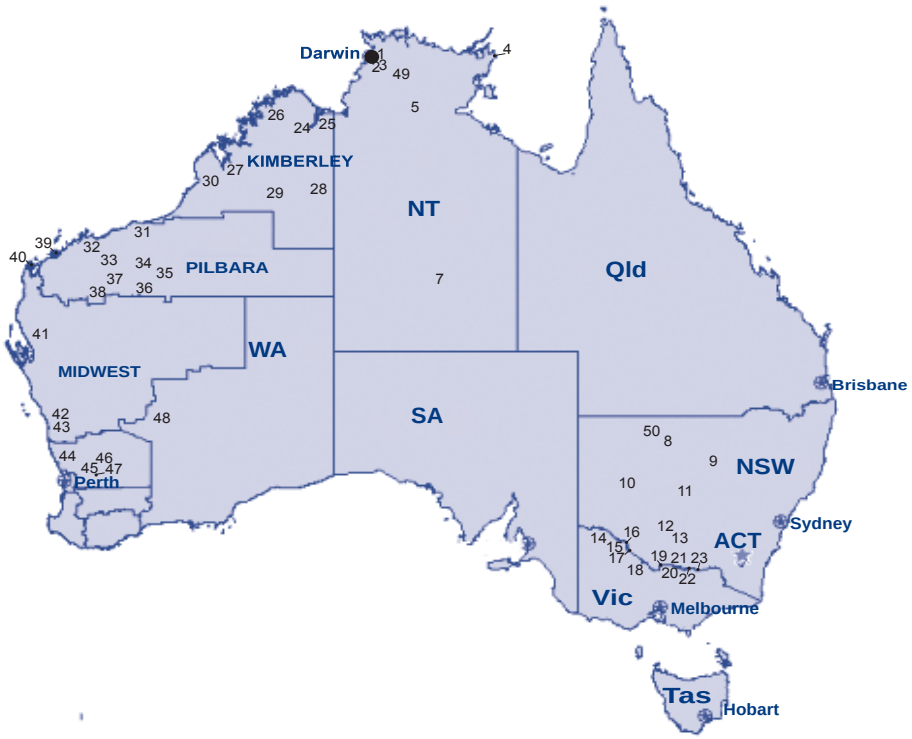
Chickens are replaced at least annually and more frequently if birds die or if a large proportion seroconvert. They are well positioned to detect flavivirus activity near the principal towns of the Northern Territory and hence provide timely and accurate indication of risk to people in those towns.

In the 2004–05 season, MVEV activity was detected in Katherine in January, February and June (probably May seroconversion), Tennant Creek in March (probably January or February seroconversion, Jabiru in April and June (probably May seroconversion), Nhulunbuy in April and May, and Darwin (Howard Springs) and Adelaide River in June. This reflects historical patterns of MVEV seroconversions usually starting in the more inland regions early in the wet season and soon after in the coastal/sub-coastal regions further north.

The MVEV total seroconversions this year (n=13) was considerably more than the last two years (n=2 and n=1), with most seroconversion this year (n=4) occurring in the Katherine flock, followed by the Jabiru flock (n=3). Most seroconversion this year occurred in June (n=6) while the long-term seroconversion peak occurred in May closely followed by March and then February.

There were no seroconversions in the two Alice Springs flocks, most probably due to the below average summer rainfall and low vector numbers. In addition, the successful effluent swamp drainage and better effluent management from nearby sew-

Map. Sentinel chicken testing sites, Australia, 2004–05



Northern Territory

1. Howard Springs
2. Beatrice Hill Farm
3. Leanyer
4. Gove
5. Katherine
6. Tennant Creek
7. Alice Springs (2)
49. Jabiru

New South Wales

8. Bourke
9. Macquarie Marshes
10. Menindee
11. Lake Cargellico
12. Griffith
13. Leeton
50. Wanaaring

Victoria

14. Mildura
15. Robinvale
16. Tooleybuc
17. Swan Hill
18. Kerang
19. Barmah
20. Toolamba
21. Barooga
22. Retherglen
23. Wodonga

Western Australia

24. Wyndham
25. Kununurra
26. Kalumburu
27. Derby (2)
28. Halls Creek
29. Fitzroy Crossing
30. Broome (2)
31. Port Hedland
32. Karratha
33. Harding Dam (2)
34. Marble Bar
35. Nullagine
36. Newman (2)
37. Tom Price
38. Paraburdoo
39. Onslow
40. Exmouth
41. Carnarvon
42. Geraldton
43. Dongara
44. Three Springs
45. Gingin
46. York
47. Bindoon
48. Leonora

age facilities in the Ilparpa area led to an overall reduction in vector numbers near the Alice Springs outskirts during summer.

The seroconversion in Tennant Creek was in a year of low rainfall and reinforces previous findings that MVEV is endemic in the Northern Territory as far south as Tennant Creek.

One human case of MVEV disease was reported (Table 2) in a 3-year-old boy from a community in Arnhem Land who was transferred to Royal Darwin Hospital for treatment. The boy had a relatively mild illness and made a complete recovery. The boy's community was located near an extensive freshwater wetland with numerous water birds and frequently high numbers of common banded mosquitoes *Culex annulirostris* and *Culex palpalis*, two vectors of MVEV.

KUNV activity appeared to be restricted to coastal sites around the Top End of the Northern Territory, with seroconversion to KUNV in Darwin (Howard Springs) in January, May (possible April seroconversion), in Darwin (Leanyer) in March and May (possible April seroconversion) and Nhulunbuy in May. The seroconversions in Darwin (Leanyer) in August and September were probably a continuation of activity from the previous year, but could also reflect a perennial focus of activity in the nearby permanent wetland. The lack of activity in the Top End from the Adelaide River flock was surprising, as the sentinel flock is within five kilometres of a large wetland with water birds. However the flock is also near cattle that could be acting as vector diversions.

There has been a trend over the last 10 years to increasing numbers of seroconversions to KUNV, with this year's total (n=12) higher than last year (n=9) and the highest since the program started in 1992. Most seroconversions occurred in the Leanyer (n=5) and Nhulunbuy (n=5) flocks, which were the highest number for these flocks since surveillance commenced. Most seroconversions occurred this year in May (n=7), while the long term peak was also observed in May, followed by a substantially reduced level in April.

The KUNV activity this year was considerably less than the activity of MVEV, and probably reflects the different vector and virus ecologies of the two arboviruses. The Northern Territory did not report any human cases of KUNV infection this year. The last reported KUNV case from the Northern Territory was in a 23-year-old female from Alice Springs in May 2001.

Western Australia

The Map shows the location of the 31 Western Australian sentinel chicken flocks. Flocks are located in the Kimberley, Pilbara, Gascoyne, Goldfields and Midwest regions in the north and central areas of the state. Environmental Health Officers or trained volunteers usually bleed flocks once a fortnight from December to May (the major MVEV 'risk' season) and monthly at other times. Samples are tested for specific antibodies to MVEV and KUNV by the Arbovirus Surveillance Laboratory at the University of Western Australia. Sometimes for operational reasons, chickens are not bled fortnightly and hence a seroconversion shown in the next bleed could have occurred earlier. Media warnings are issued by

Table 2. Number and rate of flavivirus notifications, 1 July 2004 to 30 June 2005, Australia, by jurisdiction

Jurisdiction	DENV		Flavi NEC		KUNV		MVEV	
	Notifications	Rate*	Notifications	Rate*	Notifications	Rate*	Notifications	Rate*
ACT	2	NA	0	NA	0	0	0	0
NSW	33	NA	1	NA	0	0	0	0
NT	16	NA	0	NA	0	0	1	0.5
Qld	113	NA	19	NA	3	0.08	1	0.03
SA	4	NA	0	NA	0	0	0	0
Tas	0	NA	0	NA	0	0	0	0
Vic	8	NA	0	NA	1	0.02	0	0
WA	12	NA	0	NA	0	0	0	0
Australia	188	NA	20	NA	4	0.02	2	0.01

* Rate per 100,000 population.

NA Not applicable, rates not calculated since most cases of dengue (outside Queensland) and flavivirus infections not elsewhere classified were acquired overseas or unknown country of acquisition.

the Western Australian Department of Health when chickens from a flock in a particular region first sero-convert to MVEV. Additional warnings are issued if high levels of MVEV activity are detected in other flocks in the same region or if activity is detected in a second region in the north of the state. These warnings advise residents of the need to take added precautions to avoid mosquito bites.

Average summer rainfall (December 2004 to March 2005) was recorded in most areas of the Kimberley region but Pilbara rainfall was generally below average for the season. MVEV activity in sentinel chickens was only detected at Kununurra (Map, north-east Kimberley) during the 2004–05 wet season. Heavy rain and flooding was recorded at Kununurra in March 2005 and there were a total of six MVEV seroconversions occurring from March to June 2005. No KUNV activity was detected in sentinel chickens. In contrast to previous years, flavivirus activity was not detected in the Pilbara region.

The Western Australian Department of Health issued health warnings to residents and visitors to the Kimberley region in March and May 2005 warning of the increased risk of MVEV infection. No human cases were reported from Western Australia (Table 2). The last reported KUNV case from Western Australia was in a 27-year-old female from Kununurra in May 2001. The last reported MVEV case from Western Australia was in March 2002, when infection in a 32-year-old male from Bunyip was notified.

New South Wales

Samples from sentinel chicken flocks were tested weekly for flavivirus antibodies in New South Wales from mid-November 2004 to April 2005. There were no seroconversions to MVEV or KUNV during this period. There were no human cases reported from New South Wales for either MVEV or KUNV. The last reported case of KUNV from New South Wales was notified in May 2001 from a 58-year-old female from Griffith. There have been no recorded cases of MVEV to date in NNDSS from New South Wales.

Victoria

Samples from sentinel chicken flocks were tested weekly for flavivirus antibodies from October 2004 to March 2005. No MVEV or KUNV activity was detected in this region. There were no human cases reported from Victoria for MVEV. In October 2004, a 35-year-old female was notified as having acquired KUNV infection. The person lived in metropolitan Melbourne, but a detailed investigation did not reveal any likely exposure within Victoria, nor was there any other evidence of KUNV activity. She had travelled extensively overseas and it is assumed that she acquired KUNV or a closely-related virus while overseas. The only other Victorian case of KUNV was notified in May 2001, from a 67-year-old male from the northern suburbs of Melbourne. There have been no recorded cases of MVEV from Victoria in NNDSS.

Queensland

There were no sentinel chicken flocks in Queensland during 2004–05 although flocks have been maintained in previous years. One case of MVEV was reported in a 30-year-old male from Normanton in March 2005 (Table 2). The previously last reported MVEV case from Queensland was in a 3-year-old boy from Mount Isa in 2001. Queensland reported three sporadic cases of KUNV infection in July 2004, December 2004, and February 2005.

Japanese encephalitis virus infections

There were no cases of Japanese encephalitis virus infections (JEV) during this reporting period. The last reported case was in February 2004, when Queensland notified that a 66-year-old male acquired JEV from Papua New Guinea. There have been nine other cases of JEV reported to NNDSS since 1995, although JEV was not nationally notifiable until 2001. Four of these notifications were reported in Torres Strait islanders from the Badu Island community. The other locally acquired JEV case was reported in a resident from the Cape York Peninsula, Queensland. The remaining four cases were reported as acquired from overseas countries.

AQIS, through the Northern Australia Quarantine Strategy (NAQS) program, conducted monitoring for JEV for the 2005 wet season using sentinel pigs at sites on Badu Island in Torres Strait and its northern peninsula area (NPA) site at Injinoo airport. The four sentinel pigs on Badu Island all sero-converted (based on results of testing at Queensland Health Scientific Services and the CSIRO Australian Animal Health Laboratory). JEV was also identified through the detection of viral RNA in a pool of culicine mosquitoes collected on Badu Island. This was collaborative mosquito trapping performed by NAQS for Queensland Health.

The five NPA sentinel pigs did not seroconvert and there was no evidence of transmission of JEV to the mainland in 2005.

Flavivirus infections not elsewhere classified

There were 20 flavivirus infections not elsewhere classified (NEC) notifications during the 2004–05 season, of which four were acquired overseas and two were locally acquired. The country of acquisition was unknown for the remaining 14 notifications.

Queensland reported 19 of the 20 flavivirus (NEC) notifications. Of these, there were four Kokobera and one Stratford virus. The 14 other flavivirus infections (NEC) were of unknown type. New South Wales reported the remaining one notification of flavivirus infection (NEC) which was of an unknown type.

Dengue virus infections

There were 188 notifications of DENV during the 2004–05 season. Table 2 shows that the cases were mainly from Queensland (n=113, 60%), New South Wales (n=33, 18%), the Northern Territory (n=16, 9%) and Western Australia (n=12, 6%). Figure 16 shows that the number of DENV notifications received during the 2004–05 season was much lower than the two previous seasons. There were two outbreaks caused by different genotypes of dengue type 4 during this season (Jeffery Hanna, Tropical Public Health Unit, personal communication). There was a peak in DENV notifications in March 2005 (n=50) which reflected an outbreak in the Torres Strait Islands. A separate smaller outbreak in Townsville involving 18 cases with an onset date in April 2005, is still ongoing as of November 2005.

Dengue notifications were reported as acquired from overseas in 86 of the 188 notifications. Locally acquired cases were reported by Queensland (n=21) and Western Australia (n=2), although the Western Australian cases were Western Australian residents who acquired DENV in Queensland (Gary Dowse, personal communication). The country of acquisition for the remaining notifications was either unknown or not stated (n=78).

Of the 188 notifications, dengue serotype 4 was reported in 32 per cent (n=60) of cases (Table 3). Serotype information was either not stated or unavailable for 54 per cent of the notifications (n=102).

Figure 17 shows that the largest number of notifications was reported in the 45–49 year male age group (n=17) and the 30–34 year female age group (n=13). Dengue virus infections notifications were higher for males than for females (102 cases: 86 cases).

Figure 16. Dengue notifications (locally acquired and imported cases), select jurisdictions, January 1998 to June 2005, by month and season of onset

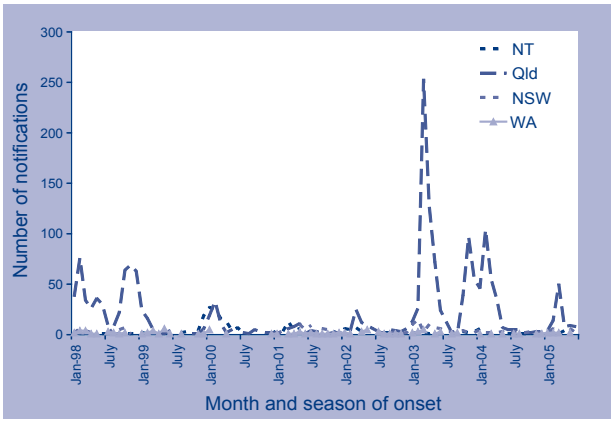


Figure 17. Dengue notifications (locally acquired and imported cases), Australia, 1 July 2004 to 30 June 2005, by age group and sex

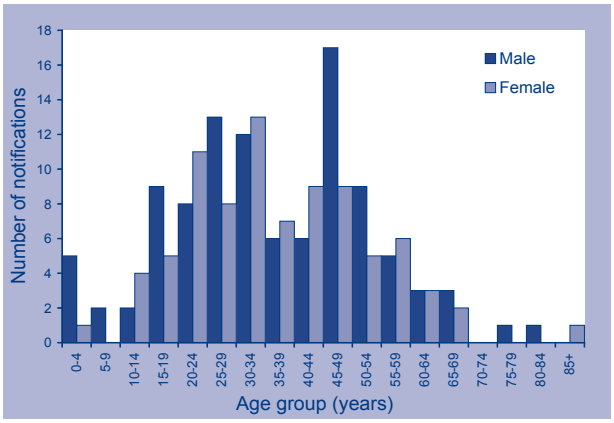


Table 3. Dengue notifications (locally acquired and imported cases), Australia, 1 July 2004 to 30 June 2005, by serotype

	Serotype					Total
	Not typed	Serotype 1	Serotype 2	Serotype 3	Serotype 4	
Notifications	102	8	10	8	60	188
Serotype (%)	54	4	5	4	32	100

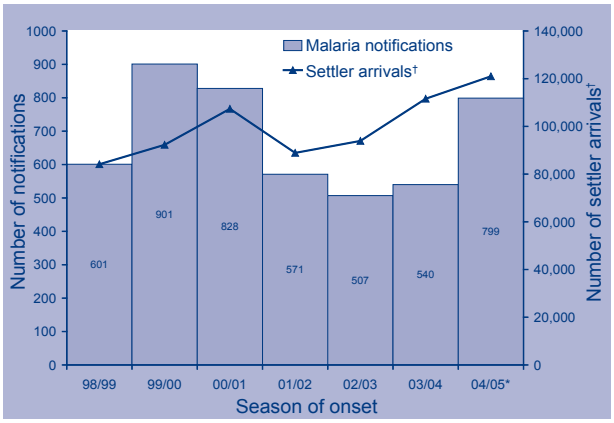
Malaria

There were 799 notifications of malaria in Australia in the period 1 July 2004 to 30 June 2005. Queensland reported the majority of cases (n=309, Table 4). There were no reports of locally acquired malaria during the reporting period.

Figure 18 shows that the 2004–05 season was the third highest reporting period for malaria notifications since 1998, and that malaria notifications may be associated with increases in settler arrivals from overseas countries. Previously published work has shown that of Victorian malaria notifications reported in 1999–2000, malaria was most often acquired in Papua New Guinea (36%) and East Timor (13%).[4] Data published on Western Australian notifications[5] from 1990–2001 has shown that a rise in falciparum malaria was reflected by an influx of Indonesian immigrants in detention and the deployment of Australian Defence personnel to East Timor. More recent surveillance status reports from New South Wales,[6] the Northern Territory,[7] Victoria[8] and Queensland have associated malaria notifications with immigrant arrivals from African countries. This has lead to the development of protocols in some jurisdictions for the screening of malaria parasites upon arrival for persons from high risk areas.[9]

Malaria notifications were reported to be highest in children in the 5–9 year age group (Figure 19), which is the first time that notifications in children have predominated. Overall, male notifications (n=473) were more common than female notifications (n=296). Males in the 20–24 year age group were the largest reported sex-specific cohort whereas the largest reported numbers of female cases were observed in the 5–9 year age group. Appendix 5 shows that since 1998, the highest number of cases has been in the young adult age groups of 20–24 and 25–29 years. These young adult age groups were also the most affected in Australia from 1991–1997.[10]

Figure 18. Number of notifications of malaria and DIMIA settler arrivals[2,3] from all overseas countries, Australia, 1998–2005, by season of onset



* Annualised settler arrivals based on July to December 2004 data.

† Settler arrivals from all overseas countries.

Table 4. Malaria notifications in Australia, 1 July 2004 to 30 June 2005, by parasite type and jurisdiction

Parasite type	Type (%)	Jurisdiction								
		ACT	NSW	NT	Qld	SA	Tas	Vic	WA	Australia
Plasmodium species	1	0	1	0	2	0	1	0	7	11
Plasmodium falciparum	57	3	131	37	159	31	16	34	46	457
Plasmodium malariae	1	1	1	0	5	0	0	1	1	9
Plasmodium ovale	3	1	6	1	9	1	0	4	5	27
Plasmodium vivax	34	8	53	14	134	2	7	51	2	271
Mixed infection (unspecified)*	0.4	0	0	0	-	0	0	0	3	3
Mixed *P. falciparum* and *P. vivax**	0.5	0	1	2	-	1	0	0	0	4
Mixed *P. falciparum* and *P. ovale**	1.0	0	6	0	-	0	0	5	0	11
Mixed *P. falciparum* and *P. malariae**	0.8	0	3	2	-	1	0	0	0	6
Total	100	13	202	56	309	36	24	95	64	799

* New South Wales, South Australia, Tasmania, Victoria and Western Australia report mixed species infections as one notification. Queensland, the Northern Territory and the Australian Capital Territory report one notification for each species in a mixed infection.

– Unknown

Figure 19. Number of notifications of malaria, Australia, 1 July 2004 to 30 June 2005, by age group and sex

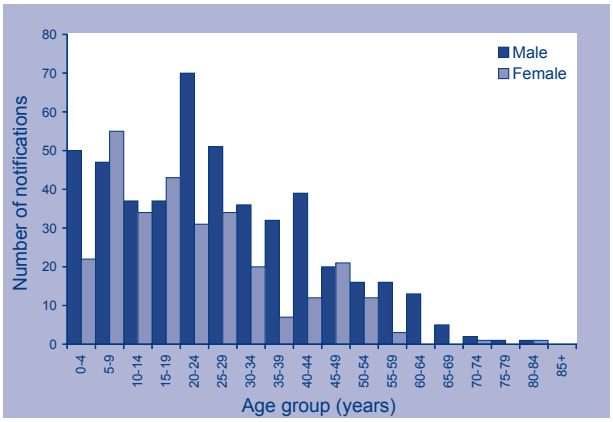
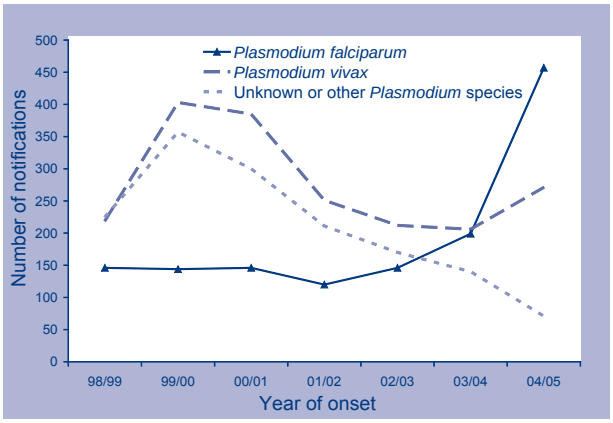


Table 4 shows that the infecting *Plasmodium* species were reported for more than 98 per cent of malaria notifications in 2004–05. Of these 799 notifications, more than half were *P. falciparum* (57%, n=457). *P. vivax* accounted for 34 per cent of cases (n=271) while untyped *Plasmodium* species accounted for one per cent (n=11). The remaining cases were *P. ovale* (3%, n=27) and *P. malariae* (1%, n=9). It should be noted that mixed infections (3%, n=24) are underestimated due to the variation in reporting practice in different states and territories.

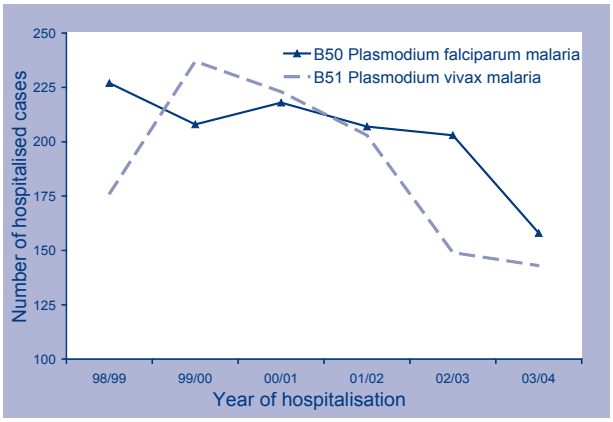
Figure 20 and Table 5 show that in 2004–05 the number of falciparum malaria notifications (n=457) in Australia increased significantly from previous years and was three times the five-year mean for the same species. This appears to be the first time[11] in which *P. falciparum* has become the predominant infecting species for malaria notifications in Australia, as previously published data in earlier years showed *P. vivax* accounting for 64–70 per cent of cases and *P. falciparum* 20–32 per cent of cases both nationally[10,12] and in individual states.[4,5]

Figure 20. Malaria notifications in Australia, by select species and year of onset



Australian malaria hospitalisation data[13] shows that falciparum malaria was the predominant species for malaria hospitalisations in 2002–03 and 2003–2004 (Figure 21).

Figure 21. Malaria hospitalisations in Australia, 1998 to 2004, by species and principal diagnosis (ICD10-AM)



Source: Australian Institute of Health and Welfare.

Table 5. Malaria notifications in Australia, by select species and year of onset

Infecting species	Year of onset							Last 5 year mean	Ratio 04/05 5 year mean
	98/99	99/00	00/01	01/02	02/03	03/04	04/05		
Plasmodium falciparum	146	144	146	120	146	199	457	151	3.0
Plasmodium vivax	219	403	385	251	212	206	271	291	0.9
Unknown or other *Plasmodium* species	225	357	300	211	170	140	71	236	0.3
Total	590	904	831	582	528	545	799		

Malaria mortality

There have been 12 deaths due to malaria reported in Australia, of which seven have been published.[12,14] Of these seven deaths, six were notified to the Australian Malaria Register, and one to NNDSS. The most recent deaths occurred in 2002 (a 26-year-old, New South Wales) and 2003 (a 1-year-old, Queensland). Table 6 shows the 12 deaths due to malaria, the infecting malaria species and the country of acquisition.

The Australian Institute of Health and Welfare (AIHW) National Mortality Database contains eight deaths for the years of registration 1998–2003 where falciparum malaria was listed as the underlying cause of death (AIHW, personal communication). *Plasmodium vivax* was listed as an associated cause of death for one of the falciparum malaria deaths most probably representing death resulting from a mixed infection of *P. falciparum* and *P. vivax* (for which there is no ICD10-AM code).

Acknowledgements

The National Arbovirus and Malaria Advisory Committee members are: *(in alphabetical order)* Annette Broom, Bart Currie, Peter Daniels, Mike Lindsay, John Mackenzie, Moira McKinnon, Rodney Moran, Aileen Plant, Scott Ritchie, Richard Russell, Linda Selvey, David Smith, James Walker, John Walker, Peter Whelan, Phil Wright (Secretariat).

In addition, the authors would like to thank:

Jenean Spencer, Paul Roche and Alison Milton, Australian Government Department of Health and Ageing

Tim Kerlin, Australian Quarantine and Inspection Service

Mark Bartlett, NSW Health Department

David Coleman, Tasmanian Department of Health and Human Services

Craig Davis, Queensland Health

Gary Dowse and Carolien Giele, Western Australia Department of Health

Peter Markey, Northern Territory Department of Health and Community Services

Robert Van der Hoek and Cherie Du, Australian Institute of Health and Welfare

Simon Forsyth, Australian Malaria Register

Table 6. Malaria deaths in Australia, 1992 to 2005, by year, species and country of acquisition

Year of death	Age at death (years)	Infecting species	Country of acquisition
1992	43	*Plasmodium falciparum*	Solomon Islands
1992	71	Plasmodium falciparum	Papua New Guinea
1992	21	*Plasmodium falciparum*	Papua New Guinea
1992	17	*Plasmodium falciparum*	Nigeria
1992	86	*Plasmodium falciparum*	Australia (blood transfusion)
1993	46	*Plasmodium vivax*	Indonesia
1995	58	Plasmodium falciparum	Indonesia
1995	58	*Plasmodium falciparum*	Kenya
1996	33	*Plasmodium falciparum*	Indonesia
2000	32	*P. falciparum* and *P. vivax*	East Timor
2002	26	*Plasmodium falciparum*	Indonesia
2003	1	Plasmodium vivax	Papua New Guinea

Source: National Notifiable Diseases Surveillance System, ICPMR, Australian Malaria Register, and States and Territories.

Appendix 1. Notifications and notification rates of mosquito-borne diseases, Australia, 1 July 1998 to 30 June 2005, by season of onset*

Mosquito-borne disease	Notifications							Rate per 100,000 population						
	98/99	99/00	00/01	01/02	02/03	03/04	04/05	98/99	99/00	00/01	01/02	02/03	03/04	04/05
Barmah Forest virus infection	618	556	1,134	943	1,299	967	1,256	3.3	2.9	5.9	4.8	6.6	4.8	6.2
Dengue virus infection	352	221	122	185	664	551	188	NA	NA	NA	NA	NA	NA	NA
Flavivirus infection NEC	104	66	32	68	40	89	20	NA	NA	NA	NA	NA	NA	NA
Japanese encephalitis virus infection	– 1	–			1	1	0	–	–	0	0	<0.1	<0.1	0
Kunjin virus infection	†2	†4	5		17	11	4	†	†	<0.1	0	<0.1	<0.1	<0.1
Malaria	590	904	831	582	528	545	799	NA	NA	NA	NA	NA	NA	NA
Murray Valley encephalitis virus infection	†	†16	5	3		1	2	†	†	<0.1	<0.1	0	<0.1	<0.1
Ross River virus infection	4,592	4,265	3,629	1,580	3,162	4,735	1,858	24.4	22.4	18.8	8.1	16.0	23.7	9.2
Total	6,259	6,032	5,758	3,361	5,711	6,900	4,127							

* 1 July to 30 June.

† Included in 'Arbovirus NEC' 1999–2000 except for the Northern Territory and Western Australia.

– Not notifiable in these years except in Western Australia.

NA Not applicable. Rates for dengue virus infection, flavivirus infection NEC and malaria were not calculated since the majority of cases were acquired overseas.

Appendix 2. Notifications of mosquito-borne diseases, Australia, 1 July 2004 to 30 June 2005, by month of onset

Mosquito-borne disease	Total	Jul-04	Aug-04	Sep-04	Oct-04	Nov-04	Dec-04	Jan-05	Feb-05	Mar-05	Apr-05	May-05	Jun-05
Barmah forest virus infection	1,256	47	66	79	80	109	85	93	131	135	166	168	97
Dengue virus infection	188	10	14	6	6	8	5	12	28	56	13	18	12
Flavivirus infection NEC	20	4	1	1	0	3	2	1	0	4	0	2	2
Japanese encephalitis virus infection	0	0	0	0	0	0	0	0	0	0	0	0	0
Kunjin virus infection	4	1	0	0	1	0	1	0	1	0	0	0	0
Malaria	799	50	46	40	53	49	47	66	110	171	57	63	47
Murray Valley encephalitis virus infection	2	0	0	0	0	0	0	0	0	2	0	0	0
Ross River virus infection	1,858	54	51	47	62	89	87	143	304	469	312	155	85

Appendix 3. Notification rates* for Ross River virus infections, select jurisdictions, 1 July 2004 to 30 June 2005, by age group and sex

Age group	State or territory														
	NSW			NT			Qld			WA			Australia		
	Male	Female	Persons	Male	Female	Persons	Male	Female	Persons	Male	Female	Persons	Male	Female	Persons
0–4	1	0	0.2	0	11.7	5.7	0.8	2.5	1.6	0	0	0	0.3	0.6	0.5
5–9	0	0	0.2	0	0	0	3.6	2.3	3.0	0	0	0	1.0	0.6	0.8
10–14	0	0.9	0.4	11.8	50.9	30.7	4.2	3.7	3.9	0	4.4	2.1	1.0	2.2	1.6
15–19	2.2	4.5	3.3	13.0	114.6	61.4	13.6	16.5	15.0	5.4	2.8	4.1	4.1	6.4	5.2
20–24	3.4	5.8	4.6	23.2	96.5	56.7	19.0	23.6	21.3	2.8	4.4	3.5	5.7	8.6	7.1
25–29	5.2	4.8	5.0	92.5	111.1	101.5	35.3	24.7	30.0	3.0	7.6	5.3	10.1	8.7	9.4
30–34	3.1	8.9	6.0	150.5	165	157.7	29.7	26.6	28.1	12.1	10.9	11.5	10.2	12.3	11.3
35–39	11.2	9.1	10.1	114.5	138.7	126	44.4	58.0	51.3	6.8	10.9	8.9	14.6	17.7	16.2
40–44	5.8	9.0	7.4	161.8	119.2	142	49.2	42.7	45.9	15.5	23.2	19.3	15.4	15.2	15.3
45–49	14.8	11.7	13.5	112.1	299	202.6	41.2	47.8	44.5	12.4	10.9	11.6	16.4	18.3	17.4
50–54	14.7	6.4	10.6	162.9	155.5	159.5	44.0	28.2	36.1	11.9	10.5	11.2	17.6	10.4	14.0
55–59	10.9	11.6	11.5	19.1	167.5	84.9	39.1	49.9	44.4	6.6	7.0	6.8	13.7	16.8	15.3
60–64	13.0	8.6	10.8	0	75.3	32.2	36.6	35.9	36.3	9.1	4.7	7.0	13.7	12.8	13.2
65–69	8.8	7.7	8.6	97.6	0	55.8	21.4	20.4	20.9	8.6	8.6	8.6	9.3	7.9	8.7
70–74	12.5	7.0	9.6	82.8	99	90.2	23.6	12.2	17.8	3.7	0.0	1.8	9.6	5.2	7.3
75–79	5.8	3.8	4.7	123.0	139.5	130.7	11.4	11.6	11.5	14.1	3.9	8.6	6.7	4.0	5.2
80–84	7.3	3.7	5.1	0	0	0	10.9	5.1	7.5	0.0	5.2	3.1	5.5	3.2	4.1
85+	3	0	1.0	0	0	0	17.5	2.9	7.8	0	0	0	4.4	0.5	1.7

* Age and sex specific rates by cases per 100,000 population.

Appendix 4. Notification rates* for Barmah Forest virus infections, select jurisdictions, 1 July 2004 to 30 June 2005, by age group and sex

Age group	State or territory														
	NSW			NT			Qld			WA			Australia		
	Male	Female	Persons	Male	Female	Persons	Male	Female	Persons	Male	Female	Persons	Male	Female	Persons
0–4	0	0.5	0.2	0	0	0	0.8	0	0.4	0	0	0	0.2	0.2	0.2
5–9	0	0.5	0.2	0	0	0	0.7	0.8	0.7	0	0	0	0.1	0.3	0.2
10–14	0.8	0.4	0.7	0.1	0	0.1	4.2	6.6	5.4	0	2.9	1.4	1.3	1.8	1.5
15–19	5.2	6.3	5.7	0	0	0	8.6	12.7	10.6	1.3	1.4	1.4	3.7	4.9	4.3
20–24	3.8	2.2	3.0	0.1	0.4	0.3	12.0	17.0	14.4	6.9	2.9	5.0	4.6	5.0	4.8
25–29	2.6	4.4	3.5	0	0.3	0.1	21.5	18.6	20.0	4.4	3.0	3.8	5.4	5.6	5.5
30–34	6.7	7.0	6.8	0.1	0	0.1	28.3	18.4	23.3	2.7	0	1.4	8.5	6.1	7.3
35–39	8.3	10.3	9.7	0.3	0.1	0.2	34.9	17.7	26.2	8.2	6.8	7.5	10.6	8.2	9.5
40–44	8.1	8.6	8.3	0.5	0.3	0.4	32.8	32.0	32.4	2.6	6.4	4.5	10.0	10.1	10.1
45–49	14.8	12.6	13.7	0.4	0.6	0.5	29.4	25.4	27.4	2.8	5.4	4.1	12.1	10.8	11.4
50–54	16.5	10.5	13.5	0.3	0.9	0.6	27.5	23.5	25.5	5.9	4.5	5.2	12.1	9.6	10.8
55–59	9.4	6.1	7.8	0.9	0.2	0.5	25.0	30.1	27.5	1.7	5.3	3.4	9.9	9.5	9.7
60–64	9.1	11.9	10.5	0.2	0.5	0.3	25.5	13.9	19.8	2.3	9.5	5.8	9.1	9.3	9.2
65–69	10.3	6.9	8.6	0.3	0.0	0.1	22.8	14.5	18.7	0	5.7	2.9	8.5	6.0	7.2
70–74	12.5	8.8	10.6	0	0.3	0.2	18.2	14.0	16.0	11.1	3.4	7.1	9.6	6.1	7.8
75–79	3.5	2.8	3.1	0	0	0	4.6	7.7	6.3	0	0	0	2.5	2.3	2.4
80–84	11.0	1.2	5.1	0	0	0	7.2	10.2	9.0	0	0	0	6.1	2.3	3.8
85+	0	2.8	1.9	0	0	0	5.8	5.8	5.8	0	0	0	1.1	2.0	1.7

* Age and sex specific rates by cases per 100,000 population.

Appendix 5. Malaria notifications, Australia, 1 July 1998 to 30 June 2005, by age group and sex

Age group	Year of onset																				
	1998–1999			1999–2000			2000–2001			2001–2002			2002–2003			2003–2004			2004–2005		
	M	F	P	M	F	P	M	F	P	M	F	P	M	F	P	M	F	P	M	F	P
0–4	16	11	27	7	7	14	8	10	18	5	5	10	7	18	25	14	11	25	50	22	72
5–9	13	8	21	13	16	29	8	6	14	10	15	25	16	17	33	15	16	31	47	55	102
10–14	15	7	22	19	22	41	18	7	25	11	12	23	12	6	18	24	14	38	37	34	71
15–19	42	32	74	58	18	76	41	21	62	36	16	52	34	10	44	32	10	42	37	43	80
20–24	47	22	69	151	27	178	144	21	165	51	22	73	50	13	63	55	17	72	70	31	101
25–29	59	24	83	127	19	146	109	34	143	47	16	63	41	36	77	41	20	61	51	34	85
30–34	47	17	64	87	16	103	92	26	118	53	18	71	33	14	47	28	26	54	36	20	56
35–39	43	19	62	69	15	84	51	23	74	38	24	62	22	10	32	36	7	43	32	7	39
40–44	33	10	43	59	9	68	43	24	67	38	15	53	29	18	47	25	18	43	39	12	51
45–49	26	6	32	31	19	50	35	14	49	29	16	45	23	10	33	22	10	32	20	21	41
50–54	21	8	29	36	11	47	22	11	33	24	11	35	20	6	26	15	4	19	16	12	28
55–59	15	4	19	12	5	17	16	6	22	25	5	30	22		22	23	7	30	16	3	19
60–64	11	1	12	12	3	15	11	4	15	13	2	15	9	8	17	14	4	18	13	0	13
65–69	6	4	10	5	2	7	9	1	10	3	2	5	4	1	5	3	2	5	5	0	5
70–74	2	3	5	4	1	5	2	1	3	2	1	3	5	0	5	2	1	3	2	1	3
75–79	1	0	1	2	1	3	0	0	0	3	0	3	0	0	0	0	0	0	1	0	1
80-84	0	0	0	1	1	2	0	0	0	1	0	1	1	0	1	1	0	1	1	1	2
85+	1	0	1	1	0	1	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0
Total	398	176	574	694	192	886	609	209	818	389	180	569	328	167	495	350	167	517	473	296	769
Excluded*	16			18			13			13			33			28			30		

* Notifications for which date of birth, or sex, or date of birth and sex was not supplied.

M Male

F Female

P Persons

References

1. Broom AK, Azuolas J, Hueston L, Mackenzie JS, Melville L, Smith DW*, et al.* Australian encephalitis: Sentinel Chicken Surveillance Programme*. Commun Dis Intell* 2001;25:157–160.

2. DIMIA Settler Arrivals: Selected Countries of Birth by Migration Stream, for the Financial Year 2002–03. Available from: http://www.immi.gov.au/statistics/stat_info/oad/settlers/setdatb.htm Accessed on 24 August 2005.

3. Australian Government Department of Immigration, Multicultural and Indigenous Affairs. Immigration Update July – December 2004. 2004. Available from: http://www.immi.gov.au/statistics/publications/immigration_update/update-dec-04.pdf Accessed on 24 August 2005.

4. Skull SA, Tallis G. Epidemiology of malaria in Victoria 1999–2000: East Timor emerges as a new source of disease*. Commun Dis Intell* 2001;25:149–151.

5. Charles DM, Hart J, Davis WA, Sullivan E, Dowse GK, Davis TM. Notifications of imported malaria in Western Australia, 1990–2001: incidence, associated factors and chemoprophylaxis*. Med J Aust* 2005;182:164–167.

6. Walker J, Taylor R, Figtree M. Imported malaria notified in New South Wales and the Australian Capital Territory, including trends in notifications of *Plasmodium falciparum*, 1989 to 2003*. N S W Public Health Bull* 2005;16:89–91.

7. Zweck N, Anstey N, Currie B. Guidelines for screening and management of infectious diseases in refugees from sub-Saharan Africa – based on a single initial blood sample after arrival in the Northern Territory. *Northern Territory Disease Control Bulletin* 2004;11:13–17.

8. Victorian Department of Human Services. Victorian Infectious Diseases Bulletin. 2005;8. Available from: http://www.health.vic.gov.au/ideas/downloads/bulletin/vidbv8i1.pdf Accessed on 24 August 2005.

9. Malaria Protocol. Guidelines For Health Professionals in the Northern Territory. 4th edition. *Northern Territory Disease Control Bulletin* 2004;11:6–17.

10. Harvey B. Trends in malaria in Australia, 1991–1997. *Commun Dis Intell* 1998;22:247–248.

11. Communicable diseases surveillance: highlights for 1st quarter, 2005*. Commun Dis Intell* 2005;29:206–225.

12. Bryan J, Fa'afoi E, Forsyth S. Report of the Australian Malaria Register for 1992 and 1993*. Commun Dis Intell* 1998;22:237–245

13. Principal diagnosis data cubes, National Hospital Morbidity Database. 1998–99 to 2003–04. Available from: http://www.aihw.gov.au/hospitals/datacubes/datacube_pdx.cfm#pdc Accessed on 2 September 2005.

14. Davis TM, Breheny FX, Kendall PA, Daly F, Batty KT, Singh A*, et al.* Severe *falciparum* malaria with hyperparasitaemia treated with intravenous artesunate*. Med J Aust* 1997;166:416–418.

Guidelines for the use and interpretation of nucleic acid detection tests for *Neisseria gonorrhoeae* in Australia: A position paper on behalf of the Public Health Laboratory Network

David W Smith,[1] John W Tapsall,[2] Gary Lum[3]

Summary

The Public Health Laboratory Network (PHLN) convened a workshop of Australian experts in Melbourne on 23 March 2005 to identify laboratory issues of relevance and suggest guidelines for use of nucleic acid detection tests (NADT) for diagnosis of gonorrhoea in Australia. The proceedings of that meeting were endorsed by the members of the PHLN and the Communicable Diseases Network of Australia.

Given the present state of knowledge and experience of conditions currently existing in Australia, the following recommendations were made:

Recommendation 1: Assays using detection of the *cppB* gene should not be used for either screening or supplemental assays.

Recommendation 2: All in-house screening assays that are positive should also be positive on a reliable supplemental assay before a positive result is reported.

Recommendation 3: All commercial screening assays that are positive should also be positive on a reliable supplemental assay before a positive result is reported.

Recommendation 4: If a sample is positive in a screening assay but a suitable supplemental assay is negative, then the result should be reported as negative.

Recommendation 5: Laboratories should ensure that the test combination they use would yield a positive predictive value of at least 90 per cent in a population with a prevalence of 1 per cent.

Recommendation 6: For the purposes of test evaluation, as distinct from diagnostic testing, true positives be defined by meeting one or more of the following criteria:

1) culture positive using contemporary isolation and identification techniques;

2) positive result on NADTs directed to targets on three separate genes that are known to have discriminatory capacity;

3) sequencing of a gene known to separate gonococcal from non-gonococcal species.

Recommendation 7: Inhibitor controls should be routinely included in all NADT.

Recommendation 8: Cultures are the preferred test for samples from non-genital sites. If however it is necessary to perform a NADT, then more stringent criteria should be applied, and positive samples should meet the 'test evaluation' criteria for a 'true positive'.

1. Clinical Director, Microbiology and Infectious Diseases, PathWest Laboratory Medicine WA, Nedlands, Western Australia
2. Director, WHO Collaborating Centre for STD and HIV, Microbiology Department, The Prince of Wales Hospital, Randwick New South Wales
3. Director, Pathology Department, Northern Territory Government Pathology Service, Tiwi, Northern Territory

Corresponding author: Dr David W Smith, PathWest Laboratory Medicine WA, Locked Bag 2009, Nedlands, WA 6909. Telephone: +61 8 9346 3122. Facsimile: +61 8 9346 3960. Email: david.smith@health.wa.gov.au

Recommendation 9: In order to properly assess the routine diagnostic system in Australia, the following quality assurance samples should be distributed in addition to the routine samples currently used:

1. *cppB* negative *N. gonorrhoeae;*
2. Non-gonococcal species known to cause false positive reactions: these should be dispatched both as a single species, as well as mixture with *N. gonorrhoeae.* In the latter circumstance, the non-gonococcal species should be present in 10-fold excess;
3. Urine samples: preferably a single patient sample, otherwise a spiked sample.
4. Validation panels should be made that include samples that are culture positive but PCR negative. True positive samples should also be made available.

In addition, a process should be established for full phenotypic and genotypic characterisation of unidentified species that yield false positive results in NADT for gonococci.

Recommendation 10: Strategies should be put in place to ensure that sufficient numbers of gonococcal isolates are obtained to allow reliable monitoring of antimicrobial resistance.

Recommendation 11: Public health practitioners need to define the relevant populations that need to be targeted and identify any that require enhanced surveillance.

Background

In recent years there has been a significant change in the approach to the diagnosis of gonorrhoea. Traditional culture methods for isolation of *Neisseria gonorrhoeae* have been increasingly replaced by nucleic acid detection tests, especially in remote areas of Australia. This has occurred for a number of reasons, in particular the increased robustness and the improved sensitivity for testing of patents outside hospital clinics. In general NADT have performed well, especially as reliable and sensitive tests for the exclusion of gonococcal infection.

However, after increasing experience with these tests, some problems have been identified with NADT. The most widely used commercial assay, the Cobas Amplicor, was found to produce a large number of false positive results[1] and it was realised that the target sequence in the cytosine DNA methyltransferase (CMT) gene of *N. gonorrhoeae* was also present in some strains of other *Neisseria* species, including *N. cinerea* and *N. subflava*. These are normal flora in the upper respiratory tract, and may also be present in the genital tract. It is still not clear whether this is the sole mechanism of false positive results with this assay.

Subsequently, a number of supplemental assays have been developed and used in Australia. The initial ones were directed at targets in the 16S RNA and the *cppB* genes. Neither of these targets was, in themselves, fully specific but the combination of two assays substantially improved specificity. However, some clearly problematic results remained, with some false positives still occurring in genital tract and urine samples, and more commonly in results with throat swabs, irrespective of the assay combination used.

In 2002 the Public Health Laboratory Network was asked by the Communicable Disease Network of Australia to provide an opinion on the Cobas Amplicor test. PHLN recommended that it should only be used for urine or genital tract samples (or, cautiously, for normally sterile specimens, such as joint fluids) unless validated for other samples. They also recommended that a screening assay reactive on the Cobas Amplicor should not be reported as positive unless there was also at least one positive supplemental PCR assay or a positive culture. Furthermore, screening and supplemental assays should be directed at a different targets selected from *cppB,* 16S rRNA and CMT genes. Since 2002, a number of Australian laboratories have evaluated further a number of existing assays and devised alternative methods for improving the specificity of gonococcal NADT.

In addition, it was recognised that the shift to NADT means that there may not be sufficient gonococcal cultures to provide enough isolates to properly monitor antimicrobial susceptibility testing. There are not currently any NADT–based methods for reliably determining antimicrobial resistance in gonococci. It is therefore essential that pragmatic strategies be implemented that guarantee that sufficient isolates, representative of the relevant Australian populations, are available for antimicrobial resistance surveillance in gonococci, despite the shift to NADT.

A meeting of laboratory experts (see Appendix 1 for attendees) was convened by the PHLN in Melbourne on 23 March 2005 to identify laboratory issues of relevance and suggest guidelines for use of NADT for diagnosis of gonorrhoea in Australia. A review of the currently available NADT for gonococci and their potential (or actual) roles as screening and/or supplemental assays was undertaken in order to establish estimates of the performance characteristics of different screening/supplemental combinations in symptomatic, asymptomatic, urban and remote area populations. The meeting also discussed the quality assurance and quality control requirements for these tests. Recommendations were also generated

for the surveillance of antimicrobial susceptibility, with particular reference to strategies for obtaining the requisite number of isolates.

Recent developments in in-house NADT for *Neisseria gonorrhoeae* in Australia

Since 2002, a number of Australian laboratories have evaluated further a number of existing assays and devised alternative methods for improving the specificity of gonococcal NADT.

cppB gene based assays

Assays using *cppB* gene targets have been used widely as both screening and supplemental assays. In addition to the specificity problems mentioned above, there has been recent emergence of a *cppB* gene negative subtype of *N. gonorrhoeae.* As a result assays that target *cppB* gene may fail to detect up to 10 per cent of cases if used as a screening assay[2] and may fail to confirm some true positive results in other screening assays.[3]

porA pseudogene assay

A real-time PCR directed at the *porA* pseudogene was developed in Queensland[4] and was shown to reliably discriminate between gonococci and a large range of non-*Neisseriae* and non-gonococcal *Neisseriae.* It was equivalent in sensitivity to the Cobas Amplicor and more sensitive than culture, particularly in samples with low organism numbers. The additional positives using the *porA* pseudogene were mainly in throat or rectal swabs. All were shown to be true positives by discrepant analysis. A multi centre evaluation of this assay is continuing.

opa gene assay

A real-time PCR directed at a 90 bp target within a highly-conserved region of the *opa* gene, which is present in multiple copies has been developed in Melbourne.[5] Like the porA pseudogene assay, it showed excellent discrimination between gonococci (including those that were negative in the *cppB* gene-based assay) and a large range of non-*Neisseriae* and non-gonococcal *Neisseriae.* When compared with a range of other NADT for gonococci, the *opa* gene assay was as sensitive, and with high specificity.

CMT gene assay

A group from Perth reported on their experience with a range of CMT and *cppB* gene targets (Harnett G, personal communication). The best combinations using nested PCRs showed a significant false positive rate of up to 10 per cent for genital tract samples and a very high false positive rate for throat swabs. Subsequently a more specific sequence was identified that showed excellent discrimination between gonococci and non-gonococcal *Neisseria* in a real time PCR. Evaluation of over 7,000 specimens demonstrated a sensitivity equivalent to the nested PCRs but with a specificity of 99.8 per cent. Some of the few false positives had a single base pair difference from *N. gonorrhoeae* that did not reduce the probe binding sufficiently to give negative result. Further refinements are underway.

Recent changes in commercial NADT in Australia

A number of commercial assays have been withdrawn because of problems noted only after extended use. The Roche 16S RNA assay and the Abbott LCR assay, both of which were previously used in Australia, were no longer available at the time of this meeting.

Strand displacement amplification

Becton Dickinson developed a strand displacement amplification assay, the SD ProbeTec, directed to a pilin gene target, which is claimed to have high sensitivity and specificity. However, published data indicate that this assay has a false positive rate of 67 per cent for weakly reactive samples and 3.3 per cent for strongly reactive samples.[6] Other studies also suggest difficulties with this assay,[7,8] if performed in isolation.

Transcription mediated amplification

The APTIMA Combo 2 uses transcription-mediated amplification (TMA) to amplify rRNA. It is claimed to have high sensitivity and specificity,[9,10] though concerns were expressed by participants that the supplemental assay used in the published evaluation was an earlier version of the same assay, and therefore may not be a suitable as a discriminator because of the risk that it would yield the same false positives as the primary assay.

Neither the SD ProbeTec nor the APTIMA Combo 2 has yet been properly evaluated in Australian populations.

Funding for gonococcal testing

The meeting also recognised that current funding for gonococcal nucleic acid testing does not allow for supplemental assays. There is a need to address this if supplemental assays are to become standard practice.

Antimicrobial susceptibility surveillance

Accurate and timely diagnosis, if accompanied by effective treatment, has been shown to make a significant contribution to the control of gonococcal disease.[11] High and increasing rates of antimicrobial resistance in *N. gonorrhoeae* have reduced the treatment options and limited the effectiveness of gonococcal control programs in Australia.[12]

It is therefore most important that surveillance of antimicrobial resistance in gonococci, currently performed by the National Neisseria Network, continues. This requires examination of viable cultures of gonococci and sample numbers must be sufficiently large and be adequately representative of gonococci and of human populations.[12] Currently it is recommended that standard treatment schedules should be changed when the proportion of resistant isolates reaches 5 per cent. Therefore sample size should be sufficient to reliably detect proportional resistance rates around 5 per cent.

Workshop discussion and recommendations

The Workshop discussed the above issues and considered *inter alia* approaches recommended by the US Centers for Disease Control and Prevention (CDC)[14] for use of NADT in the USA. The CDC recommendations for suggested testing algorithms for NADT for gonococci may be paraphrased as follows:

(i) repeat testing of the same sample with the same assay;

(ii) repeat testing of the same sample with a different NADT (presumably with a different target sequence);

(iii) repeat testing of a repeat sample with the original NADT;

(iv) repeat testing of a repeat sample with a second NADT (presumably with a different target sequence).

The Working Group agreed that option (i) was not sufficiently stringent, while options (iii) and (iv) were impractical for Australian conditions and logistics.

Discussion was therefore focussed on option (ii) and the characteristics required for screening and supplemental NADT for gonococcal detection in Australia given the current state of knowledge. It was agreed that the terms 'screening assay' and 'supplemental assay' be used and that the term 'confirmatory assay' was misleading as no assay could truly confirm identity.

The Workshop noted that defects in both commercial and in-house assays became apparent despite initial favourable evaluations, and that in some instances those commercial NADT initially had US FDA approval. The defects became apparent only after more extensive testing of different gonococcal populations and as testing experience accumulated. This phenomenon is consistent with the known characteristics of *N. gonorrhoeae*, especially its capacity for genetic recombination through horizontal genetic exchange.[8,15–19] This capacity for continuing genetic diversification in gonococci means that regular and wide ranging appraisals of NADT are prudent. In the absence of these continuing appraisals at the present time, as well as the logistic problems referred to above, the participants agreed that option (ii) should be used as the basis for test algorithms for NADT for gonococci in Australia.

Screening assays

The requisite features of a screening NADT are that it should have a high sensitivity and thus a high negative predictive value. It is recognised that it has proved difficult to develop a single NADT assay that is both highly sensitive and highly specific, though tests are continuing to improve. Nevertheless it is desirable for screening NADT to be as specific as possible while retaining their sensitivity and negative predictive value.

In house assays directed at *cppB* targets should be used very cautiously as a screening or supplemental assay due to the presence of *cppB* gene negative strains in some Australian populations.[2] In addition, *cppB* targets are less specific than alternative targets, so that there is no imperative to maintain it for confirmatory purposes.

Recommendation 1: Assays using detection of the cppB gene should not be used for either screening or supplemental assays.

While the other in-house assays that have been described above are substantial improvements, workshop participants did not feel that there are as yet sufficient available data, local or otherwise, to recommend their use as a single assay. Therefore for the in-house NADT a supplementary assay should be performed on the original sample unless a positive culture has been obtained from the same site at the same time.

Recommendation 2: All in-house screening assays that are positive should also be positive on a reliable supplemental assay before a positive result is reported.

All of the commercial assays appear to have high sensitivity and a good negative predictive value. However the Amplicor and ProbeTec assays are known to have problems with false positives reactions, while current Australian data on the APTIMA Combo 2 NADT are inadequate to be confident of specificity. Therefore for the commercial NADT a supplementary assay should be performed on the original sample unless a positive culture has been obtained from the same site at the same time.

Recommendation 3: All commercial screening assays that are positive should also be positive on a reliable supplemental assay before a positive result is reported.

Desirable characteristics of a supplemental assay

Any supplemental assay must be as sensitive as the screening assay and must be directed at a target on a different gene that is known to have discriminatory value in identifying gonococcus. While it may be possible to use two targets on the same gene, there is insufficient data currently to support this proposition. In particular, the propensity for genetic exchange among *Neisseria* species raises the possibility of exchange of a large section of the gene that may include both of these targets.

Currently the acceptable assays are directed at various targets in the CMT gene, the pilin gene, 16S RNA gene, the *opa* gene and the *porA* pseudogene. Laboratories need to assess the value of particular targets either based on their own data or the data in the literature, noting again that the assessment should be on-going due to the propensity for genetic exchange in gonococcal populations.

Recommendation 4: If a sample is positive in a screening assay but a suitable supplemental assay is negative, then the result should be reported as negative.

Evaluating test performance

The workshop participants recognised that to implement the recommendations it is necessary to define the acceptable performance criteria for test combinations. The reported result should have a positive predictive value (PPV) of at least 90 per cent in the population being tested, based on current US recommendations for chlamydia and gonorrhoea testing.[14] However, calculating the PPV depends on disease prevalence, which is often difficult for laboratories to determine accurately. This may be because it is not known which population the patient belongs to, because the prevalence in the test population is unknown, because the test population contains mixed groups with differing prevalences and/or because there is a reasonable chance that an individual within that population may have a substantially different pretest probability from the average for that population. In view of these difficulties, it is preferred that the test PPV is calculated based on a prevalence of 1 per cent as that represents a low prevalence population. Therefore if the assay has a sensitivity of 99 per cent, then a specificity of 99.9 per cent is required to yield a PPV of >90 per cent. Getting a good negative predictive value in populations (or patients) with a pretest probability less than 1 per cent is extremely difficult and no test combination can be expected to yield a high PPV in that situation.

Recommendation 5: Laboratories should ensure that the test combination they use would yield a positive predictive value of at least 90 per cent in a population with a prevalence of 1 per cent.

For the purposes of test evaluation it is important that there is an accepted definition of genuine positive samples for comparing tests. Detection of *N. gonorrhoea* by culture using contemporary culture and identification methods is regarded as suitable confirmation that a positive NADT is genuine. While it is recognised that, in theory, one could get a false positive NADT accompanied by a genuine positive culture, this situation is unlikely and should become apparent during the process of continuing test evaluation.

Often culture is not suitable as a confirmatory test as either no culture has been performed, or it is less sensitive than the NADT being evaluated. In those circumstances other nucleic acid-based methods are required. Use of two separate targets may still yield false positive results, and therefore it should require detection of at least three separate targets that have discriminatory value in order to be acceptably confident that it is genuine. Alternatively, the identity may be confirmed by DNA sequencing. The propensity for genetic recombination in and between pathogenic and non-pathogenic *Neisseria* species has been referred to above. This raises theoretical concerns regarding sequencing for gonococcal confirmation, in that the sequenced gene may be residing in a non-gonococcal organism. There is now a considerable amount of literature on the use of sequencing for identification and typing of Neisseria species and, while misidentification due to genetic exchange remains a theoretical possibility, it is a highly unlikely, particularly if an internal gene such as CMT is sequenced.

Recommendation 6: For the purposes of test evaluation, as distinct from diagnostic testing, true positives be defined by meeting one or more of the following criteria:

1) *culture positive using contemporary isolation and identification techniques;*

2) *positive result on NADTs directed to targets on three separate genes that are known to have discriminatory capacity;*

3) *sequencing of a gene known to separate gonococcal from non-gonococcal species.*

Inhibitory samples

It is known that samples may contain substances that inhibit NADT and unless a suitable inhibitor control is included, the test may yield a false negative result.

Recommendation 7: Inhibitor controls should be routinely included in all NADT.

Testing of samples from extra-genital sites

As there is still limited data on the performance of NADT on these samples, and mixed *Neisseria* species are more likely, traditional cultures are the preferred test.

Interpretation of a negative result on NADT from extra-genital sites depends on sensitivity of the assay, and currently there is little data on this. Experience with genital tract samples strongly suggests that NADT will be more sensitive than culture. Currently we do not have sufficient Australian data, partly due to the lack of positive samples. The available sensitivity data needs to be reviewed before further recommendations can be made on this issue. Similarly there are unresolved concerns about specificity for non-genital samples.

Recommendation 8: Cultures are the preferred test for samples from non-genital sites. If however it is necessary to perform a NADT, then more stringent criteria should be applied, and positive samples should meet the 'test evaluation' criteria for a 'true positive'.

Quality assurance (QA)

Reference is made above for the need for ongoing assessment and evaluation of any NADT or NADT combination selected for use. A properly constituted and thoughtful approach to external QA would simplify compliance with this requirement. Australian laboratories mainly test samples from low prevalence populations and therefore encounter relatively few positive samples. Also gonococcal strains that pose difficulties may be uncommon or may emerge within restricted populations. In order to properly monitor test performance quality assurance samples should include a wide range of problem specimens or organisms.

Recommendation 9: In order to properly assess the routine diagnostic system in Australia, the following quality assurance samples should be distributed in addition to the routine samples currently used:

1. cppB *negative* N. gonorrhoeae;

2. *Non-gonococcal species known to cause false positive reactions: these should be dispatched both as a single species, as well as mixture with* N. gonorrhoeae. *In the latter circumstance, the non-gonococcal species should be present in 10-fold excess;*

3. *Urine samples: Preferably a single patient sample, otherwise a spiked sample;*

4. *Validation panels should be made that include samples that are culture positive but PCR negative. True positive samples should also be made available.*

In addition, a process should be established for full phenotypic and genotypic characterisation of unidentified species that yield false positive results in NADT for gonococci.

Antimicrobial resistance (AMR) surveillance requirements – isolate numbers

Although the issues involved are complex, it is presently accepted that a sample size should be large enough to detect a proportional resistant rate of 5 per cent. Statistically the sample size requirements for this threshold can be quite demanding e.g. up to 1,200 isolates per study period. While comprehensive and continuous sampling is important to maximise isolate numbers to increase the validity of the surveillance, use of other approaches such as discontinuous samples and a Lot Quality Assurance assessment is possible. The latter approach requires that a *minimum* of 200 isolates from each population group for each study period should be examined. If it is found that either no or a very low proportion of isolates are resistant, or alternatively a high proportion are resistant, the basic question to continue or discontinue with a standard treatment is answered. No further enhancement of surveillance is required. However if the detected resistance is in the critical range where a change in treatment may be contemplated, 'enhanced surveillance', i.e. an enlarged sample, is required until such time as the question of treatment alteration is resolved.

In order to maintain these minimum numbers, the capacity for high quality gonococcal culture methods should be maintained within all the relevant populations.[14] All isolates should have susceptibility testing performed in a laboratory that reports to the

National Neisseria Network (NNN) or else those isolates should be referred to the NNN laboratory in the relevant jurisdiction.

Cultures should be routinely performed on swabs where there is ready access to a suitable laboratory. Also, patients who have had a positive NADT and have not been treated should have a culture performed if that was not done initially.

A strategy that may be used to achieve the appropriate numbers of isolates in areas where primary culture is not routinely performed is to undertake 'targeted culture' of populations where a significant yield of gonococci may be expected so that a significant cost/benefit for culture is obtained. Such situations include culture of males with urethral discharge, especially in clinic situations, or culture of urine samples collected for NADT and that are positive on this test. Data collected indicate that up to 70 per cent of urine samples positive on NADT will provide an isolate of GC if properly cultured within 24 hours of collection.[20]

Recommendation 10: Strategies should be put in place to ensure that sufficient numbers of gonococcal isolates are obtained to allow reliable monitoring of antimicrobial resistance.

These targets need to be met within each population in which there is expected to be a difference in rates of resistance or in which the risk of emergence of resistance is different. For example, it is important to ensure that sufficient samples are obtained to detect resistance within both Indigenous and non-Indigenous communities throughout Australia. Identification of the relevant population groups should be undertaken by public health practitioners experienced in the epidemiology and control of sexually transmitted infections.

Recommendation 11: Public health practitioners need to define the relevant populations that need to be targeted and identify any that require enhanced surveillance.

Appendix. List of attendees and relevant affiliations

David Smith, Perth: Public Health Laboratory Network; PathWest Laboratory Medicine WA

John Tapsall, Sydney: National Neisseria Network; Public Health Laboratory Network

Gary Lum, Darwin: National Neisseria Network; Public Health Laboratory Network

Sue Alderson, Sydney: Institute of Clinical Pathology and Medical Research

Miles Beaman, Perth: Western Diagnostic Pathology

Sue Garland, Melbourne: Women's and Children's Hospital

Rod Givney, Adelaide: Communicable Diseases Network Australia

Gerry Harnett, Perth: PathWest Laboratory Medicine WA

Geoff Higgins, Adelaide: IMVS, Public Health Laboratory Network

Geoff Hogg, Melbourne: Microbiological Diagnostic Unit, Public Health Laboratory Network

David Leslie, Melbourne: Victorian Infectious Diseases Reference Laboratory

Theo Sloots, Brisbane: Sir Albert Sakzewski Virus Research Centre

Sepehr Tabrizi, Melbourne: Women's and Children's Hospital

David Whiley, Brisbane: Sir Albert Sakzewski Virus Research Centre

References

1. Farrell DJ. Evaluation of AMPLICOR *Neisseria gonorrhoeae* PCR using c*ppB* nested PCR and 16S rRNA PCR. *J Clin Microbiol* 1999;37:386–390.

2. Lum G, Freeman K, Nguyen NL, Limnios EA, Tabrizi SN, Carter I, *et al*. A cluster of culture positive gonococcal infections but with false-negative *cppB* gene based PCR. *Sex Transm Infect* 2005;81:400–402.

3. Tapsall JW, Limnios EA, Nguyen NL, Carter I, Lum G, Freeman K, *et al*. Problematic *cppB*-gene based assays may contribute to failure to confirm BD ProbeTEC PCR for *Neisseria gonorrhoeae* with a *cppB*-gene based PCR. *J Clin Microbiol* 2005;43:2036–2037.

4. Whiley DM, Buda PJ, Bayliss J, Cover L, Bates J, Sloots TP. A new confirmatory *Neisseria gonorrhoeae* real-time PCR assay targeting the *porA* pseudogene. *Eur J Clin Microbiol Infect Dis* 2004;23:705–710.

5. Tabrizi SN, Chen S, Tapsall J, Garland S. Evaluation of *opa*-based real time PCR for detection of *N. gonorrhoeae*. *Sex Transm Dis* 2005;32:199–202.

6. Culler EE, Caliendo AM, Nolte FS. Reproducibility of positive test results in the BDProbeTec ET system for detection of *Chlamydia trachomatis* and *Neisseria gonorrhoeae*. *J Clin Microbiol* 2003;41:3911–3914.

7. Palmer HM, Mallinson H, Wood RL, Herring RJ. Evaluation of the specificities of five DNA amplification methods for the detection of *Neisseria gonorrhoeae*. *J Clin Microbiol* 2003;41:835–837.

8. Koenig, MG, Kosha SL, Doty BL, Heath DG. Direct comparison of the BD ProbeTec ET system with in-house LightCycler PCR assays for detection of *Chlamydia trachomatis* and *Neisseria gonorrhoeae* from clinical specimens. *J Clin Microbiol* 2004;42:5751–5756.

9. Golden MR, Hughes JP, Cles LE, Crouse K, Gudgel K, Hu J, *et al.* APTIMA Combo 2 testing for *Neisseria gonorrhoeae* in a population of women with low prevalence of *N. gonorrhoeae* infection. *Clin Infectious Dis* 2004;39:1387–1390.

10. Gaydos CA, Quinn TC, Willis D, Weissfeld A, Hook EW, Martin DH, *et al*. Performance of the APTIMA Combo 2 assay for detection of *Chlamydia trachomatis* and *Neisseria gonorrhoeae* in female urine and endocervical swab specimens *J Clin Microbiol* 2003;41:304–309.

11. Miller PJ, Torzillo PJ, Hateley W. Impact of improved diagnosis and treatment on prevalence of gonorrhoea and chlamydial infection in remote aboriginal communities on Anangu Pitjantjatjara Lands. *Med J Aust* 1999;170:429–432.

12. Annual report of the Australian Gonococcal Surveillance Programme, 2004. *Commun Dis Intell* 2005;29:136–141.

13. World Health Organization. Surveillance standards for antimicrobial resistance. 2001. Available from http://www.who.int/drugresistance/publications/WHO_CDS_CSR_DRS_2001_5/en/

14. Johnson RE, Newhall WJ, John R, Papp JR, Knapp JS, Black CM, *et al*. Screening tests to detect *Chlamydia trachomatis* and *Neisseria gonorrhoeae* infections—2002 *MMWR Recomm Rep* 2002; 51(RR15);1–27.

15. Frosch M, Meyer TF. Transformation-mediated exchange of virulence determinants by co-cultivation of pathogenic *Neisseriae. FEMS Microbiol Lett* 1992;79:345–349.

16. Halter R, Pohlner J, Meyer TF, Abeck D, Johnson AP, Alexander FE, *et al*. Plasmid content and protein I serovar of non-penicillinase-producing gonococci isolated in Munich. *Epidemiol Infect* 1988;100:345–349.

17. Kroll JS, Wilks KE, Farrant JL, Langford PR. Natural genetic exchange between *Haemophilus* and *Neisseria:* intergeneric transfer of chromosomal genes between major human pathogens. *Proc Natl Acad Sci U S A* 1998;95:12381–12385.

18. Linz B, Schenker M, Zhu P, Achtman M. Frequent interspecific genetic exchange between commensal *Neisseriae* and *Neisseria meningitidis*. *Molecular Microbiol* 2000;36:1049–1058.

19. Rosson, S, de Gooyer E, Moore A, Morcom J, Davies JK. 1985. Site-specific recombination systems in gonococcal plasmids, p. In: Schooling GK ed. *The pathogenic Neisseria*. American Society for Microbiology, Washington, D.C. 1985;175–179.

20. Tapsall JW, Ray S, Limnios EA. Targeted culture for *N. gonorrhoeae* from FVU increases isolate numbers for gonococcal antimicrobial susceptibility monitoring: importance of time to culture and FVU physical characteristics. Abstract WP 010 16th Biennial meeting of the International Society for STD Research (ISSTDR), Amsterdam 10 –13 July 2005.

Surveillance of Shiga toxigenic *Escherichia coli* in Australia

Barry G Combs,[1] Jane CA Raupach,[1] Martyn D Kirk[2]

Abstract

All Australian States and Territories have low rates (≤ 0.32 cases per 100,000 population) of notification for Shiga toxin-producing *Escherichia coli* (STEC), except for South Australia where the rates are ten-fold higher at 2.58 cases per 100,000 population. To explore possible reasons for the variation in rates we surveyed public health reference laboratories to determine the methods used and number of specimens tested for these organisms. Only five of eight jurisdictions routinely conducted testing for STEC, and polymerase chain based tests were most common. Culture was also common and in one jurisdiction that tests specimens with culture, approximately 1.2 per cent of specimens were positive. The notification rates for different jurisdictions reflected the number of specimens tested, with jurisdiction testing ≤ 500 specimens having rates ≤ 0.32 cases per 100,000 population. The use of culture as a test method may also influence notification rates. Public health agencies must consider the number of specimens tested in interpreting surveillance data. *Commun Dis Intell* 2005;29:366–369.

Keywords: diagnoses, Escherichia coli, *Shiga toxin, surveillance*

Introduction

Shiga toxin-producing *Escherichia coli* (STEC) was first reported as a significant foodborne pathogen in the United States of America (USA) where it caused outbreaks of gastroenteritis associated with the consumption of undercooked beef mince in 1982.[1] In Australia, there have been eight reported outbreaks of STEC. The largest outbreak with 23 cases of haemolytic uraemic syndrome occurred in South Australia in 1995, as a result of *E. coli* O111:H contaminated mettwurst.[2] However, most STEC infections in Australia are sporadic with between 43–60 cases notified to health departments each year.[3,4] In 2001, the majority of jurisdictions reported low rates of STEC notification (0–0.4 STEC cases/100,000 population).[5] The exception was South Australia with a notification rate of 1.7 STEC cases per 100,000 population. South Australia's notification rate increased to this rate of notification following the introduction of testing of all bloody stools with a polymerase chain reaction test (PCR) in 1997, as a response to the large STEC outbreak in 1995.[2]

Reports have shown that different surveillance[6] and diagnostic methods[7] are critical factors in determining the number of STEC infections identified in the community. This report describes surveillance and diagnostic practices in Australian reference laboratories that may influence notification of STEC in Australia.

The survey

We surveyed State and Territory reference laboratories in September 2003 with a semi-structured questionnaire on screening and diagnostic practices for STEC. OzFoodNet epidemiologists conducted face-to-face interviews with staff of reference laboratories for each Australian State and Territory. In Australia, most STEC screening and diagnosis is carried out in each jurisdictional reference laboratory. This information was collated and crude proportions calculated using Microsoft Excel.

Results

Reference laboratories in three jurisdictions, Tasmania, the Australian Capital Territory and the Northern Territory reported that they do not carry out any testing for STEC. If STEC testing is requested these laboratories refer samples to other interstate reference laboratories.

Reference laboratories in Queensland, Western Australia, New South Wales and South Australia have standing requests for other laboratories in the jurisdiction to send stool samples for testing. The screening criteria for each of these reference laboratories to determine if a sample was tested for STEC were: if the test was requested by the physician; if the patient had clinical evidence of

1. OzFoodNet, South Australian Department of Health, Adelaide, South Australia
2. OzFoodNet, Department of Health and Ageing, Canberra, Australian Capital Territory

Corresponding author: Dr Barry Combs, OzFoodNet, Communicable Disease Control Branch, Department of Health, PO Box 6, Rundle Mall, Adelaide, SA 5000. Telephone: +61 8 8226 6318. Facsimile: +61 8 8226 7187. Email: barry.combs@health.sa.gov.au

recent bloody diarrhoea; and/or if the stool sample had evidence of macroscopic blood. Western Australian laboratories also tested stool samples if there was evidence of blood microscopically. The number of samples tested for STEC during 2002, ranged from 141 samples in Queensland to 1,665 samples in South Australia (Table 1). Most of the 123 specimens tested in Victoria were presumptive STEC isolates sent from other laboratories.

Diagnostic methods varied between reference laboratories and are described in Table 2. The Queensland laboratory reported using a combination of PCR, chromogenic agar, sorbitol MacConkey agar, the Premier EHEC ELISA (Meridian Diagnostics, Inc) and a chromatography method using Duopath (Merck). The New South Wales laboratory used chromogenic agar and some regional laboratories also used either PCR, chromogenic agar, sorbitol MacConkey agar and washed sheep blood agar plates. Victoria used a number of methods including culture, immunological assays to detect toxin and PCR to test mainly presumptive STEC isolates. The South Australian laboratory used PCR only and the Western Australian laboratory used sorbitol MacConkey agar only.

South Australia was the only jurisdiction that did not routinely attempt to culture for STEC isolates if a stool sample was positive (i.e. by PCR). Presumptive STEC isolates were confirmed by PCR methods in all jurisdictions. Toxin activity was also carried in tissue culture by Victoria and Western Australia. Flagella and phage typing was only available in the Victorian reference laboratory. Pulse field gel electrophoresis was carried out for cluster investigations by Queensland, Victorian and South Australian laboratories but no common method was used.

In Western Australia, approximately 1.2 per cent of the tested samples/specimens were positive for STEC. For other jurisdictions the percentage of tested samples/specimens that met jurisdictional screening criteria and were positive varied from 2.3 per cent to 6.9 per cent (Table 1).

Table 1. Sample testing rates for STEC in Australian State Reference Laboratories

Jurisdiction	Number of laboratories referring samples	Samples tested (2002)			Notification rate 2002 (per 100,000 population)
		Number of specimens tested	Number of specimens positive	Percentage of specimens positive	
Queensland	20	141	5	3.5	0.14
New South Wales	13	145	10	6.9	0.16
Victoria	12	123*	5	4.1	0.11
South Australia	4	1,665	39	2.3	2.58
Western Australia	Not available	500†	6	1.2	0.32

* Most of these specimens were presumptive isolates.

† Approximate number only.

Table 2. Laboratory methods commonly used (80–100% of the time) by Jurisdiction Reference Laboratories to identify STEC in faeces

Laboratory method used	State				
	NSW	Qld	SA	WA	Vic
ELISA detection of toxin in stool		✓			
Pre-enrichment (PES) of stool			✓		✓
Culture on Sorbitol MacConkey agar		✓		✓	✓
PCR for *stx1* & *stx2* of stool					✓
PCR for *stx1* & *stx2* of PES		✓	✓		✓
Immunological toxin detection		✓			✓
Toxin detection in isolate (Duopath)		✓			
Chromogenic agar culture	✓	✓			

Discussion

There is a wide range of diagnostic practices used for testing for STEC among jurisdictional reference laboratories in Australia. These practices lead to vastly different notification rates between individual jurisdictions, ranging from 0.14 to 2.58 cases per 100,000 population.

Although many jurisdictions had the same screening criteria for testing samples and request that other laboratories send bloody stools for STEC screening, there is a large range in the number of specimens tested, from 123 in Victoria to 1,665 specimens tested in South Australia. The difference in the numbers of specimens tested could be due to a number of reasons, including whether the reference laboratory conducts primary diagnosis (South Australia, Western Australia), which would allow easier access to specimens for testing.

The percentage of tested samples/specimens that were positive for STEC in Australia varied from 1.2 per cent to 6.9 per cent, which may be due to the origin of specimens, whether they are presumptive isolates, and the diagnostic method used. The types of diagnostic tests used ranged from culture only in Western Australia and in New South Wales to a range of methods including culture, immunological methods to detect toxin and PCR methods in Queensland and Victoria. It has been reported that culture methods for detection of STEC are less sensitive than other methods such as PCR or the Premier EHEC ELISA.[7,8] This may explain part of the reason why in Western Australia, where specimens were tested by culture, there was a low proportion of specimens testing positive for STEC. However, Western Australia did have a higher notification rate (≤0.32 cases per 100,000 population) than Queensland, Victoria and New South Wales, which is likely due Western Australia testing a much larger number of stool specimens than these other jurisdictions. South Australia had the highest notification rate of 2.58 cases per 100,000 population, which could be due to the testing of all bloody stools with a PCR based method. South Australia and the Hunter Valley region of New South Wales obtained similar proportions of samples positive for STEC when using similar PCR-based methods.[9]

The differences in notification rates between jurisdictions could also be in part due to real differences in prevalence. This has been observed in the USA, with northern states having a higher prevalence of STEC O157:H7 than southern states, which may be associated with large rural populations in northern states and contact with farm animals.[7] There could also be other differences that may affect notification rates including variability in health care systems, access to medical care, farm animal husbandry practices and susceptibility of the population (younger and older age groups are at higher risk of STEC infection).

With the exception of South Australia, most rates of STEC notification in Australian jurisdictions are lower than those reported internationally, including the USA[10] and Wales.[11]

This survey of reference laboratories and STEC cases notified in Australia indicate that the number of stool samples tested and the sensitivity of the diagnostic test may explain much of the variability in notification rates between jurisdictions.

Since this survey, some jurisdictions have made changes to the surveillance of STEC. In the first half of 2004 and January 2005 respectively, Western Australia and Victoria started using PCR to diagnose STEC in stool samples. Victoria also increased the number of stools tested. New South Wales is proposing similar changes. These changes are likely to increase notification rates and should also give a better understanding of the prevalence of STEC in Australia.

Acknowledgements

We thank staff in State and Territory reference laboratories for participating in the survey, and providing useful feedback on the manuscript. We would also like to thank the OzFoodNet Working Group for administering the questionnaire and providing comments on the manuscript.

References

1. Riley LW, Remis RS, Helgerson SD, McGee HB, Wells JG, Davis BR, *et al.* Hemorrhagic colitis associated with a rare *Escherichia coli* serotype. *N Engl J Med* 1983;308:681–685.

2. Community outbreak of haemolytic uremic syndrome attributable to *Escherichia coli* 0111:NM—South Australia, 1995. *MMWR Morb Mortal Wkly Rep* 1995;44:550–551, 557–558.

3. Australian Government Department of Health and Ageing. Communicable Diseases Network Australia—National Notifiable Diseases Surveillance System. Notifications of STEC/VTEC, Australia 1996–2002. Australian Government Department of Health and Ageing. Canberra; 2003.

4. OzFoodNet Working Group. Foodborne disease investigation across Australia: annual report of the OzFoodNet network, 2003. *Commun Dis Intell* 2004;28:359–389.

5. OzFoodNet Working Group. Enhancing foodborne disease surveillance across Australia in 2001: the OzFoodNet Working Group. *Commun Dis Intell* 2002;26:375–406.

6. Gavin PJ, Thomson Jr RB. Diagnosis of enterohemorrhagic *Escherichia coli* infection by detection of Shiga toxins. *Clin Micro Newsletter* 2004;26:49–54.

7. Fey PD, Wickert RS, Rupp ME, Safranek TJ, Hinrichs SH. Prevalence of non-O157:H7 Shiga toxin-producing *Escherichia coli* in diarrheal stool samples from Nebraska. *Emerg Infect Dis* 2000;6:530–533.

8. Mackenzie AM, Lebel P, Orrbine E, Rowe PC, Hyde L, Chan F, *et al.* Sensitivities and specificities of premier *E. coli* O157 and premier EHEC enzyme immunoassays for diagnosis of infection with verotoxin (Shiga-like toxin)-producing *Escherichia coli*. The SYNSORB Pk Study investigators. *J Clin Microbiol* 1998;36:1608–1611.

9. Doyle R, Watson K, Unicomb LE, Lancer JA, Wise R, Ratcliff R, *et al.* Laboratory surveillance of Shiga toxin producing *Escherichia coli* in South Australia and the Hunter Health Area, New South Wales, Australia. *Commun Dis Intell* 2004;28:390–391.

10. Preliminary FoodNet data on the incidence of foodborne illnesses—selected sites, United States, 2000. *MMWR Morb Mortal Wkly* Rep 2001;50:241–246.

11. Chalmers RM, Parry SM, Salmon RL, Smith RM, Willshaw GA, Cheasty T. The surveillance of vero cytotoxin-producing *Escherichia coli* O157 in Wales, 1990 to 1998. *Emerg Infect Dis* 1999;5:566–569.

Epidemic viral gastroenteritis in Queensland coincides with the emergence of a new norovirus variant

Michael J Lyon,[1] Gang Wei,[2] Greg A Smith[1]

Abstract

Norovirus infections cause widespread morbidity and have significant economic impact on the community. An increase in outbreaks of norovirus gastroenteritis in hospitals, nursing homes and in the community was observed in Queensland in 2004. Molecular analysis of positive samples indicated the emergence of a single strain of norovirus. A 252 nucleotide sequence from the polymerase region (POL) was compared to sequences of the new variant genotype GII.4 that has caused epidemics in the Northern Hemisphere in 2002 and 2003. Sequence analysis indicated greater than 95 per cent similarity in the POL between the Queensland strain and the Northern Hemisphere 2002/3 GII.4 variant. Phylogenetic analysis revealed that the Queensland strain forms a branch within the GII.4 genotype separate from the 2002 variant from Europe and North America. Although norovirus genotype GII.4 had circulated in Queensland in the past, the 2004 strain was characterised specifically by three nucleotides not present in any other sequences held in our database covering the years 2002–June 2004. *Commun Dis Intell* 2005;29:370–373.

Keywords: norovirus, epidemic, new variant

Introduction

It has long been recognised that noroviruses (previously called Norwalk–like viruses) are one of the common causes of outbreaks of acute non-bacterial gastroenteritis.[1] Members of the Family *Caliciviridae,* noroviruses are non-enveloped positive strand RNA viruses.[2,3,4] Noroviruses that infect humans cannot, presently, be grown in tissue culture or cultivated in suitable animal models. Noroviruses have a low infectious dose, are able to withstand harsh environmental conditions and can be spread by a variety of routes.[5,6] In 1993, Jiang and colleagues published the full length sequence of Norwalk virus.[3] Since then a myriad of different molecular detection and molecular analytical techniques have been developed and applied to a number of epidemiological studies.[7,8] The genome of 7.2–7.7 kb can be divided into three open reading frames; ORF 1, ORF 2 and ORF 3. Open reading frame 1 contains a RNA dependent RNA polymerase, whilst ORF 2 and ORF 3 code for a capsid protein and a minor structural protein respectively.

Norovirus strains have been traditionally named according to the geographical location from which they were first identified (e.g. Bristol, Toronto, Hawaii).[9] It has been proposed that noroviruses associated with human infection be divided into a number of genogroups namely GI, GII and GIV. These genogroups can then be further divided into at least 31 genotypes.[10]

The last decade has seen genogroup II, genotype 4 (GII.4) activity on a number of continents. In 1995–1996 a Lordsdale–like norovirus variant was demonstrated to have circulated worldwide. This strain predominated in different locations within the United States of America (USA) and was also demonstrated to have circulated in seven countries on five continents.[11] In 2002 and 2003 a large number of outbreaks of non-bacterial gastroenteritis occurred in North America and in Europe. The aetiological agent of the outbreaks was identified as norovirus.[12,13] This increase in norovirus activity occurred at the same time as the emergence of a new variant of GII.4 was identified. This variant had a major economic impact on cruise ships, hotels,

1. Public Health Virology Laboratory, Queensland Health Scientific Services, Coopers Plains, Queensland
2. Respiratory and Enteric Viruses Branch, Division of Viral and Rickettsial Diseases, Centers for Disease Control and Prevention, Atlanta, Georgia, United States of America

Corresponding author: Mr Michael Lyon, Public Health Virology Laboratory, Queensland Health Scientific Services, PO Box 594, Archerfield, Brisbane, Queensland 4108. Telephone: +61 7 3000 9180. Facsimile: +61 7 3000 9186.
Email: Michael_Lyon@health.qld.gov.au

nursing homes, hospitals and other public health facilities with a large number of staff, guests and residents of these facilities affected.[14]

At the beginning of 2004, Queensland experienced a large epidemic of viral gastroenteritis comprised of numerous outbreaks. These outbreaks were characterised by high attack rates and rapid spread throughout the community (Table). The virus was first observed circulating in the heavily populated south east corner of the state in January before spreading north to Cairns by June 2004. Virus activity was also reported in the west of the state with isolated outbreaks identified in Toowoomba and Biloela in June 2004. Outbreaks occurred in northern New South Wales including Lismore and Tweeds Heads in May 2004.

Institutional spread occurred despite the efforts of public health officials who provided advice on food handling and infection control procedures. The virus was responsible for ward closures in six Brisbane metropolitan public hospitals as well as a number of the larger private hospitals. Aged-care facilities were particularly hard hit. Sequence analysis of a proportion of samples revealed a single GII.4 variant was responsible for the recent outbreaks.

Methods

Sample collection

Eight hundred and fifty samples from private pathology laboratories,[14] public health units and public hospitals throughout Queensland were submitted to this laboratory for norovirus analysis between January 2004 and June 2004 representing 159 outbreaks of community acquired and institutional viral gastroenteritis (Table). Fifty-seven of these norovirus isolates separated chronologically and geographically were then selected for sequence analysis.

RNA extraction and reverse transcriptase polymerase chain reaction protocol

Faecal samples were prepared as a 10 per cent suspension in phosphate buffered saline and centrifuged for one minute at 20,000 *g* to pellet solid material. Viral RNA was then extracted from 140 µl of the supernatant using the QIAamp Viral RNA Mini Kit (QIAGEN) according to the manufacturer's instructions. Reverse transcriptase polymerase chain reaction (RT-PCR) was used to amplify a 319 bp section of ORF 1 using primer pair p289/290, based on a modified method by Jiang, *et al.*[15] (personal communication). All products were analysed by gel electrophoresis.

Nucleotide sequencing and phylogenetic analysis

Sequence of the amplified product generated from isolates was used to characterise the strain(s) responsible for the recent outbreaks. Fifty-seven positive samples from 41 hospital and nursing home outbreaks, and 11 community-acquired outbreaks were subjected to sequence analysis. Amplified PCR products of 252 nucleotides of the polymerase region (POL) were purified using QIAquick® Gel Extraction Kit according to the manufacturer's instructions (QIAGEN). Products were sequenced in both directions using Applied Biosystems ABI Prism BigDye Terminator Cycle Sequencing Ready Reaction Kits. Sequences were compared to databases at the Centers for Disease Control and Prevention (USA) and the National Institute of Public Heath and the Environment (Netherlands). Sequences were aligned using ClustalW. Phylogenetic analysis was performed and unrooted trees constructed using neighbour–joining.[16]

Results

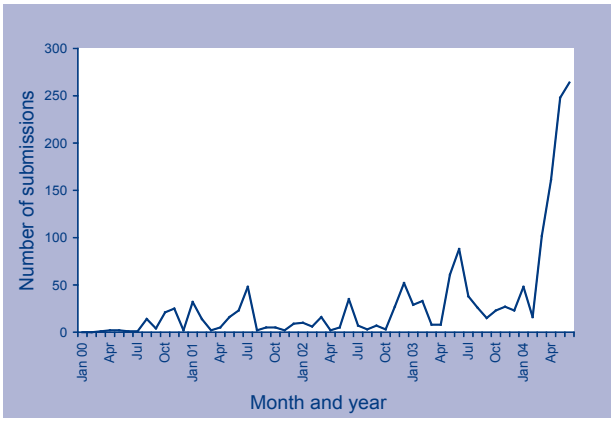
Queensland experienced higher rates of norovirus-related gastrointestinal illness in the first six months of 2004 than was usually the case for the previous three years (Table, Figure 1). This affected six major metropolitan public hospitals, six regional hospitals, a further 29 aged-care facilities, and, in addition, there was evidence of community spread. In the interest of public health we initiated a molecular epidemiological study of the epidemic.

A representative proportion of the norovirus PCR-positive samples were sequenced (57 of 97 positive samples). Analysis of the sequences obtained from a fragment of the POL region indicated that 95 per cent (55 of the 57 representative samples) belonged

Table. Details of outbreaks of gastroenteritis linked to norovirus between 2002 and 2004

Year	Number of samples	Number of samples positive for norovirus	Number positive (%)	Number of samples sequenced	Number of GII.4	Number of Queensland strain, 2004
2002	156	57	37	27	10	0
2003	387	121	31	61	32	0
January–June 2004	850	362	43	57	55	54

Figure 1. Number of samples submitted for norovirus analysis between January 2000 and June 2004



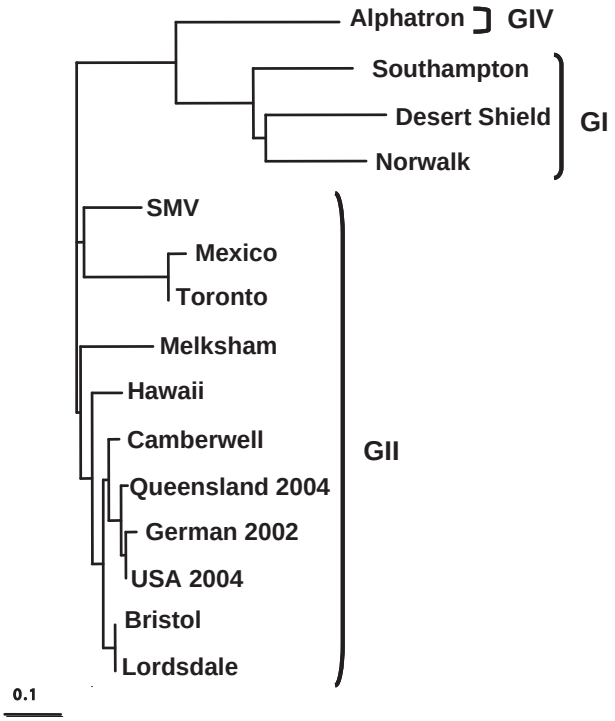
to genotype GII.4 (Figure 2). Two isolates were distinctly different from GII.4 and grouped in GII.8 and GI.2, respectively (data not shown). All of the 55 GII.4 isolates sequenced had sequence similarity of 99 per cent. More significantly, when these sequences were compared to GII.4 strains circulating in 2002 and 2003, differences were observed with these earlier strains. A three nucleotide signature sequence was identified which distinguished the recent strain from all previous GII.4 noroviruses isolated in Queensland. The Queensland strain had 95 per cent sequence identity to a strain circulating in the United States in 2002 (GenBank accession number AY502023). Phylogenetic analysis revealed that the Queensland strain forms a branch within the GII.4 genotype separate from the 2002 variant from Europe and North America (Figure 2).

Discussion

Norovirus infections will continue to cause significant illness in Australia and worldwide. This latest Queensland epidemic is significant because, by comparison with our in-house norovirus sequence database of previous years, it is apparent that a single genotypically homogenous strain has predominated. This observation is not due to increased reporting as surveillance activities by health authorities in 2004 have been no different to previous years.

Experience in the past has shown that generally outbreaks of norovirus infections have a variety of genotypic and phenotypic presentations. The Northern Hemisphere strain, with which the Queensland strain is closely related, has been associated with a severe pathology.[13,14] Interestingly, anecdotal evidence from clinicians and public heath physicians indicated that the severity of symptoms of the recent Queensland strain was greater than has been previously seen (Dr Brad McCall, personal communication); however, we have no clinical data to support this observation.

Figure 2. Phylogram indicating the relationship between norovirus strains* based on 252 nucleotides in the RNA POL gene



* Strain identification and GenBank accession number in parentheses for human noroviruses listed according to a recently proposed classification system.[2,17] Proposed Genogroup IV Hu/NLV/Alphatron (personal communication Harry Vennema), Genogroup 1, Hu/NLV/Southampton 1991/UK (L07418), Hu/NLV/Desert Shield 395/1990/SA (U04469), Hu/NLV/Norwalk/8FIIa/1968/JP (M87661),Genogroup II, Hu/NLV/Snow Mountain/2003/US (AY134748), Hu/NLV/Mexico/1989/Mx (U22498), Hu/NLV/Melksham/1989/UK (X81879), Hu/NLV/Hawaii/1971/US (U07611), Hu/Camberwell/1994/AU (U46500), Hu/NoV/Brisbane/01/2004/AU (AY780432), Hu/NoV/Farmington Hills/2002/US (AY502023), Hu/NLV/GII/Langen1061/2002/DE (AY485642), Hu/NLV/Bristol/1993/UK (X76716), Hu/NLV/Lordsdale/1993/UK (X86557).

The cost to the community in terms of hospital ward closures, deferred medical interventions, extensive and repeated decontamination of aged facilities as well as lost productivity has been substantial.

In terms of improvements to future methodology, analyses including sequences from both the conserved capsid and POL regions would allow for a more accurate analysis in genotyping studies. In addition, we suggest epidemiological studies of norovirus outbreaks would be enhanced by standardisation of detection and genotyping procedures nationally, and mandatory reporting to a central database. This would be particularly useful for detection

of foodborne norovirus from foods such as imported oysters. Recently Vinjé and colleagues[17] reported on an international collaborative study to compare RT-PCR assays for the detection and genotyping of noroviruses. The laboratories participating in this study were located in the United States of America, the United Kingdom and Europe. Collaboration and standardisation of testing procedures with laboratories in the Southern Hemisphere and a central database could link researchers to provide an enhanced global surveillance system.

Acknowledgements

Harry Vennema (National Institute for Public Health and the Environment, Diagnostic Laboratory for Infectious Diseases and Perinatal Screening, National Institute of Public Health and The Environment Bilthoven, The Netherlands) kindly provided sequence information and valuable advice. The authors would also like to thank Marion Koopmans (Co-ordinator 'Foodborne viruses in Europe' Head of Virology Section, Diagnostic Laboratory for Infectious Diseases and Perinatal Screening, National Institute of Public Health and The Environment Bilthoven, The Netherlands) and David Warrilow (HIV Molecular Biology Laboratory, Queensland Institute of Medical Research, Brisbane, Queensland) for their advice and for reviewing this article.

References

1. Fankhauser RL, Noel JS, Monroe SS, Ando T, Glass RI. Molecular epidemiology of 'Norwalk-like viruses' in outbreaks of gastroenteritis in the United States. *J Infect Dis* 1998;178:1571–1578

2. Green KY, Kapikian AZ, Chanock RM. Human caliciviruses. In: Griffin DM, *et al,* eds. *Fields virology*, 4th edition. Philadelphia: Lippincott-Raven 1999; 841–874.

3. Jiang X, Wang M, Wang K, Estes MK. Sequence and genomic organization of Norwalk virus. *Virology* 1993; 195:51–61.

4. Kapikian AZ, Ed. Norwalk and norwalk-like viruses. In: *Viral Infections of the Gastrointestinal Tract* 2nd edition. New York: Marcel Dekker Inc, 1994; pp 471–518.

5. Parashar UD, Monroe S. Norwalk-like virsues as a cause of foodborne disease outbreaks. *Rev Med Virol* 2001;243–252.

6. Rockx B, De Wit M, Vennema H, Vinjé J, De Bruin E, Van Duynhoven Y, *et al*. Natural history of human calicivirus infection: a prospective cohort study. *Clin Infect Dis* 2002;35:246–253.

7. Kageyama T, Kojima S, Shinohara M, Uchida K, Fukushi S, Hoshino FB, *et al.* Broadly reactive and highly sensitive assay for Norwalk-like viruses based on real-time quantitative reverse transcription-PCR. *J Clin Microbiol* 2003;41;4:1548–1557.

8. Greene SR, Moe CL, Jaykus LA, Cronin M, Grosso L, van Aarle P. Evaluation of the NucliSens® basic kit assay for detection of Norwalk virus RNA in stool specimens. *J Virol Methods* 2003;108:123–131.

9. Glass RI, Noel J, Ando T, Fankhauser R, Belliot G, *et al.* The Epidemiology of enteric caliciviruses from humans: a reassessment using new diagnostics [Review]. *J Infect Dis* 2000;181 Suppl 2:S254–S261.

10. Jothikumar H, Lowther J, Henshilwood K, Lees ND, Hill VR, Vinjé J. Rapid and sensitive detection of noroviruses by using TaqMan-based one-step reverse transcription-PCR assay and application to naturally contaminated shellfish samples. *Appl Environ Microbiol* 2005;1870–1875.

11. Noel JS, Fankhauser RL, Ando T, Monroe SS, Glass RI. Identification of a distinct common strain of 'Norwalk-like viruses' having a global distribution. *J Infect Dis* 1999;179:1334–1344.

12. Jansen A, Beyer A, Brandt C, Hohne M, Schreier E, Schulzke J, *et al.* The norovirus-epidemic in Berlin-clinic, epidemiology, and prevention. *Z Gastroenterol* 2004;42:311–316.

13. Centers for Disease Control and Prevention. Norovirus Activity—United States, 2002. *MMWR Morb Mortal Wkly Rep* 2003;52:41–45.

14. Lopman B, Vennema H, Kohli E, Pothier P, Sanchez A, Negredo A, *el al*. Increase in viral gastroenteritis outbreaks in Europe and epidemic spread of new norovirus variant. *Lancet* 2004;363:682–688.

15. Jiang X, Huang PW, Zhong M, Farkas T, Cubitt DW, Matson DO. Design and evaluation of a primer pair that detects both Norwalk- and Sapporo- like caliciviruses by RT-PCR. *J Virol Methods* 1999;83:145–154.

16. Thompson JD, Higgins DG, Gibson TJ. CLUSTAL W: improving the sensitivity of progressive multiple sequence alignment through sequence weighting, position-specific gap penalties and weight matrix choice. *Nucleic Acids Res* 1994;22:4673–4680.

17. Vinjé J, Vennema H, Maunula L, von Bonsdorff C, Hoehne M, Schreier E, *et al.* International collaborative study to compare reverse transcriptase PCR assays for detection and genotyping of noroviruses. *J Clin Microbiol* 2003;4:1423–1433.

Salmonella Typhimurium phage type 170 in a tertiary paediatric hospital with person-to-person transmission implicated

Noore KM Alam,[1,2] Paul K Armstrong,[3] Oanh TK Nguyen,[1] Alison M Kesson,[4] Terri M Cripps,[4] Stephen J Corbett[1]

Abstract

Nosocomially-acquired salmonellosis is uncommonly reported in Australia. We report a cluster of gastroenteritis caused by *Salmonella* Typhimurium phage type 170 (STm 170) centred on a tertiary paediatric hospital in Sydney, New South Wales from 8 to 19 May 2004. A total of 12 children had STm 170 isolated from faecal specimens. Of the 12 cases, seven were acquired in hospital and five in the community. The mean age of the cases was 4.1 years (range: 2 months to 11.2 years). We conducted a case series investigation to generate hypotheses regarding the cause of this outbreak. Standardised interviews with cases' parents were conducted to identify potential exposures including in recently consumed food. An environmental investigation mapped the food preparation and storage areas, movements of staff caring for cases, relative case-bed locations, and duration of stay in these locations. Five of the seven hospital-acquired cases were immunocompromised with a history of prolonged and/or multiple hospital admissions. We found that STm 170 was probably brought into the hospital by a community-acquired case and spread to other in-patients through person-to-person transmission by hospital staff and/or patients. This study emphasises the importance of stringent compliance with hospital infection control practices at all times. *Commun Dis Intell* 2005;29:374–378.

Keywords: Salmonella *Typhimurium phage-type 170, salmonellosis, outbreak, nosocomial infection, paediatric hospital*

Introduction

Salmonella species are a common cause of gastrointestinal illness in humans. *Salmonella enterica* serotype Typhimurium (STm) is the most frequently notified serotype in Australia. The most commonly reported phage type (PT) among *S.* Typhimurium is PT170. Notifications of STm 170 have increased in eastern Australia since 2002 and comprised 16 per cent of all salmonellosis notifications in New South Wales in 2004.[1]

Reported outbreaks of nosocomially-acquired salmonellosis are rare in Australia but can be serious and difficult to eradicate.[2] Nosocomially-acquired salmonellosis outbreaks are most commonly linked to contaminated food.[2–6] Person-to-person spread can readily occur in the hospital environment.[6,7] A 10-year (1978–1987) study of 248 outbreaks of nosocomially-acquired salmonellosis in the United Kingdom found 30 per cent of infections were linked to person-to-person spread.[4] Salmonellosis outbreaks in paediatric hospitals are of particular concern due to the increased susceptibility and associated high morbidity in this group.[8] Person-to-person transmission can prolong the duration of an outbreak, particularly when immunocompromised patients are involved.[5]

On 24 May 2004, a cluster of 12 cases of gastroenteritis caused by *Salmonella* species in in-patients of a tertiary referral paediatric hospital was reported to the Western Sydney Centre for Public Health, and an investigation was commenced. This outbreak is notable for two reasons: this is an uncommon nosocomial salmonellosis cluster linked to person-to-person transmission in Australia and, the *Salmonella* infections were acquired by mostly immunocompromised

1. Centre for Population Health, Sydney West Area Health Service, New South Wales
2. National Centre for Epidemiology and Population Health, Australian National University, Canberra, Australian Capital Territory
3. Communicable Diseases Branch, NSW Health, New South Wales
4. The Children's Hospital at Westmead, Sydney, New South Wales

Corresponding author: Mr Noore Alam, Centre for Public Health, Western Sydney Area Health Service, Locked Bag 7118, Parramatta BC, NSW 2150. Telephone: +61 2 9840 3603. Facsimile: +61 2 9840 3591. Email: Noore_Alam@wsahs.nsw.gov.au

hospital in-patients. This report documents the outbreak investigation, suggests possible means of transmission and describes the importance of hospital infection control measures.

Methods

Epidemiological investigation

We defined a probable case as any in-patient, staff member or visitor with laboratory-confirmed STm 170 isolated from a clinical specimen collected on or after 10 May 2004. A confirmed case was defined as either community-acquired or nosocomially-acquired depending on whether the case resided or worked in the hospital in the three days prior to the onset of symptoms. We checked with the Western Sydney Public Health Unit to ascertain whether or not this was part of a community-wide *Salmonella* outbreak. We also checked the Notifiable Diseases Database of NSW Health through the Health Outcomes Information Statistical Toolkit to compare the current pattern of notifications of STm 170. We collected and reviewed the inventory of hospital supplied food items. Parents confirmed which foods, if any, their children had eaten. The parents of all nosocomially-acquired cases and four of five community-acquired cases were interviewed using a standardised questionnaire. This questionnaire explored possible sources of infection during the 3-days prior to onset of symptoms. Sources investigated included food and water consumption, travel history, and environmental contacts such as animals and other ill people. We mapped both cases and staff movements within the hospital. To ascertain whether staff may have been vehicles for transmission, we examined hospital staff absenteeism data in May 2004 and traced staff contact with cases from clinical notes. Temporal and spatial relationships between the cases during likely incubation periods were examined by reviewing case-records.

Laboratory investigation

All initial *Salmonella* isolates were performed in the Department of Microbiology at The Children's Hospital at Westmead, New South Wales. These isolates were serotyped at the Institute of Clinical Pathology and Medical Research, Westmead. Phage typing of all isolates was carried out at the Microbiological Diagnostic Unit, Melbourne University, Melbourne, Victoria.

Environmental investigation

We inspected preparation, storage and handling of food in the hospital, including the preparation of infants formula and observed general food handling and cleanliness in the food preparation areas. Environmental sampling was not done as we considered it impractical nine days after the last reported case.

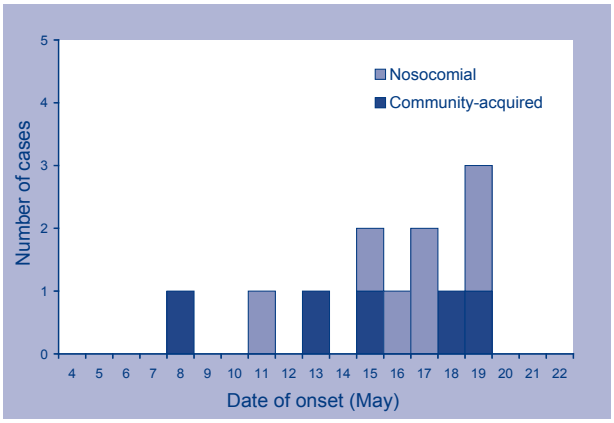
Results

Epidemiological investigation

Of the 12 STm 170 cases fitting the case definition, seven (58%) were hospital-acquired and five (42%) were community-acquired. Of the seven nosocomially-acquired cases, six were male and the mean age was 4.1 years. Nosocomially-acquired cases occurred between 11 and 19 May 2004, and community-acquired cases between 8 and 19 May 2004. Our local public health unit was not aware of any localised community-wide *Salmonella* outbreaks. The HOIST database however showed an upswing of notifications of STm 170 especially in metropolitan areas of New South Wales in the preceding months, which is consistent with the seasonal variations.[1] No links were found between the five community-acquired cases. Five of the seven nosocomially-acquired cases were immunocompromised. The background data on each of the cases are listed in the Table.

An epidemic curve for both community-acquired and nosocomially-acquired cases is shown in Figure 1.

Figure 1. *Salmonella* Typhimurium PT170 cases at a paediatric hospital, Sydney, May 2004, by date of onset and source of exposure



The characteristics of the epidemic curve do not suggest a point-source outbreak. Reviews of hospital supplied food items did not implicate food being the vehicle of transmission of STm 170. No food handlers were reported to have been sick during the period of investigation. Interviews with parents did not implicate particular food types as the source of the infection in either the nosocomially-acquired or community-acquired cases. No food samples were

Table. Distribution of age, dates of onset and admission, place of illness and underlying medical conditions of patients of STm 170 cases

Case number	Age (month)	Date of onset	Date of admission	Community-acquired (C) or nosocomially-acquired (N)	Underlying medical condition	Reason for admission
Case 1	36	8 May	10 May	C	Viral gastroenteritis	Diarrhoea, blood in stool
Case 2	6	11 May	4 May	N	Immune deficiency	Failure to thrive
Case 3	36	13 May	15 May	C	Viral gastroenteritis	Diarrhoea, vomiting
Case 4	42	15 May	12 May	N	Cardiac/juvenile arthritis	Pain in lower limb and abdomen, high fever
Case 5	2	15 May	16 May	C	Vomiting and nausea	Vomiting, nausea
Case 6	12	16 May	9 March	N	Liver disease	Liver transplant
Case 7	36	17 May	15 May	N	Acute laryngotracheitis	'Barking' cough, difficulty in breathing
Case 8	36	17 May	6 April	N	Leukaemia	Bone marrow transplant
Case 9	48	18 May	19 May	C	Congenital heart disease/ lower respiratory tract infection	Diarrhoea, vomiting, fever, cough
Case 10	132	19 May	20 May	C	Chronic headache	Fever, headache, vomiting, diarrhoea
Case 11	108	19 May	18 May	N	Rheumatic heart disease	Heart murmur, arthritis, rash, fever, haematuria
Case 12	96	19 May	13 May	N	Leukaemia	Bone marrow transplant

available for laboratory investigation. Three sub-clusters of two to four cases had possible common links; either the same staff member attended two or more cases within the infectious period and/or cases shared the same ward within their infectious period (Figure 2).

In each of the sub clusters we found circumstantial evidence—proximity in terms of time (infectious period) and place (ward)—of contact between community-acquired (n=5) and nosocomially-acquired cases (n=7), indicating likely person-to-person transmission. The index case (Case 1) was admitted to the hospital on 10 May with gastrointestinal illness later confirmed as STm 170. Case 4 was admitted on 12 May and shared a common ward with Case 1. Staff member 1 attended both patients within the infectious period. This may suggest a possible person-to-person spread either by the cases themselves or by the attending staff. Similarly, Staff member 2 attended Cases 2 and 4 within a 72-hour period. Case 4 also shared Staff member 1 with Case 1 within a 48-hour period. Case 5 and Case 7 shared the same ward (Ward 1) while Case 5 was probably infectious. Case 7 and Case 11 are both nosocomially-acquired Cases. Both stayed in the same ward on 18 May and shared Staff member 3 for 24 hours. Cases 8 and 12 also shared the same ward, and Staff member 4 attended both within 48 hours when Case 8 could still have been infectious. However, we do not have an objective explanation as to the source of acquisition of STm 170 for three in-patients; Cases 2, 6 and 8, all of whom had been hospital in-patients for a prolonged period (2 to 6 months). Our review of the hospital staff absenteeism data found no staff reported illness during the period of our investigation.

Environmental investigation

The hospital has a Hazard Analysis Critical Control Point (HACCP) system in operation which is responsible for monitoring potential points as a source of food contamination and taking early action to control food safety concerns from microbiological, chemical and physical hazards. The hospital had two food preparation areas for its in-patients; an infants formula preparation area, which was a protected sterile area and a separate kitchen where food was prepared for other in-patients. Our investigation of the food handling, preparation and storage did not reveal any obvious potential causes of *Salmonella* contamination.

Discussion

Nosocomial infections are a continual challenge to any healthcare system.[9] Foodborne salmonellosis outbreaks in a hospital environment have been previously reported.[3,6,8,10] However, *Salmonella* infections due to person-to-person transmission in paediatric healthcare facilities are only rarely reported.[7,11] In Australia, of the three reported *Salmonella* outbreaks linked to person-to-person spread in 2004,

(Martyn Kirk, personal communication, 2005) this is the only outbreak reported in the hospital environment. The notification of this STm 170 cluster, warranted prompt public health intervention because of the potentially serious consequences of this infection in already ill children, especially those who are immunocompromised.

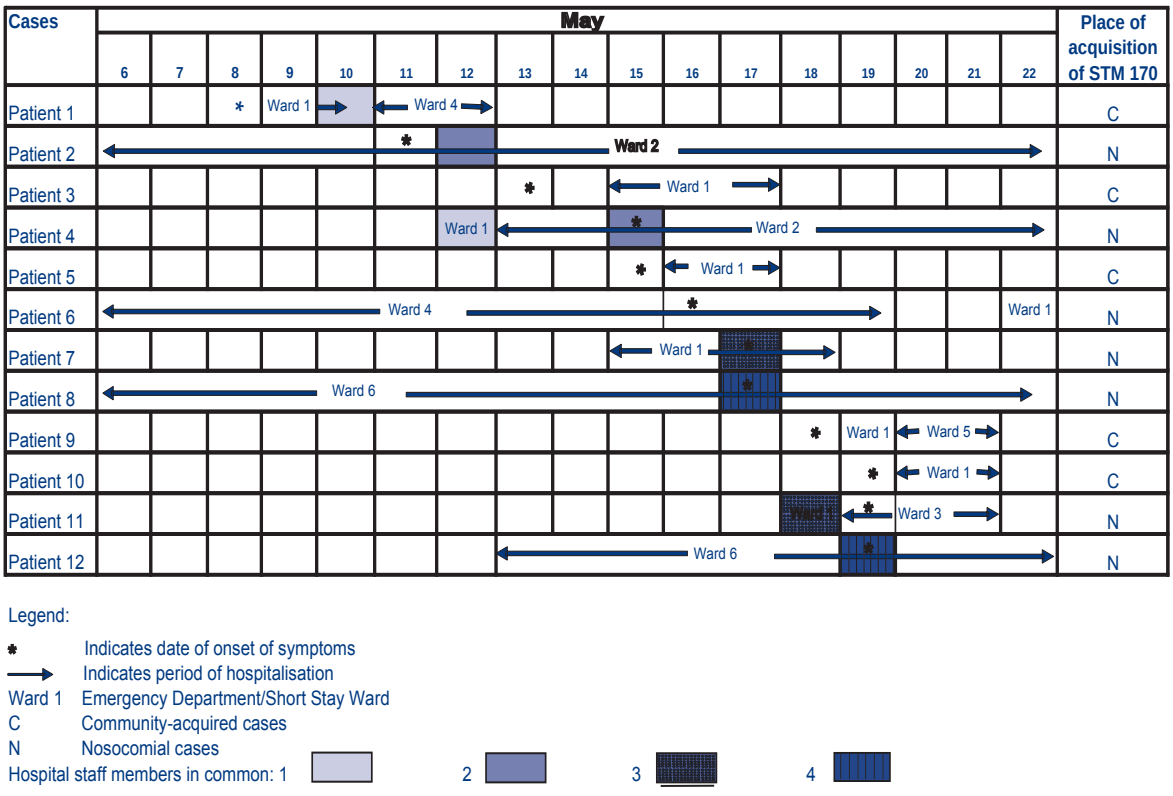
We hypothesise that this outbreak was the result of person-to-person transmission of infection from community-acquired cases to hospital in-patients. This is evidenced by both the temporal and spatial relationships between cases within the hospital and lack of evidence implicating food or any other common exposures. Analysis of the sub-clustering of cases by ward and hospital staff members who attended the cases strengthens the hypothesis that staff members contributed to person-to-person transmission (Figure 2).

The transmission of infection within the hospital is likely to have occurred as a result of direct contact between a case and a susceptible in-patient, or else facilitated by staff members attending both. Person-to-person transmission was most likely to occur at the time of children being in the Emergency Department (ED) or Short Stay Ward or SSW (Ward 1 shown in Figure 2) where patients staying longer than four hours in ED are transferred to SSW.

Although we did not investigate the hospital's infection control procedures for the management of diarrhoeal disease, stringent compliance with appropriate infection control policies are widely regarded as effective measures for prevention of nosocomial outbreaks of infectious diseases. As there is always *Salmonella* activity in the community, there is an ongoing risk of hospital outbreaks through introduction by community-acquired cases. During nosocomial outbreaks, instituting measures to enable the early detection of cases and to prevent ongoing spread are vital.[7] Methods to enhance surveillance are based around improving ward and laboratory staff's awareness, testing and reporting of in-patients with symptoms of gastroenteritis. Measures to prevent further spread include strict enforcement of hand-washing practices, cohorting or isolating infected patients, and enhanced cleaning and disinfection. We found that such measures were undertaken during the early stages of this outbreak and it is likely that this limited the duration of the outbreak.

The preliminary results of this investigation were communicated to the hospital infection control unit. The final report of this investigation was presented at a meeting of the hospital's infection control physicians on 22 June 2004. The hospital accepted the recommendations in terms of enhancement of infection control measures in all areas of the hospital and particularly in the ED and SSW areas at all times.

Figure 2. Epidemiological links between date of onset, ward and attending staff for STM 170 nosocomial and community-acquired cases



In summary, this report documents an outbreak of salmonellosis amongst paediatric hospital in-patients. The source of the outbreak was one or more community-acquired cases admitted to the hospital, with subsequent direct spread to in-patients or facilitated by staff members. Although the exact nature of the breakdown in infection control measures that lead to nosocomial transmission could not be ascertained, prompt action to enhance infection control measures terminated the outbreak. It reinforces the importance of maintaining strict infection control measures at all times in order to prevent outbreaks in vulnerable hospitalised populations.

Acknowledgements

Ms D Dalton and Ms K Hale, Infection Control; Ms L Luther, Food Services; Mr M Tuck and Mr M Kirk, OzFoodNet; Mr P Thomas, Sydney West Area Health Service.

References

1. Communicable Diseases Branch and Epidemiology and Research Branch. Notifiable Diseases Database. Health Outcomes Information and Statistical Toolkit, New South Wales Department of Health; 2004.

2. Spearing NM, Jensen A, McCall BJ, Neill AS, McCormack JG. Direct costs associated with a nosocomial outbreak of *Salmonella* infection: an ounce of prevention is worth a pound of cure. *Am J Infect Control* 2000;28:54–57.

3. McCall BJ, McCormack JG, Stafford R, Towner C. An outbreak of *Salmonella* Typhimurium at a teaching hospital. *Infect Control Hosp Epidemiol* 1999;20:55–56.

4. Joseph CA, Palmer SR. Outbreaks of *Salmonella* infection in hospitals in England and Wales 1978–87. *BMJ* 1989;298:1161–1164.

5. Matlow A, Streitenberger L. Nosocomial salmonellosis: implications for microbiologic processing of stools in hospitalized patients. *Am J Infect Control* 2001;29:65–66.

6. Bornemann RD, Zerr DM, Heath J, Koehler J, Grandjean M, Pallipamu R, *et al.* An outbreak of *Salmonella* serotype Saintpaul in a children's hospital. *Infect Control Hosp Epidemiol* 2002;23:671–676.

7. Umasankar S, Mridha EU, Hannan MM, Fry CM, Azadian BS. An outbreak of *Salmonella enteritidis* in a maternity and neonatal intensive care unit. *J Hosp Infect* 1996;34:117–122.

8. Matsuoka DM, Costa SF, Mangini C, Almeida GM, Bento CN, Van Der Heijden IM, *et al.* A nosocomial outbreak of *Salmonella enteritidis* associated with lyophilized enteral nutrition. *J Hosp Infect* 2004;58:122–127.

9. Rossello-Urgell J, Vaque-Rafart J, Hermosilla-Perez E, Allepuz-Palau A; EPINE Working Group. An approach to the study of potentially preventable nosocomial infections. *Infect Control Hosp Epidemiol* 2004;25:41–46.

10. Sion C, Garrino MG, Glupczynski Y, Avesani V, Delmee M. Nosocomial outbreak of *Salmonella enteritidis* in a university hospital (Letter to the editor). *Infect Control Hosp Epidemiol* 2000;21:182–183.

11. Cartolano GL, Moulies ME, Seguier JC, Boisivon A. A parent as a vector of *Salmonella* Brandenburg nosocomial infection in a neonatal intensive care unit. *Clin Microbiol Infect* 2003;9:560–562.

Investigation of a multi-state outbreak of *Salmonella* Hvittingfoss using a web-based case reporting form

Christopher J Oxenford,[1,2] Andrew P Black,[3] Robert J Bell,[4] Sally A Munnoch,[5] Melissa J Irwin,[6] Rachel N Hanson,[7] Rhonda L Owen,[2] and the Outbreak Investigation Team[8]

Introduction

Geographically dispersed outbreaks of disease pose challenges for investigators due to the difficulty of collecting and collating comparable data in a timely fashion. In Australia, when cases are detected in more than one jurisdiction, epidemiologists in different health departments must send results of their investigations by fax or email to a coordinating site for analysis. This process can result in considerable time delays and the possibility of data transcription errors. Web–based case reporting systems which house questionnaires on a secure web site allow investigators from multiple sites to enter interview data directly into the outbreak database. NetEpi Case Manager[1] is an open source software tool designed to assist with epidemiological investigations, analyses, and other aspects of public health practice. This system is currently being developed at the Centre for Epidemiology and Research in the New South Wales Department of Health. In 2004, OzFoodNet[2] trialled NetEpi for the Australian Government Department of Health and Ageing to assess its usefulness as an interim outbreak case reporting system for the Biosecurity Surveillance System.[3] The trial demonstrated the benefits of a web-based case reporting system and real-time access to collated uniform data. OzFoodNet decided to use NetEpi in future investigations of geographically dispersed outbreaks.

Between April and June 2005, Victoria reported an increase in the number of cases of *Salmonella enterica* ser Hvittingfoss (*S*. Hvittingfoss), with cases also detected in New South Wales, the Northern Territory, the Australian Capital Territory, Western Australia and southern Queensland. *S*. Hvittingfoss is a serovar often isolated from human, animal and environmental sources in northern Queensland, but is rarely identified in other parts of Australia. OzFoodNet used NetEpi to investigate this outbreak on behalf of the Communicable Diseases Network Australia.

Methods

An epidemic curve was constructed using *S*. Hvittingfoss notifications data from the National Notifiable Diseases Surveillance System. Hypothesis generating interviews were conducted with all notified cases of *S*. Hvittingfoss in the Australian Capital Territory, New South Wales, southern Queensland, Victoria and Western Australia whose symptom onset was between 24 April and 31 May 2005. Hypothesis generating interviews in each jurisdiction were conducted using a standard questionnaire with data being entered into a corresponding data form in NetEpi. Case name and address were not recorded. A unique identifier was created for each case using a name code and the residential post code of the case to allow individual cases to be linked to the

1. National Centre for Epidemiology and Population Health, Australian National University, Canberra, Australian Capital Territory
2. Australian Government Department of Health and Ageing, Canberra, Australian Capital Territory
3. Department of Health and Community Care, Canberra, Australian Capital Territory
4. Queensland Health, Brisbane, Queensland
5. Hunter New England Area Health Service, Newcastle, New South Wales
6. NSW Health Department, Sydney, New South Wales
7. Victorian Department of Human Services, Melbourne, Victoria
8. The Outbreak Investigation Team was (*in Alphabetical order*): Jenny Barralet (Qld), Robert Bell (Qld), Andrew Black (ACT), Gerard Fitzsimmons (DoHA), Joy Gregory (Vic), Rachel Hanson (Vic), Melissa Irwin (NSW), Martyn Kirk (DoHA), Karin Lalor (Vic), Dot Little (Hunter, New England), Tony Merritt (Hunter New England), Sally Munnoch (Hunter New England), Leonie Neville (NSW), Rhonda Owen (DoHA), Chris Oxenford (DoHA), Russell Stafford (Qld), Kay Sturge (Vic), Barbara Telfer (NSW), Hassan Vally (WA)

Corresponding author: Mr Chris Oxenford, Australian Government Department of Health and Ageing, GPO Box 9848 (MDP 15), Canberra ACT 2601. Telephone: +61 2 6289 9014. Facsimile: +61 2 6289 7104. Email: chris.oxenford@health.gov.au

notifiable diseases database in the respective jurisdiction. OzFoodNet Central Office epidemiologists at the Australian Government Department of Health and Ageing extracted the data and calculated the proportion of cases exposed to different food items.

A case-control study was conducted on newly notified cases with onset of symptoms after 1 June 2005 to measure any association between illness and the exposures most commonly identified in the hypothesis generating interviews. A case was defined as any person with *S*. Hvittingfoss isolated from faeces with onset of diarrhoea after 1 June 2005 who was not a resident of north Queensland. Cases must have been the primary case in the household and interviewed within 30 days of onset of diarrhoea. Using progressive digit dialling from the case's telephone number, three controls per case were enrolled. Cases and controls were interviewed by telephone using specific questionnaires with the results of the interviews being entered into case or control data forms in NetEpi. Data were extracted from NetEpi by the coordinating site and analysed using Stata release 8.0.[4]

Results

The epidemic curve showed an increase in cases of *S.* Hvittingfoss in multiple jurisdictions beginning in April 2005, with the peak of 42 cases in May 2005 being three and a half times the number of cases reported in May 2004 or 2003 (Figure).

Hypothesis generating interviews were conducted with 34 cases. These were in the Australian Capital Territory (3 cases), New South Wales (7 cases), southern Queensland (10 cases), Victoria (12 cases) and Western Australia (2 cases). The median age of cases was 15 years (range 1–87 years) and 46 per cent of cases were male. In addition to diarrhoea, other symptoms reported by cases included: lethargy (68%), abdominal pain (64%), fever (59%), vomiting (46%), and nausea (44%). Duration of illness ranged from 36 hours to more than 21 days. Eleven cases (32%) visited hospital and seven (20%) were admitted. A high proportion of cases reported consuming a range of fruits and vegetables in the 14 days prior to illness, including: bananas (80%), tomatoes (50%), strawberries (44%) mandarins (30%) and grapes (20%). We hypothesised that infection occurred as a result of consumption of fruits or vegetables grown in north Queensland where *S.* Hvittingfoss is endemic.

Eight cases were enrolled in the case-control study, four from New South Wales, two from Victoria and two from southern Queensland. The median age of cases was 42 years (range 6 months to 80 years) and 62 per cent were male. Twenty-four controls were recruited into the study. The median age of controls was 42 years (range two years to 94 years) and 37 per cent were male. No food items were significantly associated with *S*. Hvittingfoss infection (Table).

Figure. Notifications of *Salmonella* Hvittingfoss, January 2003 to August 2005, by jurisdiction

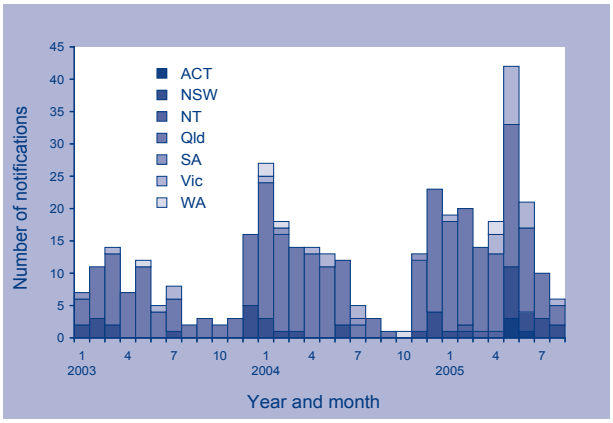


Table. Exposure to selected foods amongst *Salmonella* Hvittingfoss cases and controls in the three days prior to illness or interview, Australia, 2005

Exposure	Cases (exposed/n)	Controls (exposed/n)	Odds ratio	95 %CI
Apples	5/8	9/24	2.8	0.4–21.7
Bananas	5/8	10/24	2.3	0.3–18.1
Chicken	5/8	15/22	0.8	0.1–6.5
Lettuce	3/8	6/24	1.7	0.2–12.2
Strawberries	2/8	3/24	2.3	0.2–25.0
Sultanas	2/8	3/24	2.3	0.2–25.0
Watermelon	1/8	1/24	3.2	0.04–268.9

Discussion

Web–based case reporting systems offer advantages both for the coordinating site and the sites interviewing cases remotely. In our investigation, it was a dramatic improvement for the coordinating site to have direct access to the data over the manual system where data was emailed or faxed irregularly. The use of a common instrument with a consistent method for recording results also improved data quality during this multi-centre outbreak investigation.

Electronic communication is increasingly being used during outbreak investigations to share information such as organism profile patterns[5,6] or to disseminate information and questionnaires.[7,8] Additional web-based investigation techniques include contacting study participants by email and requesting them to complete questionnaires that are loaded onto a secure website.[9,10] Valuable time and resources can be saved using self-answered web-based tools but its usefulness will be limited by email (web) access.

No food vehicle was identified as being responsible for this outbreak. With only eight cases notified after the case-control study commenced the study lacked sufficient power to detect an association between illness and an exposure. It was decided not to include cases with onset of symptoms prior to 1 June 2005 due to the potential recall bias since cases had previously been interviewed extensively during hypothesis generating interviews. The majority of cases and controls were unable to recall specific brands of food, making measurement of exposure very non-specific.

In the future, public health investigations will increasingly use internet-based methods to collect data in outbreak settings.

References

1. NetEpi. Free, open source, network-enabled tools for epidemiology and public health practice. http://www.netepi.org Accessed on 5 October 2005.
2. OzFoodNet. A health network to enhance the surveillance of foodborne disease in Australia. http://www.ozfoodnet.org.au Accessed on 20 October 2005.
3. Biosecurity Surveillance System. http://www.health.gov.au/internet/wcms/publishing.nsf/content/biosecurity+surveillance+system-1 Accessed on 14 October 2005.
4. StataCorp. Stata Statistical Software: Release 8.0. College Station, Texas: Stata Corporation; 2003.
5. Crook PD, Aguilera JF, Threlfall EJ, O'Brien SJ, Sigmundsdottir G, Wilson D, *et al.* A European outbreak of *Salmonella enterica* serotype Typhimurium definitive phage type 204b in 2000. *Clin Microbiol Infect* 2003;9:839–845.
6. Kirk MD, Little CL, Lem M, Fyfe M, Genobile D, Tan A, *et al.* An outbreak due to peanuts in their shell caused by *Salmonella enterica* serotypes Stanley and Newport—sharing molecular information to solve international outbreaks. *Epidemiol Infect* 2004;132:571–577.
7. Passaro DJ, Scott M, Dworkin MS. E-mail surveys assist investigation and response: a university conjunctivitis outbreak. *Epidemiol Infect* 2004;132:761–764.
8. Boland M, Sayers G, Coleman T, Bergin C, Sheehan N, Creamer E, *et al.* A cluster of leptospirosis cases in canoeists following a competition on the River Liffey. *Epidemiol Infect* 2004;132:195–200.
9. Srikantiah P, Bodager D, Toth B, Kass-Hout T, Hammond R, Stenzel S, *et al.* Web-based investigation of multistate salmonellosis outbreak. *Emerg Infect Dis* 2005;11:610–612.
10. Burnett C. Use of a web-based questionnaire to investigate a large outbreak of GI illness. In: CDC/APHL National Foodborne Epidemiologists Meeting; 2005; Seattle, Washington, USA; 2005.

OzFoodNet: enhancing foodborne disease surveillance across Australia: Quarterly report, July to September 2005

The OzFoodNet Working Group

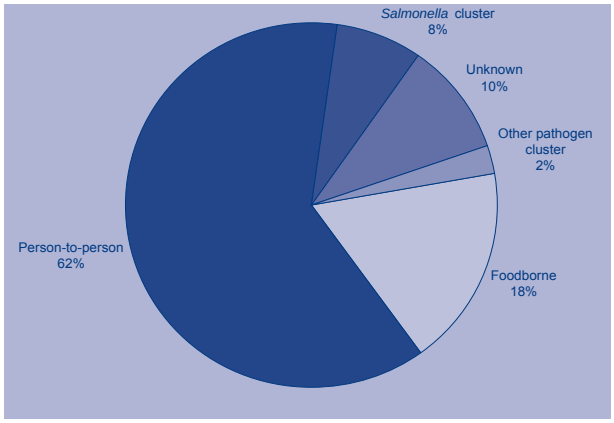
Introduction

The Australian Government of Health and Ageing established the OzFoodNet network in 2000 to collaborate nationally to investigate foodborne disease. OzFoodNet conducts studies on the burden of illness and coordinates national investigations into outbreaks of foodborne disease. This quarterly report documents investigations of outbreaks of gastrointestinal illness and clusters of disease potentially related to food occurring around the country.

This report summarises the occurrence of foodborne disease outbreaks and cluster investigations between July and September 2005. Data were received from OzFoodNet representatives in all Australian states and territories and a sentinel site in the Hunter/New England region of New South Wales. The data in this report are provisional and subject to change, as results of outbreak investigations can take months to finalise. We would like to thank 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 collected data used in this report.

During the third quarter of 2005, OzFoodNet sites reported 170 outbreaks of gastroenteritis. Outbreaks of gastroenteritis are often not reported to health agencies or the reports are delayed, meaning that these figures significantly under–represent the true burden of these infections. In total, these outbreaks affected more than 2,435 people and resulted in 90 persons being admitted to hospital. No deaths were reported. As has been the case in previous reports, the majority (62%, n=105) of outbreaks resulted from infections suspected to be spread by person-to-person transmission (Figure). Forty–two per cent of these person-to-person outbreaks occurred in aged care facilities, 38 per cent in child care centres and 10 per cent in hospitals.

Figure. Mode of transmission for outbreaks of gastrointestinal illness reported, July to September 2005, by OzFoodNet sites



Foodborne disease outbreaks

There were 30 outbreaks of illness where consumption of contaminated food was suspected or proven to be the primary mode of transmission. These outbreaks affected 209 people. This compares with 25 outbreaks for the third quarter 2004 and 27 outbreaks in the second quarter of 2005.

The OzFoodNet Working Group is *(in alphabetical order)*: Jenny Barralet (Qld), Robert Bell (Qld), Andrew Black (ACT), Barry Combs (SA), Craig Dalton (Hunter New England), Gerard Fitzsimmons (DoHA), Joy Gregory (Vic), Gillian Hall (NCEPH), Raj Patil (DAFF), Geoff Hogg (MDU), Melissa Irwin (NSW), Martyn Kirk (DoHA), Karin Lalor (Vic), Deon Mahoney (FSANZ), Peter Markey (NT), Tony Merritt (Hunter New England), Rosanne Muller (NT), Sally Munnoch (Hunter New England), Lillian Mwanri (SA), Chris Oxenford (DoHA, NCEPH), Rhonda Owen (DoHA), Nevada Pingault (WA), Jane Raupach (SA), Minda Sarna (WA), Cameron Sault (TAS), Robert Soloman (FSANZ), Russell Stafford (Qld), Chris Sturrock (FSANZ, NCEPH), Barbara Telfer (NSW), Kefle Yohannes (DoHA).

Corresponding author: Mr Martyn Kirk, Coordinating Epidemiologist, OzFoodNet, Australian Government Department of Health and Ageing, GPO Box 9848 (MDP 15), Canberra, ACT 2601. Telephone: +61 2 6289 9010. Facsimile: +61 2 6289 5100. Email: martyn.kirk@health.gov.au

All data are reported using the date the report was received by the health agency.

Ciguatoxin was responsible for four outbreaks and scombroid poisoning for three outbreaks during the quarter. *Campylobacter* and *Clostridium perfringens* were each responsible for two outbreaks. *Salmonella* Typhimurium 9, *Salmonella* Typhimurium 135a, *Salmonella* Infantis, *Staphylococcus aureus*, and norovirus each caused an outbreak during the third quarter of 2005. No aetiological agent was identified for the remaining 43 per cent (13/30) of outbreaks.

Sixteen of the outbreaks reported in the quarter were associated with meals served in restaurants, six with food prepared in private residences but includes four instances that can be attributed to the contamination of the primary produce (fish), three within aged care facilities, and two with food prepared by commercial caterers. Single outbreaks were associated with food prepared at a camp, a takeaway store, and an unknown setting. One of the outbreaks occurred in June, seven in July and eleven in both August and September.

To investigate these outbreaks, sites conducted six cohort studies and one case control study. For 23 outbreaks, only descriptive data were collected. Investigators obtained microbiological evidence linking a food vehicle to illness in two outbreaks and analytical epidemiological evidence in six outbreaks. For the remaining outbreaks, investigators obtained descriptive epidemiological evidence implicating the food vehicle or suggesting foodborne transmission.

In New South Wales there were 14 outbreaks of foodborne illness reported during the quarter. The aetiological agent was identified in three of these outbreaks. *Campylobacter jejuni* affected five people after they had eaten a restaurant meal that included chicken and lamb dishes. Ten residents in an aged care facility showed symptoms consistent with *Clostridium perfringens* infection after a meal of chicken with bacon and mushroom sauce, on rice. No food vehicle was identified for an outbreak of *Salmonella* Infantis that affected nine people during a camp. An aetiological agent was not identified in the remaining 11 outbreaks, nine of these involved restaurants and affected between two and eight patrons. An aged care facility (12 cases) and a private residence (11 cases) were the setting for the other two outbreaks in New South Wales during the quarter.

Queensland reported nine outbreaks of foodborne illness for the third quarter. Three outbreaks were caused by ciguatera fish poisoning following meals of Spanish mackerel (2 people), trevally (2 people) and black kingfish (5 people). All three fish were bought at retail outlets and then cooked at home. No food vehicle was identified in two outbreak investigations where *Campylobacter jejuni* infected three people after a common meal at an aged care facility and two people from the same household in September. A restaurant meal of yellow-fin tuna caused scombroid poisoning in two people. An outbreak affected three people after a restaurant meal of beef Rendang and their symptoms were consistent with *Clostridium perfringens* infection. *Clostridium perfringens* were detected at diagnostic levels in stool samples from two cases. The beef Rendang (food vehicle) was cooked on Sunday afternoons in large saucepans; stored for several days in a cold room and re-heated as required. A meal of chips and gravy from a takeaway hot food shop led to *Staphylococcus aureus* infection in two people who had staphylococcal enterotoxin detected in faecal specimens.

During the quarter, Queensland reported an outbreak of *Salmonella* Typhimurium 9 among students and teachers visiting from the United States of America. Eggs used in a dessert were suspected to have caused this outbreak and affected 40 cases (31 students, 3 teachers,1 tour guide and 5 restaurant staff) with 29 people requiring treatment in hospital. Contamination of the eggs and inadequate cooking temperature for the desert are the two main features that most likely contributed to the outbreak.

Victoria reported five outbreaks of foodborne disease for the quarter. There were two outbreaks of scombroid poisoning: one in July at a restaurant affected two people after a meal of tuna, and the other in August from fish (species unknown) purchased at a retail outlet and cooked at home which led to illness in two people. Snapper fillets purchased from a Fiji market and transported frozen to Australia by a relative caused five cases of ciguatera poisoning in a family. An aetiological agent was not identified in the remaining outbreak involving 11 people who had eaten Spanish mackerel from a restaurant in September. An outbreak of norovirus affected at least 36 people who had consumed assorted sandwiches at a catered function.

The Australian Capital Territory reported two foodborne outbreaks. An outbreak affected two separate groups that had dined at a Sydney restaurant, two people from one group and one from the other group became ill. *Salmonella* Typhimurium 135a infection caused the outbreak, but investigators were unable to identify the food responsible for the outbreak. In the second outbreak, at least seven people became ill following a catered function. Those that were ill were more likely to have eaten strawberries, smoked salmon and/or grapes.

Comments

During the third quarter of 2005, there were seven identified outbreaks of fish poisonings. Six of these were from fish purchased from retail outlets. It is a concern that ciguatera cases have resulted from commercial suppliers, as most cases in recent years

have occurred in association with fish caught by amateur fishermen.[1] The three outbreaks of histamine poisoning were associated with fish consumed at restaurants (2 outbreaks) and fish bought at a retail outlet and cooked at home (1 outbreak). At least two of these were tuna originating from Indonesia. It is likely that there would have been many more cases of scombroid poisoning occurring, as the illness is often mild and difficult to recognise.[2]

There were two outbreaks of *Clostridium perfringens* and another outbreak where this pathogen was suspected as the cause. Two of these outbreaks occurred in aged care facilities where *Cl. perfringens* is a common cause of outbreaks of diarrhoea amongst residents in these settings. The third outbreak in Queensland involved a curry that was stored for several days in a cool room and re-heated as required. *Cl. perfringens* commonly causes outbreaks where there is poor temperature control of meals, such as curries.[3]

During the quarter, OzFoodNet continued an investigation into a multi-state outbreak of *Salmonella* Hvittingfoss occurring in Queensland, New South Wales, the Australian Capital Territory and Victoria. No source for the outbreak was identified. For further information see the summary of the investigation in this issue of *Communicable Diseases Intelligence*.[4] Jurisdictions conducted 13 other investigations into time, place, person clustering of *Salmonella* infections, including serotypes Anatum, Bovismorbificans 24, Muenchen, Newport, Oranienburg, Poona, Saintpaul, Typhimurium 12, Typhimurium 135a, Typhimurium 170, Typhimurium 195, Typhimurium U302, Typhimurium U307. The Northern Territory also reported an increase in shigellosis, and hepatitis A, while South Australia reported an increase in hepatitis A in remote areas of the state. Victoria reported clustering of cryptosporidium, including three small outbreaks, two of which were associated with swimming pools and the other with touching animals at a festival.

References

1. OzFoodNet Working Group. Reported foodborne illness and gastroenteritis in Australia: annual report of the OzFoodNet network, 2004. *Commun Dis Intell* 2005;29:165–192.

2. Attaran RR, Probst F. Histamine fish poisoning: a common but frequently misdiagnosed condition. *Emerg Med J* 2002;19:474–475.

3. Dalton CB, Gregory J, Kirk MD, Stafford RJ, Givney R, Kraa E, *et al*. Foodborne disease outbreaks in Australia, 1995 to 2000. *Commun Dis Intell* 2004;28:211–224.

4. Oxenford C, Black A, Bell R, Munnoch S, Irwin M, Hanson R, *et al*. Investigation of a multistate outbreak of *Salmonella* Hvittingfoss using a web based case reporting form. *Commun Dis Intell* 2005:29:379-381.

Table 1. Outbreaks of foodborne disease reported by OzFoodNet sites,* July to September 2005

State	Month of outbreak	Setting prepared	Infection	Number affected	Evidence	Responsible vehicles
ACT	July	Restaurant	*Salmonella* Typhimurium 135a	3	D	Unknown
	August	Caterer	Unknown	7	A	Strawberries, smoked salmon, grapes.
NSW	June	Restaurant	Unknown	8	D	Unknown
	July	Restaurant	Unknown	2	D	Unknown
		Restaurant	Unknown	2	D	Common foods included steamed rice, miso, and chicken yakatori.
	August	Restaurant	Unknown	6	D	Unknown
		Restaurant	Unknown	2	D	Suspected chicken, rice or chilli sauce
		Restaurant	*Campylobacter jejuni*	5	D	Suspected chicken or lamb dishes
		Restaurant	Unknown	3	D	Unknown
		Aged care	*Clostridium perfringens*	10	D	Chicken with bacon and mushroom sauce, on rice
		Aged care	Unknown	12	D	Pureed corned beef dish
	September	Restaurant	Unknown	9	A	Ham or ham and pineapple pizza
		Restaurant	Unknown	2	D	Suspected beef steak
		Camp	*Salmonella* Infantis	5	D	Unknown
		Home	Unknown	11	D	Unknown
		Restaurant	Unknown	5	D	Suspect hot roast meats from carvery
Qld	July	Restaurant	*Clostridium perfringens*	3	M	Beef Rendang
		Restaurant	*Salmonella* Typhimurium 9	40	A	Bread and butter pudding
		Restaurant	Scombrotoxin	2	D	Yellow-fin tuna
	August	Home	Ciguatoxin	2	D	Spanish mackerel
	September	Takeaway	*Staphylococcus aureus*	2	M	Chips and gravy
		Aged care	*Campylobacter jejuni*	3	D	Unknown
		Home	Ciguatoxin	2	D	Trevally
		Home	Ciguatoxin	5	D	Black kingfish
		Unknown	*Campylobacter jejuni*	2	D	Unknown
Vic	July	Restaurant	Scombrotoxin	2	A	Tuna
	August	Caterer	Norovirus	36	A	3 assorted types of sandwiches
		Home	Ciguatoxin	5	D	Fijian snapper
		Home	Scombrotoxin	2	D	Fish (unknown species)
	September	Restaurant	Unknown	11	A	Spanish mackerel

* No foodborne outbreaks reported in South Australia, Tasmania, Western Australia or Northern Territory during the quarter.

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

A Analytical epidemiological association between illness and one or more foods;

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

Epidemiological features and control of an outbreak of scarlet fever in a Perth primary school

Kynan T Feeney,[1] Gary K Dowse,[2] Anthony D Keil,[3] Christine Mackaay,[4] Duncan McLellan[5]

Abstract

Scarlet fever was associated with feared outbreaks and mortality in the 19th Century. It occurs sporadically in modern society and infection is readily treated with antibiotics. We report on a scarlet fever outbreak in children attending a primary school in Perth, Western Australia, in late 2003. A total of 13 cases were identified over a five week period. Six of the cases were pre-primary children (ages 4 to 5) from the same class of 26 children (attack rate 23.1%). Three of the remaining seven cases were older siblings of pre-primary cases who developed scarlet fever after their younger siblings. Screening of the children and teachers from the two pre-primary classes at the school yielded 12 positive pharyngeal swabs for group A S*treptococcus*. *Emm*-typing of the screening isolates indicated that a common strain was circulating within the outbreak pre-primary class, with four of six isolates identified as *emm*-type 3. The overall group A *Streptococcus* carriage rate in screened students in this class was 31.6 per cent and the carriage rate for *emm*-type 3 was 21.1 per cent. Carriers were treated with oral penicillin V to eradicate carriage and control the outbreak. No further cases of scarlet fever were reported after the treatment of pharyngeal carriers. Outbreaks of scarlet fever still occur in young children and identification and treatment of carriers may still be valuable. *Commun Dis Intell* 2005;29:386–390.

Keywords: scarlet fever, Streptococcus, emm-*typing*

Introduction

Scarlet fever is caused by infection with group A *Streptococcus* (GAS), usually in the context of acute pharyngitis. It is characterised by a distinctive erythematous blanching rash that is composed of fine papules that cause the skin to feel like sandpaper and usually starts on the upper trunk, travelling distally and sparing the soles and hands.[1] Other characteristic clinical features of scarlet fever include: an initial white covering of the tongue, followed by enlargement of the papillae, giving a distinctive 'strawberry tongue' appearance; flushing of the cheeks; and circumoral pallor.[1] Although rarely seen in present times, scarlet fever can be complicated by localised extension of infection leading to mastoiditis, sinusitis and peritonsillar abscesses and can be followed by serious complications, such as acute rheumatic fever.[2]

During the 19th Century scarlet fever was a feared disease due to its infectivity and associated high mortality rate.[3–5] Severe scarlet fever is now uncommon and usually causes only mild disease, with many developed countries removing it from their notifiable disease registers over the past few decades.[2,3,6]

Group A *Streptococcus* is most commonly transmitted through direct contact or large droplet spread and the incidence of infection is highest in young children.[7] Administration of antibiotics will prevent transmission of GAS within 24 hours of beginning treatment.[1]

We describe the clinical and epidemiological features of a recent outbreak of scarlet fever in young children attending a Perth primary school, and the public health measures that were implemented to control the outbreak.

1. Public Health Registrar, Department of Health, Perth, Western Australia
2. Medical Epidemiologist, Communicable Disease Control Directorate, Department of Health, Perth, Western Australia
3. Head, Department of Microbiology, Women's and Children's Health Service, Subiaco, Western Australia
4. Communicable Diseases Coordinator, North Metropolitan Public Health Unit, Perth, Western Australia
5. Infectious Diseases Physician and Clinical Microbiologist, Fremantle Hospital and Western Diagnostic Pathology, Perth, Western Australia

Corresponding author: Dr Kynan Feeney, Communicable Disease Control Directorate, Department of Health, Western Australia, PO Box 8172, Perth Business Centre, WA 6849. Telephone: +61 8 9431 0217. Facsimile: +61 8 9431 0222.
Email: Kynan.Feeney@health.wa.gov.au

Methods

Scarlet fever remains a notifiable disease in Western Australia. Following notification of several cases of scarlet fever in children from the same primary school in Perth, Western Australia, an epidemiological investigation was performed, with a view to initiating control measures.

Case definition

Scarlet fever cases in school students and staff of the school, or family members of a child from the school who met the case definition, were categorised as possible, probable or confirmed, if they developed symptoms between 30 October and 30 November 2003 and fulfilled the following criteria:

Confirmed case: Clinical symptoms consistent with streptococcal sore throat AND at least one characteristic sign of scarlet fever AND a positive laboratory isolate of GAS from a throat swab. The characteristic signs of scarlet fever, as documented from a medical examination or reported by the child's parents, included:

- skin rash – fine erythematous, punctate, blanching on pressure and with a sandpaper texture and predominantly trucal distribution;
- strawberry tongue;
- flushing of cheeks and circumoral pallor; and
- desquamation of the skin in convalescence.

Probable case: Clinical symptoms consistent with streptococcal sore throat AND at least one characteristic scarlet fever sign AND no throat swab performed or throat swab performed and had no significant growth.

Possible case: Clinical symptoms consistent with a streptococcal sore throat AND no characteristic signs of scarlet fever AND no throat swab performed or throat swab performed and had no significant growth.

Cases were identified following notification by a doctor, or through telephone interviews of parents of children, or staff members, who were reported to have been absent from school during the period 30 October to 30 November 2003.

Screening of students and teachers in class 1 (pre-primary class, ages 4 to 5 years) and class 2 (split pre-primary/year 1 class, ages 4 to 6 years) was performed at the school on 2 December and 4 December 2003. As most of the cases were in the pre-primary age group and class 1 and 2 were sometimes joined together for group activities, screening of children in both class 1 and 2 was performed to identify possible carriers.

Screening consisted of swabbing the posterior pharynx and tonsils using a dacron swab (Transtube© Medical Wire and Equipment, UK) by two of the authors (KF and CM). Swabs were stored at room temperature and transported to the microbiology laboratory on the day of screening.

Informed consent was obtained during the outbreak to obtain samples and administer antibiotics. Other data were obtained through the notifiable diseases register and school absentee records.

Laboratory methods

Diagnosis of GAS carriage was made by culturing swabs within four hours of collection onto Columbia agar (Oxoid, Australia) with 5 per cent horse blood containing colistin 0.75 mg/L and nalidixic acid 0.5 mg/L (CNA) plates. CNA plates were incubated at 37° C for 24 hours in 5 per cent CO_2. After primary plating, swabs were then placed into Todd Hewitt broth (Oxoid, Australia) with colistin 1 per cent mg/L and nalidixic acid 1 mg/L for enrichment culture for 24 hours at 37° C. Following incubation, an aliquot was subbed onto CNA plates for a further 24 hours incubation at 37° C in 5 per cent CO_2. Beta haemolytic colonies were serogrouped using a Streptococcal Grouping Kit (Oxoid, Australia).

Epidemiological surveillance of GAS infection has traditionally depended on the Lancefield M protein serotyping system. There are a number of limitations with this approach, in particular the high proportion of isolates in Australia which are M-non-typeable.[8,9] To overcome these limitations a non-serologic typing system that involves sequencing the 5' end of the *emm* gene (*emm*-typing) was utilised in this study. This molecular typing system is based on the established relationship between published *emm* sequences and M serology and sensitively reflects the M specificity of the isolate.[10,11]

The *emm*-typing of isolates was performed by one author (DM) who was blinded to the school class of origin and clinical status of the source of each of the isolates.

Results

Cases

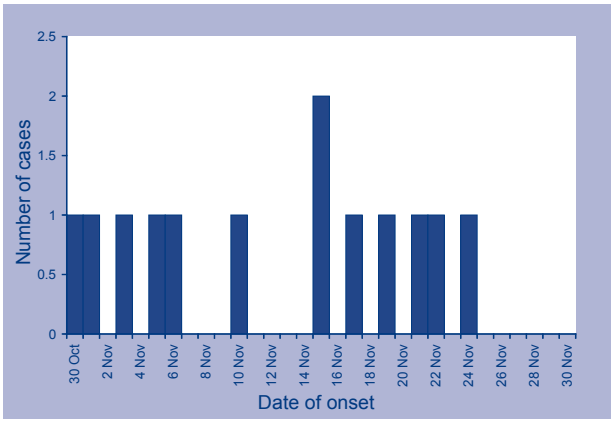
Based on notification data and interviews, there were three confirmed cases, 10 probable cases (of which eight had no throat swabs performed and two had negative throat swabs) and one possible case (Table 1). The low number of confirmed cases reflected the low rate of collection of microbiological samples to confirm GAS infection by general practitioners. Both confirmed and probable clinical cases (13 in total) are included together as cases for all subsequent analyses.

Table 1. Summary of epidemiological features of an outbreak of scarlet fever in a primary school, Perth, 2003

Variable	Number
Total number of cases:	
Confirmed	3
Probable	10
Possible	1
Background of confirmed and probable cases:	
Class 1 pre-primary students	6
Siblings of cases from class 1	3
Sibling of non-case child in class 1 (neither confirmed or probable case)	1
Student cases with no relationship to any students in class 1	2
Teacher case with no relationship to any students in class 1	1

As can be seen from the epidemic curve (Figure), the first case became symptomatic on 31 October, followed by a series of cases of scarlet fever spreading in a propagated outbreak pattern over the subsequent four weeks. The predominant clinical features experienced by the cases were sore throat (100%), fever (93%), rash (85%), headache (61%), abdominal pain (38%), strawberry tongue (31%), vomiting (31%) and skin desquamation (8%).

Figure. Epidemic curve showing number of scarlet fever cases, Perth, 2003, by date of onset of symptoms



The cases were composed of 12 children and one teacher. Six of the child cases were from class 1, giving an overall attack rate of 20 per cent (6 cases out of 26 students and 4 teachers). Among students only, the attack rate was 23.1 per cent. There were no cases in class 2.

Three of the remaining six child cases were siblings of the pre-primary cases from class 1. Two of these cases were in year 3 (aged 8 and 9 years) and one case was in year 2 (aged 7 years). All three of the older sibling cases developed symptoms of infection after their younger pre-primary sibling developed symptoms, with intervals between onset of 4, 5 and 20 days.

Of the three remaining child cases, one was in year 2 and had a younger pre-primary sibling in class 1. The latter child was the single 'possible' case who did have an upper respiratory tract illness but lacked specific signs of scarlet fever. The older sibling case developed scarlet fever after her pre-primary 'possible' sibling case was reported to be ill, suggesting the 'possible' pre-primary case from class 1 may have had GAS pharyngitis. The two remaining children, who were in years 3 (aged 9) and 5 (aged 11), did not have siblings in class 1.

The teacher with scarlet fever was not a pre-primary teacher. Her only contact with pre-primary children was during lunchtime. She had two young children, neither of whom attended the outbreak primary school or had experienced a recent illness consistent with scarlet fever or GAS pharyngitis.

Carriage studies

Table 2 summarises the epidemiological findings from classes 1 and 2. Although class 1 and 2 are in separate rooms, the two classes have intermittent combined teaching time in one room.

A total of 48 out of a possible 57 students and staff from the two classes were screened, consisting of 41 (of 50) children and all 7 teachers. Eleven of 41 students (26.8%) and one of seven (14.3%) teachers had positive throat swabs. All 12 positive throat swabs grew GAS on primary culture.

Class 1 had six children with positive swabs (carriage rate 23.1%), two of which were from previous cases of scarlet fever who had been treated with oral penicillin. The positive screening tests from class 2 were from five children (carriage rate 20.1%) and one teacher (carriage rate 33%). A course of oral penicillin V for 10 days was taken by all GAS carriers in an attempt to eradicate carriage of GAS within this population and terminate the outbreak.

Eleven of the 12 isolates were *emm* typeable: five from class 1 and six from class 2. As summarised in Table 2, four of six isolates from pre-primary class 1 were *emm*-type 3, giving a carriage rate in screened students of 21.1 per cent. Among the six isolates from class 2, two were *emm*-type 3, two were *emm*-type 2 and there was one each of *emm*-types 28 and 75.

Table 2. Epidemiological features of class 1 and 2 from affected primary school

Variable	Class 1	Class 2
Age of students	4-5 years	4-6 years
Number in each class:		
Students	26	24
Teachers	4	3
Number of cases:		
Confirmed	2	0
Probable	4	0
Attack rates:		
Students and teachers	20.0%	0%
Students only	23.1%	0%
Number screened:		
Students		
Total	19	22
Percentage positive	31.6%	22.7%
Number positive	6	5
Teachers		
Number	4	3
Percentage positive	0%	33.3%
Number positive	0	1
emm type results from throat swab screening:		
Not able to be typed	1	0
emm type:		
2	0	2
3	4	2
12	1	0
28	0	1
75	0	1

Discussion

This article reports on a school-based outbreak of scarlet fever. There were 13 confirmed or probable cases identified over a five week period. Six of these cases were from class 1 and the epidemiological and microbiological evidence suggests that this class was the epicentre for this outbreak, with most of the additional cases occurring in older siblings of children from this class.

Our investigation was limited to staff and students who attended the one primary school. As telephone questionnaires were undertaken with staff and parents of children with sick absences over the study period, we do not believe a significant number of cases were missed during the investigation.

The finding of a common *emm*-type (type 3) in four of five typeable isolates from children in the affected class suggests this was the circulating outbreak strain. Further evidence to support this theory is that of the two previously treated cases who subsequently tested positive when screened, one had an untypable isolate and the other was *emm*-type 3. This case had a short 5-day course of oral penicillin V, which is not considered as reliable as a ten day course of penicillin to reliably eradicate GAS from the pharynx.[12]

The other *emm*-types detected from screening of students and teachers were likely due to a carriage state of non-epidemic GAS. The prevalence of pharyngeal carriage of GAS can be up to 15 per cent outside of the outbreak setting.[6] Asymptomatic pharyngeal carriage can be higher during institutional outbreaks, with rates of 20–30 per cent reported.[6,13,14] Although asymptomatic pharyngeal carriers are not efficient transmitters of infection, screening and treatment of carriers is generally recommended in the outbreak setting of invasive GAS infection.[1,6,13] In this instance, 21.1 per cent (4 of 19) of students in class 1 were carrying *emm*-type 3 at the time of screening, even after five of the 19 screened students had been treated for scarlet fever.

Because of ongoing transmission of GAS causing scarlet fever within the school and family setting, the concern of parents and staff at the school, and the possible serious complications of GAS infection, it was decided to screen students and staff of the affected pre-primary school classes and treat asymptomatic carriers with the aim of interrupting transmission and preventing further cases.

No further cases of scarlet fever were notified from the primary school after screening and treatment of carriers was undertaken. Although we cannot be certain that the outbreak would not have terminated naturally at that time, the abrupt cessation suggests that the intervention of screening and administration of antibiotics to carriers of GAS was effective in halting the epidemic.

Scarlet fever is an uncommon infectious disease in Australia. There was a mean of 17.7 cases of scarlet fever notified in Western Australia per year from 1995–2004 (range 6–27). This outbreak was brought to the attention of the Department of Health Western Australia due to a small clustering of notified cases in children attending the same primary school. It is debatable whether scarlet fever should still be listed as a notifiable disease in Australia, however this outbreak and the subsequent control measures demonstrate there is some utility in continuing to monitor this infection.

Acknowledgments

We gratefully acknowledge the staff, students and families for their support during the investigation and management of the outbreak. We wish to thank the nursing staff, Ms Leanne Brown of the Department of Microbiology, Women's and Children's Health Service, Perth, Western Australia, for GAS culture and serogrouping, and the Molecular Diagnostics Unit at the PathCentre, Perth, Western Australia for performing the *emm*-genotyping.

References

1. Heymann DL, editor. *Control of Communicable Diseases Manual* 18th edn. Washington: American Public Health Association, 2004.

2. Stevens DL. Life-threatening streptococcal infections: scarlet fever, necrotizing fasciitis, myositis, bacteremia, and streptococcal toxic shock syndrome. In: Stevens DL, Kaplan EL, editors. *Streptococcal Infections: clinical aspects, microbiology, and molecular pathogenesis.* New York: Oxford University Press; 2000. p. 163–179.

3. Efstratiou A. Group A streptococci in the 1990s [Review]. *J Antimicrob Chemother* 2000;45:3–12.

4. Denny FJ. History of Hemolytic Streptococci and Associated Diseases. In: Stevens DL, Kaplan EL, editors. *Streptococcal Infections: clinical aspects, microbiology, and molecular pathogenesis.* New York: Oxford University Press; 2000. p. 1–18.

5. Duncan CJ, Duncan SR, Scott S. The dynamics of scarlet fever epidemics in England and Wales in the 19th century. *Epidemiol Infect* 1996;117:493–499.

6. Pickering L, Baker C, Overturf G, Prober C, eds. Red Book, 2003. Report of the Committee on Infectious Diseases. 26th edn. Elk Grove Village: American Academy of Pediatrics; 2003.

7. Stevens DL. Group A Beta-Hemolytic Streptococci: virulence factors, and spectrum of clinical infections. In: Stevens DL, Kaplan EL, editors. *Streptococcal Infections: clinical aspects, microbiology, and molecular pathogenesis.* New York: Oxford University Press; 2000. p. 19–36.

8. Relf WA, Martin DR, Sriprakash KS. Identification of sequence types among the M-nontypeable group A streptococci. *J Clin Microbiol* 1992;30:3190–3194.

9. Maxted W. Disease association and geographical distribution of the M-types of group A *Streptococcus*. In: Read, SE, Zabriskie, JB, eds. *Streptococcal diseases and the immune response.* New York: Academic Press; 1980. p. 763–777.

10. Beall B, Facklam R, Thompson T. Sequencing *emm*-specific PCR products for routine and accurate typing of group A streptococci. *J Clin Microbiol* 1996;34:953–958.

11. Beall B, Facklam R, Hoenes T, Schwartz B. Survey of *emm* gene sequences and T-antigen types from systemic *Streptococcus pyogenes* infection isolates collected in San Francisco, California; Atlanta, Georgia; and Connecticut in 1994 and 1995. *J Clin Microbiol* 1997;35:1231–1235.

12. Schwartz B, Marcy SM, Phillips WR, Gerber MA, Dowell SF. Pharyngitis – principles of judicious use of antimicrobial agents. *Paediatric* 1998;101:171–174.

13. Humes H, ed. *Kelley's Textbook of Internal Medicine.* 4th edn. Philadelphia: Lippincott Williams and Wilkins; 2000.

14. Falck G, Kjellander J. Outbreak of group A streptococcal infection in a day-care center. *Pediatr Infect Dis J* 1992;11:914–919.

The epidemiology of kuru in the period 1987 to 1995

Michael P Alpers [1,2,3] for the Kuru Surveillance Team[1,4]

Abstract

Kuru is an encephalopathy or neuro-degenerative disease found only in the Okapa District of the Eastern Highlands Province of Papua New Guinea. It is always fatal, with a subacute course, on average, of about 12 months from onset to death. In the 9-year period 1987 to 1995 there were 66 deaths from kuru, 17 males and 49 females. The number of deaths per year ranged from 3 to 12. All deaths occurred south of a line drawn through the centre of the kuru region perpendicular to the axis of social change. The mean age at death was 49 years, with a gradual increase in this age with time. The last patient aged in their 20s died in 1987 and the last in their 30s died in 1991. The period shows a waning epidemic, with dramatically fewer deaths than in the early years of epidemiological surveillance 30 years before. Nevertheless, the clinical features and duration of the disease were unchanged. Transmission of kuru stopped by 1960 and patients seen in the period 1987–1995 showed long incubation periods, which in 1995 would have been at least 35 years. The proportion of males was much higher than in the early years; because males were effectively exposed only in childhood their incubation periods were in many cases likely to be over 50 years. The work of the Kuru Surveillance Team in maintaining a rigorous surveillance of kuru epidemiology over this period is described. *Commun Dis Intell* 2005;29:391–399.

Keywords: kuru, epidemiology, prion diseases, transmissible spongiform encephalopathies, Papua New Guinea, field surveillance methodology

Introduction

Kuru is an encephalopathy or neuro-degenerative disease found only in the Okapa District of the Eastern Highlands Province of Papua New Guinea. It is always fatal, with a subacute course, on average, of about 12 months from onset to death. Kuru was unknown to the outside world until the 1950s when this area of the highlands was first brought under administrative control by the Australian government. Scientific investigation of kuru began in 1957.[1] From the evidence of oral history, the epidemic began in the early 1900s with a single case of kuru and slowly spread in extent and expanded in number until over 200 people were dying of the disease each year. In all, 172 villages are recorded as having a history of kuru; they occupy an area surrounding the Okapa government station that measures approximately 65 km by 40 km (the kuru region). In 145 of these villages, deaths from kuru have been recorded since the beginning of 1957 resulting in more than 2,700 deaths. Over 80 per cent of these occurred in the people of the Fore linguistic group; the remainder were in nine linguistic groups adjacent to the Fore with close cultural and marital ties with their Fore neighbours. In the initial years of kuru investigation, the disease occurred principally in women, and children and adolescents of both sexes, with only three per cent in adult males. Subsequently the disease disappeared in children and, later, in adolescents. The data presented in this paper determine, for the first time, the years in which kuru was no longer seen in adults in their third and fourth decades.

1. Papua New Guinea Institute of Medical Research, PO Box 60, Goroka, EHP 441, Papua New Guinea
2. Centre for International Health, Curtin University of Technology, Perth, Western Australia
3. MRC Prion Unit, Institute of Neurology, University College London, The National Hospital for Neurology and Neurosurgery, London, United Kingdom
4. The Kuru Surveillance Team from 1987 to 1995 consisted of Michael Alpers, Auyana Winagaiya, Anua Senavaiyo (now deceased), Igana Aresagu, Kabina Yaraki and John Anuwa, with team member-in-training Ausa Igana, the occasional assistance of Umasa Pave and Eric Yaburo in particular communities, and the participation of many members of the village communities of the kuru region.

Corresponding author: Professor Michael Alpers, Centre for International Health, Shenton Park Campus, Curtin University of Technology, GPO Box U1987, Perth, WA 6845. Telephone: +61 8 9266 4733. Facsimile: +61 8 9266 2608.
Email: m.alpers@curtin.edu.au

Clinically, kuru is a progressive cerebellar disorder.[1–5] It begins with mid-line cerebellar signs of titubation, trunkal instability and tremor, with astasia and a wide-based, ataxic gait. This progresses to dysmetria in the limbs, jerky extraocular movements, dysphagia and dysarthria. The clinical state of patients with kuru worsens steadily and inexorably through ambulant, sedentary and recumbent stages to a terminal state in which all motor activity is so uncoordinated as to be non-functional, though there is no paralysis. Patients are unable to move, eat or speak but are conscious, make eye contact and attempt appropriate responses to their environment as they wait to die. Though each patient shows minor variations in the degree and sequence of these and other signs, the general pattern of cerebellar disease progression is remarkably consistent. In addition, as the disease progresses pyramidal, extrapyramidal, cortical and brain stem signs of motor dysfunction may appear transiently, and will be detected by regular clinical follow-up. There are no sensory deficits. If death does not occur from intercurrent infection it follows when the pathological process spreads to the vital centres in the medulla.

The epidemiological study of kuru began in 1957 with the description of the disease and the mapping of the boundaries of the kuru region by Gajdusek, Zigas and Baker. Their reports[1,6] were followed by studies carried out by, among others, Alpers, Gajdusek, Hornabrook and Moir.[7–11] The epidemiological surveillance of kuru has been continuous since 1957, with the involvement of many investigators. During the period 1987 to 1995 surveillance was carried out solely by the Kuru Surveillance Team of the Papua New Guinea Institute of Medical Research (PNGIMR). During 1996 the field team was augmented by a collaboration with the MRC Prion Unit in London.

Throughout this time, field surveillance was rigorously maintained and the field records kept at the PNGIMR in Goroka. This brief report presents the data from the period 1987–1995 for the first time and examines the work of the Kuru Surveillance Team.

Methodology

The Kuru Surveillance Team comprised experienced field staff who had worked with MPA for many years as well as occasional staff and a member of the team in training. The villages of the whole of the kuru region and the groups bordering it were divided into areas of individual responsibility. In fact the senior staff ranged more widely in carrying out their work and, while doing so, would seek information in communities outside their own working areas, which provided independent checks on the data. Each staff member was well known in their area and had friends and contacts in the communities where they worked.

These field staff were experienced in field work and knowledgeable about kuru. They had seen many kuru patients in their youth, they had taken part in the examination of kuru patients many times and they had been trained to recognise the salient features of kuru. They each kept a diary of their work. On a regular basis they reported on the places they had visited and the patients they had seen; they also provided the information they had gathered from informants about known patients, rumoured patients, rejected patients and reports of new patients. They knew of the importance of establishing whether a case was real or not, and of getting accurate dates of onset and death. Their diaries and reports were reviewed in discussion with MPA as supervisor, who recorded the villages visited where there was no living case, all information about kuru patients (real and rumoured) and information about other diseases and local events. Whenever possible, patients were seen and examined by the supervisor either in the field or in Goroka.

As a measure of the comprehensive nature of this surveillance the reports for a typical year, 1991 (the middle year of the period studied), were tallied. Reports on other diseases and local events were not counted. In the twelve-month period there were 162 reports on kuru patients and 186 on ‘recovered’ or rumoured patients. For 575 visits to villages there proved to be no living case, or rumoured case.

As in all previous epidemiological studies, kuru was defined as a fatal disease with a progressive ataxic course lasting a period of some months diagnosed as kuru by the local people. During this period of surveillance cases were also assessed by the experienced members of the field team and many cases were examined by the team supervisor.

Kuru is the Fore name for the disease, which has been so common and widespread throughout the kuru region that its symptomatology is well known. A fatal outcome is essential to the diagnosis. No case of kuru diagnosed by objective signs of astasia and ataxia has ever been found to recover and all clinically documented cases have progressed to a fatal termination. Local beliefs allow people to ‘recover’ from presumed early kuru when their unsteadiness (from whatever cause) improves. If such cases are examined during the clinical phase of illness no objective evidence of kuru is found. Other rumoured cases proved to be false, or to be cases of malaria (which occurs more commonly now in the highlands) or other debilitating fevers. Sorting out these ‘cases’—including ‘recoveries’, cases reassigned to another diagnosis, or rejected cases—proves today to be a major part of surveillance activities. In the early days of kuru surveillance the ratio of real cases to spurious ones was about 5 to 1. During the period 1987 to 1995 the ratio was reversed and today the ratio of spurious cases to real cases is about 10 to 1.

Cases of kuru were mapped by their village of residence and related to the axis of social change, which was defined as the direction along which social change progressed as traditional cultural practices and behaviour were modified, directly or indirectly, by the gradual expansion of government control into the region.

The kuru studies of the PNGIMR, including the epidemiological surveillance of the disease, have been approved on scientific and ethical grounds by the Medical Research Advisory Committee of Papua New Guinea.

Results

Kuru is unusual, perhaps unique for a disease causing a major epidemic, in that the epidemiology includes all cases that exist of the disease. The results therefore describe the full data on the disease with no population sampling entailed.

The results are presented on all patients who died of kuru in each year of the 9-year period 1987 to 1995.

Table 1 shows the number of deaths from kuru, by age at death and sex, for each year, 1987–1995. The last patient to die in their 20s died in 1987. The last patient to die in their 30s died in 1991. This follows the pattern established earlier[7,11,12] of progressive deletion of age groups from the kuru mortality record, beginning with the youngest, due to the cohort effect of no transmission of kuru to those born after 1959. This was one of the major clues which established transumption of dead relatives at mortuary feasts (endocannibalism) as the mode of transmission[12,13] of the infectious agent of kuru.[14] It required the results of the transmission studies, the epidemiology and behavioural studies[15,16] to make full sense of kuru and its transmission.

Males were significantly exposed only as young boys, whereas females were exposed throughout life. There is therefore good reason to look at the epidemiological patterns of the sexes separately. Table 2 shows the mean age at death for males and females from 1987 to 1995. As one might expect, the mean age at death increased with time. Males died at a slightly younger age than females.

For both males and females the minimum incubation period can be calculated from 1959, when transmission ceased, to the year of onset (usually a year before death). The incubation period for cases who developed kuru in 1995 is therefore 35 years; and over 40 years for those patients who have sub-

Table 2. Mean age at death from kuru, 1987 to 1995, by sex and year

Year of death	Mean age at death (years)		
	Males	Females	All cases
1987	39.5	48.4	45.4
1988	39.0	48.3	47.1
1989	49.0	46.0	47.4
1990	46.0	48.9	48.2
1991	–	42.3	42.3
1992	48.5	50.8	50.1
1993	–	51.3	51.3
1994	56.0	56.3	56.2
1995	52.0	58.2	56.4
Total	46.5	49.9	49.0

Table 1. Deaths from kuru, 1987 to 1995, by age and sex

Year	Age at death (years)																	
	20–29			30–39			40–49			50–59			60–69			All ages		
	M	F	T	M	F	T	M	F	T	M	F	T	M	F	T	M	F	T
1987	1	–	1	2	3	5	–	1	1	1	4	5	–	–	–	4	8	12
1988	–	–	–	1	2	3	–	1	1	–	3	3	–	1	1	1	7	8
1989	–	–	–	1	2	3	1	3	4	2	1	3	1	–	1	5	6	11
1990	–	–	–	–	1	1	1	4	5	1	2	3	–	–	–	2	7	9
1991	–	–	–	–	1	1	–	2	2	–	–	–	–	–	–	–	3	3
1992	–	–	–	–	–	–	1	2	3	1	3	4	–	–	–	2	5	7
1993	–	–	–	–	–	–	–	1	1	–	3	3	–	–	–	–	4	4
1994	–	–	–	–	–	–	–	1	1	1	2	3	–	1	1	1	4	5
1995	–	–	–	–	–	–	1	–	1	1	3	4	–	2	2	2	5	7
Total	1	–	1	4	9	13	4	15	19	7	21	28	1	4	5	17	49	66

M Males.
F Females.
T Total.

sequently developed kuru after 2000. In addition, for males, one can estimate their likely actual incubation period (though this can never be proved) as age at onset less 6–8 years (the age at which they left their mother to join their father in the men's house and thus significantly reduced their exposure), which gives a figure of more than 50 years for a male dying in his late 50s or 60s. For women it is not possible to make such an estimate.

The overall female:male ratio of kuru cases in the 1987–1995 period was 2.9:1. This has not changed from the period 1972–1978, when it was 2.8:1, but is less than the 4.4:1 ratio in the first seven years of kuru investigation during the period 1957–1963.[11]

The year of birth of the patients is given in Table 3. The two patients who were born in 1958 were outliers. They were both born in remote communities. They were also outliers by age at death, since they died at the youngest ages recorded during this period (29 years in 1987 and 30 years in 1988); the next youngest age at death was 34 years. However, they are fully consistent with the full record of patients. Previously,[11] I listed the kuru patients in earlier years who were born since 1956—there were only seven: three in 1957, one in 1958 and three in 1959. It would have been unusual if those infected at the last mortuary feasts conducted surreptitiously in the area after 1954 had only short incubation periods, so these findings are not surprising.

While the date of death of all patients was obtained with certainty, the month of onset was more difficult to determine. Previous studies of duration of clinical course were either based on cases carefully followed from onset to death[17] or used subsets of the full dataset that were of greater reliability for this purpose.[8] When patients in this study with less certain dates of onset were excluded, a subset of 51 patients 'most reliable for duration studies' was obtained. The sex and age characteristics of the subset were similar to those of the full set. The duration of the clinical course (from onset to death) in months, by age and sex, in the patients of this subset is shown in Table 4. The overall mean duration was 13.3 months, with a range of 3 to 24 months. The previous study that was designed to address the question of age and duration[17] found an overall mean duration of 12.5 months, with a range of 3 to 23 months. That study, which included children as well as adults, found a positive relationship between age and duration. The present results have a more restricted age range but do show a slight increase in duration from those under 40 years (11.5 months) to those 40 years and over (13.9 months).

Table 3. Year of birth of kuru deaths, 1987 to 1995

Year of death		Year of birth				Total
		1920s	1930s	1940s	1950s	
1987	Male		1	1	2 (1951, 1958)	4
	Female	1	4	2	1 (1953)	8
1988	Male			1		1
	Female	1	3	1	2 (1950, 1958)	7
1989	Male	1	2	1	1 (1951)	5
	Female		1	3	2 (1953, 1953)	6
1990	Male		1		1 (1950)	2
	Female		2	4	1 (1951)	7
1991	Male					–
	Female			2	1 (1952)	3
1992	Male		1	1		2
	Female		3	1	1 (1951)	5
1993	Male					–
	Female		1	2	1 (1952)	4
1994	Male		1			1
	Female		2	2		4
1995	Male		1	1		2
	Female		4	1		5
Total		3	27	23	13	66

Table 4. Duration of clinical course in months, in patients dying in the period 1987 to 1995, by age and sex*

	Age at death (years)											
	20–39			40–49			50–69			Total		
	M	F	T	M	F	T	M	F	T	M	F	T
Number	5	7	12	4	10	14	5	20	25	14	37	51
Mean duration (months)	11.4	11.6	11.5	13.8	14.7	14.4	14.0	13.5	13.6	13.0	13.4	13.3
Range (months)	7–14	3–20	3–20	9–19	6–24	6–24	10–16	5–21	5–21	7–19	3–24	3–24

* Based on a subset of 51 patients 'most reliable for duration studies' obtained by excluding those with less certain dates of onset.

M Males.

F Females.

T Total.

The geographical location of patients in this period proved to be interesting. Table 5 sets out the geographical location by linguistic group. Of the 66 patients, 50 were from the South Fore (76%), 5 (8%) from the North Fore and 11 (17%) from other linguistic groups—Gimi (15%) and Keiagana (2%). In 1978 the percentages, for all cases of kuru diagnosed from 1957 onwards, were 62 per cent, 20 per cent and 19 per cent, respectively, for South Fore, North Fore and Other, with nine linguistic groups represented in the 'Other' category.[11] The striking changes in 1987–1995 therefore are the relative increase in cases in the South Fore and Gimi and the reduction in the proportion of cases in the North Fore and other linguistic groups (apart from the Gimi). This had been predicted since the South Fore and Gimi represent the more remote groups. They were the last to undergo the social changes associated with the transition from a traditional to a modern way of life brought about in this area by the Australian administration in the 1950s. However, the concentration of cases in the Gimi is confined to the first half of the period under study: of the 26 deaths from 1991 to 1995, only one is from the Gimi; the others include two from the North Fore and 23 from the South Fore, showing the striking dominance of the South Fore as the epidemic subsides.

Figure 1 shows all the villages with a history of kuru and the direction of the main axis of social change. A line has been drawn on the map in the centre of the kuru region perpendicular to the axis of change. In the first five years of kuru epidemiology (1957–1961) the larger proportion of cases were south of this line, when cases were plotted by their village of location

Table 5. Geographical location of kuru patients, 1987 to 1995, by linguistic group

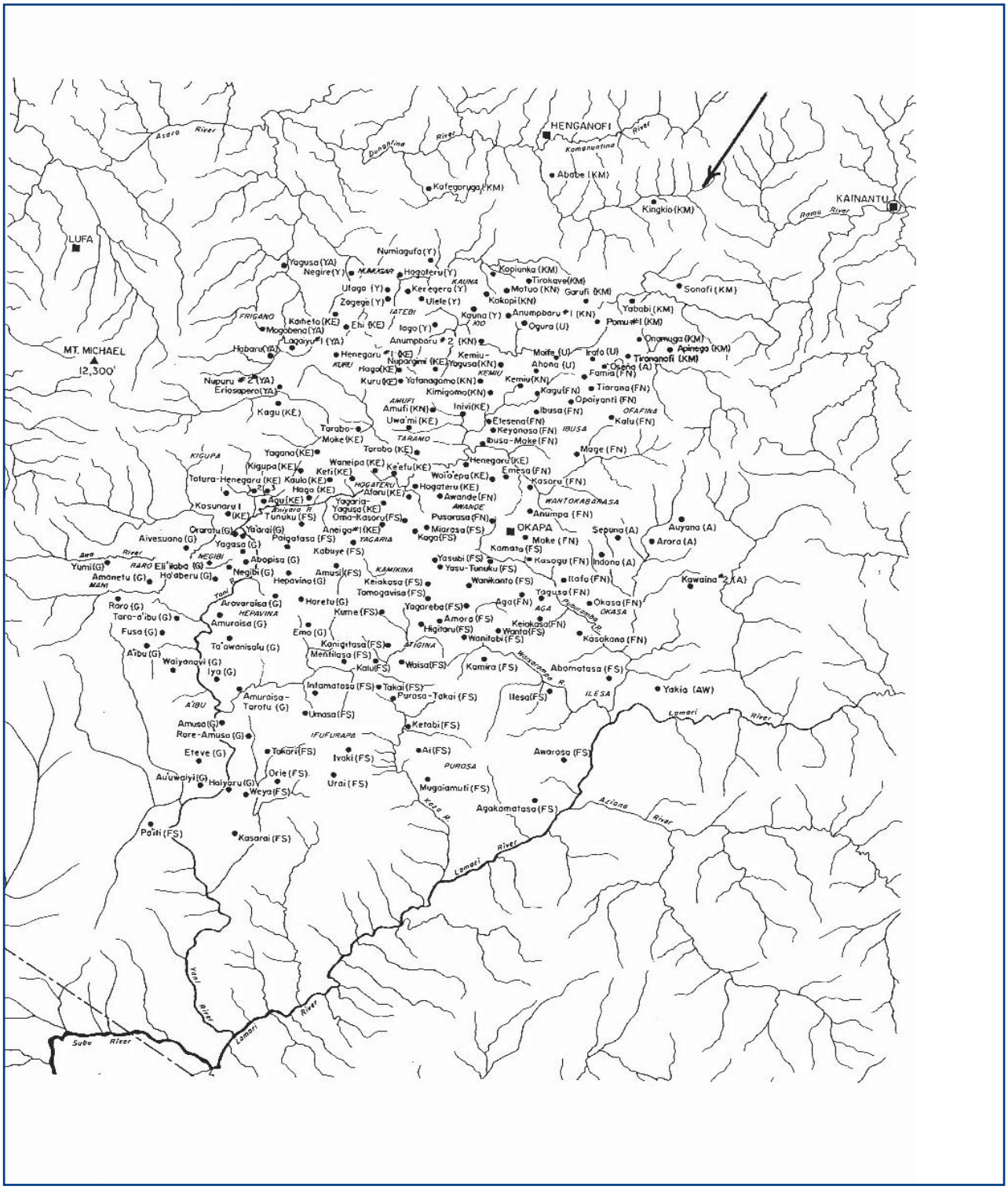
Year of death	South Fore			North Fore			Keiagana			Gimi			Total		
	M	F	T	M	F	T	M	F	T	M	F	T	M	F	T
1987	4	6	10	–	–	–	–	–	–	–	2	2	4	8	12
1988	1	4	5	–	1	1	–	1	1	–	1	1	1	7	8
1989	4	3	7	1	1	2	–	–	–	–	2	2	5	6	11
1990	1	4	5	–	–	–	–	–	–	1	3	4	2	7	9
1991	–	3	3	–	–	–	–	–	–	–	–	–	–	3	3
1992	2	5	7	–	–	–	–	–	–	–	–	–	2	5	7
1993	–	3	3	–	–	–	–	–	–	–	1	1	–	4	4
1994	1	3	4	–	1	1	–	–	–	–	–	–	1	4	5
1995	2	4	6	–	1	1	–	–	–	–	–	–	2	5	7
Total	15	35	50	1	4	5	–	1	1	1	9	10	17	49	66

M Males.

F Females.

T Total.

Figure 1. Location of all villages with a history of kuru



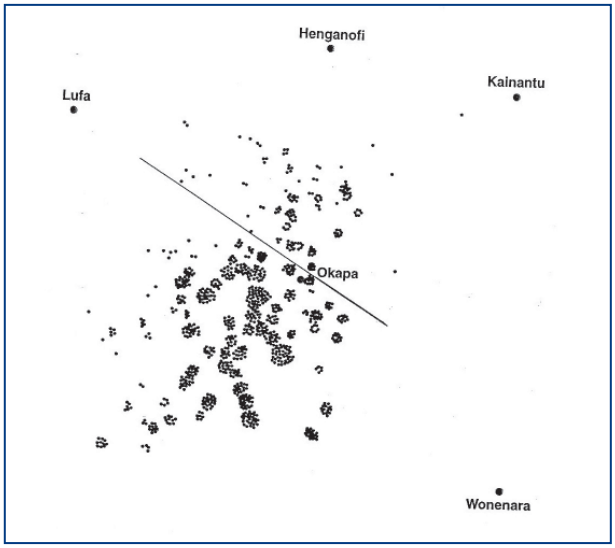
The arrow points in the direction of the axis of social change.

Figure adapted from Gajdusek, Zigas and Baker[6] and Alpers.[7]

Linguistic groups: A=Auyana, AW=Awa, FN=North Fore, FS=South Fore, G=Gimi, KE=Keiagana, KM=Kamano, KN=Kanite, U=Usurufa, Y=Yate, YA=Yagaria.

(Figure 2) although there were also many cases to the north of it. In the period 1977–1981 cases were sparser but there were still some cases in the north (Figure 3); however, for the period of the present study no case was found north of this line (Figure 4). Though empirically derived, this line has proven to be a useful indicator of social change in the area as it has affected the transmission of kuru.

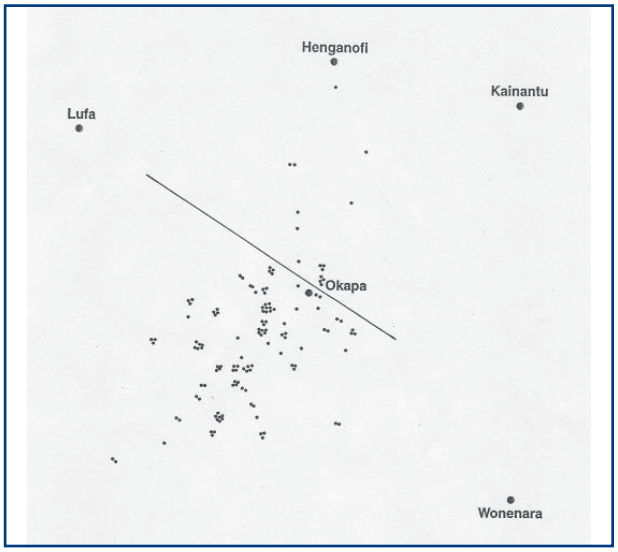
Figure 2. Location of all kuru deaths, 1957 to 1961



The line is drawn perpendicular to the axis of social change to divide the kuru region into north and south areas.

Figure adapted from Whitfield[18] and Alpers MP, unpublished seminar, 1998.

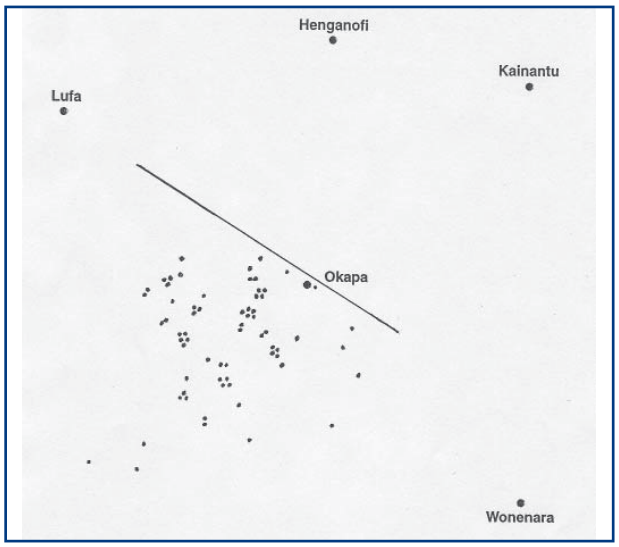
Figure 3. Location of all kuru deaths, 1977 to 1981



The line is drawn perpendicular to the axis of social change to divide the kuru region into north and south areas.

Figure adapted from Whitfield[18] and Alpers MP, unpublished seminar, 1998.

Figure 4. Location of all kuru deaths, 1987 to 1995



The line is drawn perpendicular to the axis of social change to divide the kuru region into north and south areas.

In an attempt to quantify this effect a score was obtained for each village in the kuru region according to its distance from a baseline drawn in the area between Henganofi and Kainantu (two towns on the Highlands Highway between Lae and Goroka and located in the figures). If the score were useful we might be able to show an increase in the mean score of the location of kuru patients with time. This proved not to be so, at least within this period of study. Though all patients in the period 1987–1995 were located south of the dividing line, there was no change in their overall distribution during the period. The high-scoring Gimi villages were in fact represented mostly in the first four years, and this difference between Gimi and South Fore would warrant further investigation.

Discussion

The clinical features of kuru have not changed with time and were the same in the 1987–1995 period as in previous years.[3] The duration of the clinical course in these cases of long incubation period was also similar to previous years,[17] when patients had a wide range of incubation periods.

The experimental transmission of kuru to the chimpanzee, with an incubation period of two years after intracerebral inoculation of kuru brain suspension, was reported in 1966.[14] This opened up a new branch of human medicine. Two years later, another fatal encephalopathy, Creutzfeldt-Jakob disease (CJD), was transmitted experimentally.[19] The infectious agent of these diseases, previously called a slow or unconventional virus, is now known to be a prion, which is infectious as a pure protein.

The pathology of kuru is confined to the central nervous system.[12] It shows the hallmarks of prion neuropathology: neuronal degeneration, characteristically with vacuolation, astrocytic hypertrophy and proliferation, spongiform appearance of the brain, arising from vacuolation in neuronal processes, accumulation of amyloid fibrils, often in the form of plaques, and no evidence of inflammatory encephalitis. The group of diseases with these hallmarks of prion neuropathology is now known as the transmissible spongiform encephalopathies (TSEs).

Sporadic CJD occurs at an annual rate of one per million in all human populations and in these cases the pathogenic and infectious form of the causative prion protein arises spontaneously. It is likely that the infectious prion in the first case of kuru arose in the same way.[5] Because of the mortuary practices, the agent did not disappear with the death of the patient but spread to others and thereby became the source of a slowly expanding epidemic. The original case may have been an ataxic form of CJD, which occasionally occurs, or the agent may have been modified to a 'kuru strain' by oral passage. Certainly since kuru has been investigated it has 'bred true', with a remarkable uniformity in its clinical progression and pathology.

The demography of the kuru region has been studied extensively in the past and some new work continues in addition to a government census each decade. The total population of the kuru region (all villages with a history of kuru) was 35,700 in 1958.[8] The annual growth rate from 1958 to 1963 for the South Fore was only 0.9 per cent; for the North Fore it was 2.9 per cent.[8] Since then growth rates have normalised across the region but the data are not available for detailed analysis.

Kuru, with its many interacting factors, has been reviewed[12,20] and more recently discussed in relation to another prion disease, variant Creutzfeldt-Jakob disease (first described in 1996).[21] A comprehensive review of the wide gamut of prion diseases, including scrapie in sheep, bovine spongiform encephalopathy, chronic wasting disease in deer and elk, and the human diseases, may be found in a newly updated book[22] and a clinical analysis of the human TSEs in a recent review.[23]

The epidemiology described here is work in progress and part of the ongoing description of an epidemic of prion disease caused by intraspecies recycling. The description will be completed when all the data have been extracted from the field records, entered and analysed. A number of important differences from the epidemiology of the disease when it was first described in the 1950s and 1960s, have been identified. The concentration of cases in the South Fore and Gimi and the complete absence of cases north of the central dividing line were significant changes. The effect of social change on transmission has been profound, and this change would already have affected the epidemiological patterns of kuru by 1957, when they were first recorded. This will be important to bear in mind in any detailed modelling of the epidemic.

The period of 1987–1995 shows a waning epidemic. The annual number of patients dying of kuru ranged between 3 and 12, in striking contrast to the first three studied years, when it was 203 (1957), 212 (1958) and 220 (1959). The first fall in mortality occurred in 1960, down to 184 for the year and dropping further to 170 in 1961.[11] Over the period 1987 to 1995 there was some fluctuation in the annual numbers of deaths, and a drop from 40 in the first four years to 23 in the last four years, but no striking trend in the annual numbers. Since then, there has been a marked decline. One patient died in early 1996 and from mid-1996 to the end of 2004, 11 patients died (J Collinge, *et al,* in preparation). In all, this is 12 deaths from kuru in the 9-year period 1996–2004 compared to 66 in the previous nine years. There was no death in 2002, one in 2003 and none in 2004. The epidemic of kuru is now clearly approaching its end.

The period from 1996 onwards is being written up separately in a manuscript in preparation by J Collinge, *et al.* The epidemiological data have yet to be extracted from the field notes for the period 1980 to 1986. When this has been done the results can be merged with the files already entered on computer and the data from all subsequent years can be added. This will enable the epidemiology of the whole epidemic to be analysed in detail and in various ways.

Acknowledgements

We acknowledge with gratitude the support of the Papua New Guinea Institute of Medical Research and the people of the kuru region, especially the patients suffering from kuru and their families, in the conduct of this study. Funding support for the analysis of the data was provided by a funding agreement between the Australian Government Department of Health and Ageing in Canberra and Curtin University of Technology. Additional support was provided by Curtin University and the MRC Prion Unit, London.

References

1. Zigas V, Gajdusek DC. Kuru: clinical, pathological and epidemiological study of a recently discovered acute progressive degenerative disease of the central nervous system reaching 'epidemic' proportions among natives of the Eastern Highlands of New Guinea. *P N G Med J* 1959;3:1–24.

2. Simpson DA, Lander H, Robson HN. Observations on kuru. II. Clinical features. *Australas Ann Med* 1959;8:8–15.

3. Alpers MP. Kuru: a clinical study. Mimeographed. Department of Medicine, University of Adelaide, 1964: p. 38.

4. Hornabrook RW. Kuru—a subacute cerebellar degeneration. The natural history and clinical features. *Brain* 1968;91:53–74.

5. Alpers MP, Rail L. Kuru and Creutzfeldt-Jakob disease: clinical and aetiological aspects. *Proc Aust Assoc Neurol* 1971;8:7–15.

6. Gajdusek DC, Zigas V, Baker J. Studies on kuru. III. Patterns of kuru incidence: demographic and geographic epidemiological analysis. *Am J Trop Med Hyg* 1961;10:599–627.

7. Alpers MP. Epidemiological changes in kuru, 1957 to 1963. In: Gajdusek DC, Gibbs CJ jr, Alpers M, eds. *Slow, Latent and Temperate Virus Infections*. NINDB Monograph No 2. Washington, DC: US Government Printing Office; 1965:65–82.

8. Alpers MP, Gajdusek DC. Changing patterns of kuru: epidemiological changes in the period of increasing contact of the Fore people with western civilization. *Am J Trop Med Hyg* 1965;14:852–879.

9. Hornabrook RW, Moir DJ. Kuru epidemiological trends. *Lancet* 1970;2:1175–1179.

10. Gajdusek DC. Unconventional viruses and the origin and disappearance of kuru. *Science* 1977;197:943-960.

11. Alpers MP. Epidemiology and ecology of kuru. In: Prusiner SB, Hadlow WJ, eds. *Slow Transmissible Diseases of the Nervous System, Vol 1: Clinical, Epidemiological, Genetic and Pathological Aspects of the Spongiform Encephalopathies*. New York: Academic Press; 1979:67–90.

12. Alpers MP. Kuru: implications of its transmissibility for the interpretation of its changing epidemiological pattern. In: Bailey OT, Smith DE, eds. *The Central Nervous System, Some Experimental Models of Neurological Diseases*. International Academy of Pathology Monograph No 9. Baltimore: Williams and Wilkins; 1968:234–251.

13. Mathews JD, Glasse R, Lindenbaum S. Kuru and cannibalism. *Lancet* 1968;2:449–452.

14. Gajdusek DC, Gibbs CJ jr, Alpers M. Experimental transmission of a kuru-like syndrome to chimpanzees. *Nature* 1966;209:794–796.

15. Glasse RM. Cannibalism in the kuru region of New Guinea. *Trans NY Acad Sci (Series 2)* 1967;29:748–754.

16. Lindenbaum S. *Kuru Sorcery: Disease and Danger in the New Guinea Highlands*. Palo Alto: Mayfield; 1979.

17. Alpers MP. Kuru: age and duration studies. Mimeographed. Department of Medicine, University of Adelaide, 1964: 12p.

18. Whitfield JT. Kuru. MA Thesis in Medical Anthropology, School of Oriental and African Studies, University of London, 2000.

19. Gibbs CJ jr, Gajdusek DC, Asher DM, Alpers MP, Beck E, Daniel PM, *et al.* Creutzfeldt-Jakob disease (spongiform encephalopathy): transmission to the chimpanzee. *Science* 1968;161:388–389.

20. Alpers MP. Kuru. In: Attenborough RD, Alpers MP, eds. *Human Biology in Papua New Guinea: The Small Cosmos*. Oxford: Clarendon Press; 1992: 313–334.

21. Collinge J. Variant Creutzfeldt-Jakob disease. *Lancet* 1999;354:317–323.

22. Prusiner SB, ed. *Prion Biology and Diseases*. Second edition. Cold Spring Harbor: Cold Spring Harbor Laboratory Press; 2004.

23. Collins SJ, Lawson VA, Masters CL. Transmissible spongiform encephalopathies. *Lancet* 2004; 363:51–61.

Q fever vaccine uptake in South Australian meat processors prior to the introduction of the National Q Fever Management Program

Adriana Milazzo,[1] Kathryn B Featherstone,[1] Robert G Hall[2]

Abstract

Despite the availability of a vaccine, the incidence of Q fever disease among populations at risk continues to be high. Q fever is an important cause of morbidity for workers, particularly in the meat and agricultural industries. Following an increase in 1998 in the number of Q fever notifications among meat processors to the Communicable Disease Control Branch, South Australia, a survey was conducted in the same year to assess the uptake of Q fever immunisation programs in meat processors and to identify barriers to offering these programs. This survey was conducted prior to the introduction of the National Q Fever Management Program in 2001 that provided a targeted vaccination program to specific at-risk occupations. The results of the survey highlighted that very few meat processors in South Australia offered a Q fever immunisation program to their workers. More importantly, this article highlights that there was a wide variety of attitudes and beliefs about Q fever disease and its prevention. These attitudes and beliefs have the potential to impact on whether workers at risk are offered or seek Q fever vaccination. Previous attitudes may return and levels of protection in at-risk occupations will decrease without a concerted effort at a state level. A replication of this study should benchmark the prevailing attitudes about Q fever programs. In response to the 1998 survey a number of strategies and initiatives were developed to address the barriers to Q fever vaccination in South Australian meat processors. The National Q Fever Management Program (2001–2005) further enhanced the ability to address barriers such as vaccine cost. *Commun Dis Intell* 2005;29:400–406.

Keywords: Q fever, vaccination, meat processors

Introduction

Q fever is a zoonotic disease caused by the obligate intracellular bacterium, *Coxiella burnetii*. It is an acute illness with symptoms of fever, fatigue, profuse sweats, rigors and marked loss of weight. Chronic Q fever may develop as a result of the persistence of *C. burnetii* in the body. Manifestations of this include endocarditis, hepatitis, osteomyelitis and post Q fever fatigue syndrome. The incubation period ranges from 14 to 60 days, but is usually about 20 days.[1,2] Cattle, sheep, kangaroos and goats are the main reservoirs for human infection in Australia, with transmission between animals occurring via infected ticks. Transmission of *C. burnetii* from animals to humans occurs most commonly via inhalation of infected dust or droplets.[2]

Q fever is an important cause of morbidity in meat process workers. Data from the National Notifiable Diseases Surveillance System (NNDSS) report the number of Q fever notifications between 1991 and 2003 for Australia ranged from 482 to 870 notifications. Although the numbers appear to be small it is the rate of disease in occupational groups, such as meat processors, and potential issues of turnover of workers in the industry that can impact adversely on the cost benefit of implementing Q fever vaccination programs. The epidemiology of Q fever in South Australia reveals that 54 per cent of notified cases between 1990 and 2003 were directly or indirectly linked to meat processors.

As Q fever is an occupational hazard in meat processors, employers are obliged to provide a safe work environment for employees (Section III, Occupational Health, Safety and Welfare Act, 1986, South Australia).

1. Communicable Disease Control Branch, Department of Health, South Australia
2. Public Health Group, Department of Human Services, Victoria

Corresponding author: Ms Adriana Milazzo, Communicable Disease Control Branch, Department of Health, PO Box 6 Rundle Mall, Adelaide South Australia 5000. Telephone: +61 8 8226 7177. Facsimile: +61 8 8226 7187.
Email: adriana.milazzo@health.sa.gov.au

Employers can meet their legal obligations by providing Q fever immunisation programs as recommended in the *Q fever Information Kit for the Australian Meat Industry*.[3] However, despite the availability of a vaccine since 1989 and due to the slow uptake of Q fever immunisation programs by industry, the incidence of Q fever disease among meat process workers continues to be high.

Vaccination requires two medical consultations seven days apart. Pre-vaccination testing requires a serum antibody estimation and a skin test to exclude people likely to have hypersensitivity reactions to the vaccine, or who have prior immunity. After vaccination, immunity takes two weeks to develop and appears to be long lasting. Ideally, Q fever vaccination should be offered to recruits three weeks prior to commencing work so that they are fully immune before entering the plant.

An increase in the number of Q fever notifications to the Communicable Disease Control Branch (CDCB), South Australia, was noted in May 1998. In South Australia rates were less than 0.6 per 100,000 in 1995, 1996 and 1997 but increased in 1998 to 1.68 per 100,000 population. In total, 14 of 25 (60%) cases notified in 1998 were linked to meat processors. The increase in notifications in 1998 was unexpected, particularly of workers in the meat industry. During the preceding three year period 20 cases of Q fever had been notified and less than 50 per cent reported working in a meat processor.

In Australia, few studies have been conducted to determine barriers to the uptake of Q fever immunisation programs in workplaces with this occupational hazard. Our study assessed the uptake of Q fever immunisation programs in South Australian meat processors and asked workplaces to identify barriers and factors supporting and encouraging the offering of Q fever immunisation programs.

These research questions are in line with recommendations by Bell, *et al.*[4] They recommended assessment of uptake of vaccine in meat processors, research into attitudes and barriers to vaccination at smaller meat processors and identification of barriers to program adoption.

Methods

Stakeholder working party

A stakeholder working party of representatives from peak industry and union bodies, the National Meat Association (South Australia branch), the Australian Meat Industry Employees Union and CDCB, was convened in 1998 to discuss the increase of Q fever notifications among workers employed in the meat industry. The stakeholder working party decided to assess the uptake of Q fever immunisation programs in workplaces and to identify barriers to offering these programs through a survey. The CDCB coordinated the survey.

The population

A list of accredited meat processors in South Australia in 1998 was obtained from the Meat Hygiene Unit, Department of Primary Industries. An accredited meat processor was defined as a facility processing meat for wholesale. Meat processors were included in the survey if their work involved slaughtering or boning animals that were known to transmit Q fever.

The target population comprised four categories of meat processors: domestic export (category 1); retail and country slaughterhouse (category 2); pet food (category 4) and game meat (kangaroo) (category 8). There were 70 meat processors in the four categories and 68 were eligible to participate in the survey. Of those 68 meat processors, 65 (96%) were interviewed by telephone over a three day period in September 1998. Participants were interviewed by telephone because they were geographically spread throughout South Australia. Verbal consent was obtained at the time of interview, by the CDCB interviewer, who asked to speak to the plant manager. Three could not be contacted. Respondents were usually managers and occupational health and safety officers in the larger meat processors and business owners in the smaller ones.

Data collection

A respondent from each meat processor was interviewed using a semi-structured questionnaire. There were two elements of research to this study: quantitative and qualitative. Quantitative data included information on work-site details of the number of workers employed full time, part time or casual and the time interval between a new worker being told they were employed to actual commencement of employment. Questions on pre-screening and availability of vaccination to workers and visitors were asked. Guidelines and strategies for planning a Q fever pre-screening and immunisation program are documented in the *Q Fever Information Kit for the Australian Meat Industry.* The kit was developed to ensure a uniform protocol across the meat industry for vaccination of new employees. Seven criteria were selected from the information kit as a means to measure the practice of Q fever immunisation programs in South Australia.

Respondents were also asked why they did not offer a Q fever immunisation program to employees if they did not have one in place, and were invited to make comments on Q fever prevention and immunisation in the South Australia meat industry. These

open-ended questions were used to collect qualitative data on knowledge, beliefs and attitudes about Q fever immunisation in the workplace.

The questionnaire was piloted to five meat processors. As a result of the pilot test, the questionnaire was modified and inconsistencies corrected.

Data were entered and analysed using Epi Info version 6.04. Qualitative data were coded into themes that emerged from responses. The similarities and differences between the four categories are discussed later.

Feedback of results

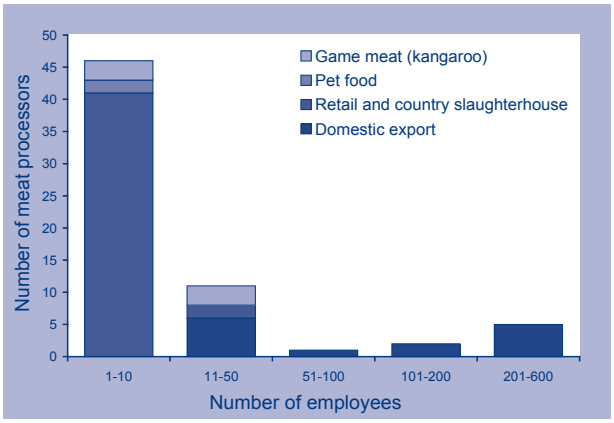
A summary of survey results was posted to all participating meat processors. The survey results were presented to the stakeholder working party and representatives reported to their respective industries.

Results

Demography of meat processors

The majority of meat processors, 43 of 65 (66%), belonged to category 2. However, category 1 meat processors employed the largest number of workers (range 18–570) (Figure). Sixty-six per cent of meat processors employed casual workers. Category 1 meat processors employed more casual workers than other meat processors. At the time of the survey 2,687 meat workers were employed in the industry. Of these, 2,353 (88%) of total number of employees were permanent workers and 334 workers were employed on a casual basis. The majority of meat processors in all categories stated that new employees commenced work within one week or less of being told they have employment.

Figure. Number of employees, South Australia, 1998, by category of meat processor



Q fever immunisation programs

Of 65 meat processors surveyed, 49 (75%) did not offer a Q fever immunisation program to employees. The majority of workers who did not have access to vaccination, worked in categories two, four and eight meat processors (Table 1).

Assessment of Q fever immunisation programs in meat processors

None of the 16 (25%) meat processors offering a Q fever immunisation program met the standard criteria in the industry information kit. Seven criteria in the industry information kit were used in this study to measure the timing and appropriateness of Q fever immunisation programs (Table 2).

Information about Q fever was provided to new employees by less than half of meat processors who had a vaccination program in place. Most of these processors provided information on commencement of employment and two provided it one week prior to commencement. Very little information about Q fever was provided to contractors or visitors to the

Table 1. Total number of employees offered Q fever immunisation program, South Australia, 1998, by category of meat processor

	Domestic export Category 1	Retail and country slaughterhouse Category 2	Pet food Category 4	Game meat (kangaroo) Category 8	Total
Number of processors with a vaccination program	10	4	0	2	16
Total number of processors per category	14	43	2	6	65
Number of staff with access to vaccination program by category*	1,870	39	0	18	1,927
Total number of staff per category*	2,393	191	5	98	2,687

* Based on survey responses.

Table 2. Criteria for Q fever vaccination programs and number of meat processors meeting each criterion, South Australia, 1998

Criteria for appropriate Q fever vaccination programs*	Total number of meat processors who implement the criterion	Comments
1. Plant informs new employees of Q fever and vaccine	19/65 (29%)†	11/19 (79%) domestic meatworks 5/19 (29%) country slaughterhouses
2. Plant informs contractors and visitors about risk of Q fever and availability of vaccine	6/65 (9%)†	5/6 domestic meatworks 1/6 game meat
Of those meat processors offering a Q fever vaccination program:		
3. New employees are screened and vaccinated prior to commencement of work	2/16 (13%)‡	Both processors vaccinated 1 month prior to commencement of work
if not 3, then 4. New employees are screened on 1st day of induction program	6/16 (38%)‡	6 processors provided a skin test within 2 weeks of commencing work
5. All employees participate in a pre-screening program	11/16 (69%)‡	5 processors vaccinated only high risk workers
6. Vaccination is given on 7th day following skin test	146/16 (88%)‡	1/16 administered within 5 days 1/16 administered after 3 weeks
7. Vaccination is performed by trained general practitioner or registered nurse	16/16 (100%)‡	

* Criteria used to measure appropriateness of Q fever vaccination programs was adapted from *Q Fever Information Kit for the Australian Meat Industry*, Meat Research Corporation, 1997.

† 65 total number of processors.

‡ 16 total number of processors with a vaccination program.

processor, while two processors invited contractors to participate in the company's immunisation program at their own expense.

Most meat processors offered pre-screening and vaccination to employees on commencement of employment and up to two weeks following commencement. Six companies offered skin testing but not in the recommended time periods. These respondents stated that skin testing was provided to new employees 'when possible', 'three times a year', 'in groups' and 'months later'.

All but one processor offered vaccination to all employees, the other offered it to permanent workers only. Skin testing and vaccination was performed mainly by general practitioners.

Barriers to implementation

Interviewers requested to speak to the plant manager for the purposes of the survey. As Q fever is an occupational hazard in meat processors, employers are obliged to provide a safe work environment for employees. While the interviewers did not clarify the specific responsibilities for occupational, health, safety and welfare (OHSW) of each respondent, respondents in larger meat processors tended to have higher levels of knowledge than respondents in smaller ones, even though their OHSW obligations in the workplace were the same.

The survey elicited a number of barriers to effective Q fever immunisation programs. Respondents from each category differed in their responses to questions about Q fever and occupational health and safety in the workplace. Three basic themes emerged.

Knowledge and attitudes

Respondents from category 1 were more knowledgeable about Q fever compared to other meat processors. Knowledge and experience of the impact of the disease on employees and their families also motivated some employers in categories 2 and eight to provide immunisation programs. The two respondents in category 4 knew little about Q fever. This category demonstrated the lowest level of awareness and knowledge of Q fever. Category 2 respondents were spread across a continuum of no knowledge to a high level of knowledge. Commensurate with the general lack of knowledge about Q fever, category 2 respondents were not sure about what is a risk, and how to assess their risks of Q fever infection:

> *'It's got to do with volume. Only a risk if large volumes of 1,000 sheep a day'.*

'… only cattle from up north are infected'.

Workers from all four categories and who had 25–30 years working experience in the industry, reported to have been told by industry trainers and others that they were not at risk.

'I was told that because I have been around goats and roos (kangaroos) etc for a long time that I have immunity to it'.

Cost

It was common for those respondents who were aware of Q fever associated with the meat industry to say that the costs of immunisation programs were a barrier, and often the reason programs were not implemented. Even category 1 respondents, who worked in meat processors with Q fever immunisation programs, spoke about cost saving practices used in the workplace, such as having two to three vaccination catch-up sessions a year.

Two respondents framed the burden of cost in the early loss of staff. Vaccinated workers who stayed for only a few days or weeks were more expensive to replace than unvaccinated workers. Employers may be reluctant to invest in high turnover workers, but willing to do so with workers who committed significant time with employers. Some respondents told of practices that they employed to avoid paying for the vaccination of employees who worked for less than two weeks.

'I wait for two weeks to see if they hang around before vaccination. If they don't, then I don't do it'.

Given that many respondents commented on the prohibitive cost of Q fever immunisation programs, it is not unexpected that they wanted the cost of vaccination to be reduced or subsidised. Cost reduction was seen as a role of any or all of the employer, employee, vaccine manufacturing company and the government. Others wished to contain cumulative costs that rise with each employee who leaves after a short work span through a system whereby employees who leave within, say two weeks, reimburse the employer for the cost of vaccination.

Provision of services

General practitioners were expected to be knowledgeable about Q fever disease and actively advise on immunisation for meat processors and their workers. They were seen as important health resources. General practitioners were relied upon to provide recommendations and Q fever vaccinations, and support advocates of immunisation programs in the workplace. In some incidences, they did not fulfil this expectation.

'When I go to the doctor, he just mentions keeping tetanus up to date'.

Likewise, respondents who sought medical advice about the appropriateness of Q fever vaccinations in their workplace told the interviewers that general practitioners generally dismissed their query. If vaccination was not actively supported by the general practitioner, respondents neglected the issue.

Respondents complained about lack of skilled general practitioners for Q fever vaccination.

'Especially rural general practitioners. They should learn how to do it (Q fever vaccination)'.

Trained Q fever immunisation providers were located sparsely around the state. For vaccination services outside the worksite, the employee must travel on two occasions to the provider and this may involve significant travel. In addition, the employee is absent from the worksite for varying lengths of time.

Factors supporting and encouraging Q fever immunisation programs

Several factors were linked to the implementation of a program. Respondents in category 1 spoke about the cost of having employees off work due to injury. They recognised the occupationally acquired nature of Q fever disease and subsequent work days lost due to illness, which has a financial impact on the business. The cost of preventing Q fever disease in employees was seen as a sound financial investment. Some respondents in all categories except category 4 were adamant that the benefits of Q fever vaccination in the meat industry outweighed the costs. The impact of Q fever on WorkCover levies is an incentive for meat processors to maintain vaccination levels in their employees, particularly for category 1 employers.

Suggestions offered by respondents to reduce the cost of vaccination in the workplace included delivering immunisation programs in a flexible way, such as utilising local registered nurses and ensuring local rural general practitioners were trained in Q fever immunisation.

Respondents were keen that information about Q fever prevention and immunisation be provided to them. They were clear that information about Q fever included risks, description of the disease, vaccination costs and employer responsibilities. The information must be presented in a way they could understand. One respondent suggested a personal visit.

'What should happen is not a report, but get a people-person to ring up, make an appointment and come here and talk to us in our country language… Someone we can relate to'.

Some respondents said that information about Q fever should not be limited to meat processors and their workers, but shared with the rural community, people who work with or are exposed to animals. One meat processor attempted to provide an incentive through sharing vaccination costs with its rural community.

'We advertised in the local paper for people in the community to participate (in the vaccination program). Only one did'.

The slaughter-floor and lairage were considered by industry to be high-risk work areas and some meat processors have policies that restricted workers and visitors without Q fever vaccination from these areas. A program that issues proof of Q fever vaccination via a personal zoonosis record card to employees enables potential employers to ascertain risk and work allocation in the meat processor, was suggested.

Respondents identified a general lack of knowledge about Q fever in the industry and rural community, from knowledge about the disease, its prevention and control, to technical implementation and legal obligations of immunisation programs in the workplace. Strategies offered included compiling a list of general practitioners knowledgeable about zoonotic disease and informing people at risk of Q fever about the disease, the risks, cost of vaccination and responsibilities of employers. Education settings were highlighted as legitimate providers of information on Q fever, both for preparing young people before entering the workplace and for participants in courses for the meat industry.

Discussion

Category 1 meat processors employed the largest number of workers (n=2,393) and of the 14 processors, 10 (71%) offered employees a Q fever immunisation program. In comparison, categories 2, 4 and 8 employed a total of 294 workers and of these 51 processors, 6 (12%) had an immunisation program in place. Differences in the values and attitudes about Q fever immunisation programs appeared to relate to the size and function of the category.

No meat processor fulfilled all seven criteria for a standard Q fever immunisation program. Reasons given by respondents for not doing so, ranged from a lack of knowledge of the risk of disease, vaccine cost and inaccessibility to trained immunisation providers. Similar barriers to vaccination have previously been reported.[4]

Respondents provided the study team with an understanding of the barriers to implementing Q fever immunisation programs in meat processors and possible strategies to encourage the uptake of Q fever immunisation programs. Once the topic of Q fever was raised with meat processors, there was an immediate demand for information about prevention and immunisation for the industry and others in the community at risk.

Subsequent to the survey, the stakeholder working party acted to increase the Q fever immunisation uptake in South Australia meat processors between 1998–2000. The stakeholder working party implemented strategies given the jurisdiction of the organisations represented on the group, that is, immunisation provision, prevention and information dissemination. Alliances with a number of organisations with a vested interest in Q fever prevention were established. Service provision gaps were mapped and identified and addressed through training of immunisation service providers. Access to services was formalised through a register of trained service providers for meat processors and was regularly maintained. Awareness raising of Q fever disease issues to at-risk, influential and other health and industry groups took place using a variety of methods and resources.

In October 2000 the Federal Minister for Health announced a National Q Fever Management Program. The program commenced in 2001 and provided free skin test and vaccination for targeted at-risk groups. The purpose of the program was to reduce the burden of Q fever disease in Australia. Meat process workers and visitors and contractors to meat processors were identified as targeted groups of the national Q Fever Vaccination Program. Funding provision for this group allowed for free skin test, vaccine and service delivery money for two doctor's visits and serology test.

The two years prior to the National Q Fever Management Program enabled South Australia to implement initiatives that provided the South Australia component of the national program with a strong basis to immediately provide funded service delivery, the major barrier the state could not address alone.

The Commonwealth Government funding for the national program ceased in South Australia in 2005. In South Australia the provision and responsibility of Q fever vaccination programs for employees reverted to meat processors' managers.

States are better situated to monitor gaps in service provision, use flexibility in service delivery such as using local registered nurses, liaise with trainers of students in accredited meat industry courses through their state systems, promote influential peers in reach-

ing specific at-risk groups, provide financial advice to smaller employers regarding the relationship of WorkCover levies and maintaining vaccination levels in employees and target all information in a way that meets the target groups' interests and needs.

The National Q Fever Management Program was funded to improve vaccine uptake, particularly for workers in rural and regional Australia; it has been a time-limited program that does not ensure sustainability and maintenance of high vaccine coverage in individuals or occupations at risk. A concerted effort at national and state level is required to address the issues of this occupationally-related disease.

Acknowledgements

The authors thank the following people for their participation and contribution to the project: Paul Sandercock, National Meat Association (South Australia branch) and Graham Smith, Australian Meat Industries Employment Union (South Australia branch). We are also grateful to the meat processors for participating in this survey.

References

1. Marrie TJ. Q fever. In: Marrie TJ, ed. *Q fever Vol 1. The Disease.* CRC Press Boca Raton, Florida; 1990.

2. Marmion B. *Q fever: Your Questions Answered.* CSL Limited. MediMedia Communications, New South Wales; 1999.

3. Meat Research Corporation. Q fever. Information Kit for the Australian Meat Industry. Australian Meat and Livestock Corporation, Sydney; 1997.

4. Bell M, Patel M, Sheridan J. Q fever vaccination in Queensland abattoirs. *Commun Dis Intell* 1997;21:29-31.

Communicable and vaccine-preventable conditions under surveillance by the APSU: 2004 update

Yvonne Zurynski,[1,2] Paula Cronin,[1] Elizabeth J Elliott[1,2,3]

Background

The Australian Paediatric Surveillance Unit (APSU) conducts national active surveillance of rare diseases of childhood, including infectious and vaccine preventable diseases, genetic disorders, childhood injuries and mental health conditions. The study of communicable and vaccine-preventable diseases is supported by the Australian Government Department of Health and Ageing through its communicable diseases program. This report is a summary of surveillance results for communicable and vaccine preventable diseases studied through the APSU in 2004.

In 2004, seven communicable or vaccine preventable conditions were studied:

- acute flaccid paralysis;*
- congenital cytomegalovirus infection;
- congenital rubella infection;
- perinatal exposure to HIV and HIV infection;
- neonatal herpes simplex virus infection;
- hepatitis C virus infection; and
- non-tuberculous mycobacterium infection.

* Although the aim of this surveillance is to identify acute flaccid paralysis due to poliomyelitis or associated with polio vaccination, there are many non-infectious causes of acute flaccid paralysis.

Methods

APSU study protocols are developed with collaborating investigators and/or institutions and the objectives and chief investigators for each study are listed in Table 1. Conditions under surveillance are listed on a report card sent monthly to practising paediatricians and other selected child health specialists (APSU contributors). Over a half of the contributors report via email. The system is efficient and economical, enabling surveillance of up to 16 different conditions simultaneously. Contributors respond whether or not they have a case to report. This enables calculation of monthly response rates and identification of non-responders. Each week the APSU forwards positive reports to study investigators who collect de-identified clinical and/or laboratory data from reporting clinicians by questionnaire. A unique identification code for each case enables identification of duplicate reports. Information reported in the questionnaire is used to ensure that case criteria are met.[1,2]

The APSU aims to provide epidemiological information that is representative of the Australian population and maximal case ascertainment is a high priority. Despite a representative mailing list and high response rates, complete case ascertainment is unlikely. This is particularly relevant in remote communities where children have limited access to paediatricians. However, for most conditions studied by the APSU no national data are available to estimate completeness of ascertainment. APSU encourages the use of complementary data sources where available and reporting by a range of specialists to maximize cases identified. Reported rates for conditions ascertained through the APSU therefore represent a minimum estimate of these conditions in the relevant Australian populations.

1. Australian Paediatric Surveillance Unit, The Children's Hospital at Westmead, Westmead, New South Wales
2. Discipline of Paediatrics and Child Health, Faculty of Medicine, The University of Sydney, Sydney, New South Wales
3. The Children's Hospital at Westmead, Westmead, New South Wales

Corresponding author: Dr Yvonne Zurynski, Assistant Director, Australian Paediatric Surveillance Unit, The Children's Hospital at Westmead, Locked Bag 4001, Westmead NSW 2145. Telephone: +61 2 9845 1202 (or ext 3005). Email: apsu@chw.edu.au

Table 1. Summary of findings

Condition and principal investigator	Objectives	Key findings
Acute flaccid paralysis (AFP) A/Prof. Heath Kelly, Victorian Infectious Diseases Reference Laboratory	To determine the notification rate of AFP in children aged <15 years; To determine whether AFP is caused by poliovirus infection and if so, whether it is a wild, vaccine, or vaccine-derived strain of poliovirus; To determine other causes and the clinical picture of AFP in Australia.	In 2004, Australia exceeded the WHO AFP surveillance target of 1 case per 10^5 persons aged <15 years, per annum. The majority (approx. 70%) of AFP cases are due to Guillain-Barré syndrome or transverse myelitis. All reported cases were classified as non-polio AFP.[3] 40% of cases had 2 faecal specimens collected within 14 days of onset of paralysis, below the 80% target level identified by WHO. Continued surveillance is required to keep Australia polio free, especially in view of recent reports of imported cases of wild poliovirus into Indonesia.
Congenital cytomegalovirus (cCMV) infection A/Prof. William Rawlinson, Virology Division, Department of Microbiology, Prince of Wales Hospital, Sydney	The study aims to determine: The incidence of congenital and suspected congenital CMV infection; The presenting features and clinical spectrum of disease due to congenital CMV; The genotypes of CMV which cause congenital disease; Current therapy for congenital CMV infection; and The epidemiology of congenital CMV prior to trials of vaccines and antivirals.	This is the first national study of cCMV in Australia, a major infectious cause of malformations. cCMV infection was not associated with maternal illness in 8/17 cases in 2004 and should be considered regardless of maternal history. cCMV remains under-diagnosed. Although most cases are diagnosed by urine culture, use of polymerase chain reaction for urinary screening for CMV may increase diagnostic yield.[4] Universal neonatal hearing screening programs may also help identify new cases.
Congenital rubella Dr Cheryl Jones, The Children's Hospital at Westmead, and Discipline of Paediatrics and Child Health, University of Sydney	To document the incidence of congenital rubella infection; To determine the vaccination status of mothers of infected infants; To monitor the effectiveness of the current vaccination program.	The only reported case in 2004 was born to an unvaccinated woman born overseas. We have previously documented this group as 'at risk.'[5] Women born in countries with poorly developed vaccination programs should have serological testing for rubella after arrival in Australia, and vaccination when appropriate. Travel to rubella endemic counties in the first trimester by women with no prior rubella immunity poses a risk to the foetus of congenital rubella.
Perinatal exposure to HIV and HIV infection Ann McDonald, National Centre in HIV Epidemiology and Clinical Research	To identify new cases of perinatal exposure to HIV, paediatric HIV infection, and AIDS; To describe the pattern of perinatal exposure to HIV in Australia; To monitor the perinatal HIV infection transmission rate and use of interventions for reducing the risk of mother-to-child transmission; To describe the natural history of paediatric HIV infection.	No new cases of HIV infection were identified in children in 2004. All cases reported in 2004 were of perinatal exposure to HIV. Consistent with our previous data 55% of these mothers were exposed to HIV through heterosexual contact in a high HIV prevalence country or in Australia with a partner from a high prevalence country and 32% used IV drugs or had a partner who used IV drug.[6] Supporting previously reported trends,[6] the proportion of children with perinatal HIV exposure who become infected, declined from 41.2% (children born 1995–1996) to 2.4% (children born 2003–2004) due to increasing use of interventions in women diagnosed antenatally.

Table 1. Summary of findings, *continued*

Condition and principal investigator	Objectives	Key findings
Neonatal herpes simplex virus infection (HSV) Dr Cheryl Jones, Herpes Virus Research Unit, The Children's Hospital at Westmead, and Discipline of Paediatrics and Child Health, University of Sydney	To determine the incidence of neonatal HSV infection in Australia, its mortality and morbidity; To determine its mode of presentation e.g. localised, disseminated or complicated by encephalitis or pneumonitis and mode of transmission; To determine whether there is delay between presentation, diagnosis and initiation of treatment.	Over a half of neonatal HSV infections in Australia are caused by HSV type 1, in contrast to the USA where HSV type 2 predominates. Typical herpetic lesions of the skin, eye or mouth were not evident in half of infants identified with neonatal HSV infection, which makes early diagnosis difficult. Disseminated HSV infection in the newborn may be associated with the early onset of pneumonitis in infants (in whom the chest X-ray may be normal). This is highly lethal unless antiviral therapy is initiated.
Hepatitis C virus infection (HCV) Dr Cheryl Jones, The Children's Hospital at Westmead, and Discipline of Paediatrics and Child Health, University of Sydney	To determine the reported incidence of newly diagnosed HCV infection in Australian children; To describe the clinical presentation, investigation and management of newly diagnosed HCV infection in Australian children; To document the presence of known risk factors for HCV infection in an Australian paediatric population; To determine the prevalence of co-infection with hepatitis B virus (HBV) and/or human immunodeficiency virus (HIV) in Australian children with newly diagnosed HCV infection.	Most (>80%) HCV infection in Australian children is acquired perinatally. In our study infants at risk were born to mothers who used IV drugs (approx. 60%), had invasive procedures overseas or had tattoos.[7] Most HCV-infected children are clinically asymptomatic with mildly elevated liver function test at diagnosis. The reported number of infected children is lower than predicted using national laboratory notifications. This may be due to under-diagnosis and/or under-reporting.
Non-tuberculous mycobacterium infection (NTM) Dr Pamela Palasanthiran, Paediatric Infectious Diseases Specialist, Department of Immunology and Infectious Diseases, Sydney Children's Hospital Randwick, NSW	To estimate the incidence of newly diagnosed NTM infection in children seen by child health specialists in Australia; To describe the epidemiology and spectrum of disease and document known risk factors; To describe diagnostic investigations used in Australia; frequency of use of skin testing and the clinical utility of the test, including differential skin testing; To describe the management of NTM in Australia and the response to treatment.	In accordance with the literature, this infection usually presents in otherwise healthy children <5 years with lymphadenopathy. Surgery is required in the majority. *Mycobacterium avium intracellulare* and *Mycobacterium fortuitum* are the commonest organisms isolated. Children with underlying conditions experience relapse regardless of management.

Cases are classified according to the following criteria:

Valid: A *confirmed* case is one that satisfies the case definition criteria and a *probable* case is one that does not completely meet the case definition criteria but is highly probable on the basis of available information.

Invalid: A *duplicate* case is one that has already been reported and an *error* is a reported case that does not fulfil the case definition criteria; or for which the diagnosis was revised by the reporting clinician; or for which the APSU report card was ticked by mistake.

Results

In 2004, 1,112 clinicians participated in the monthly surveillance of 13 conditions, with an overall monthly response rate of 91 per cent. Questionnaire return rate is >80 per cent for most studies. Table 2 shows the number of cases reported in 2004 and for the whole study period and the reported rate per 100,000 population.[2]

APSU data contribute significantly to the national surveillance effort, providing valuable information for clinicians, policy makers and the community. The APSU is often the only source of national data that includes clinical and or laboratory details and data from both inpatients and outpatients. The chief investigator, objectives and key findings for studies are summarised in Table 1.

Further information on the above studies may be obtained by contacting the APSU: website www.apsu.org.au; phone (02) 9845 3005; email: apsu@chw.edu.au, or the Principal Investigator for each study.

Acknowledgements

The APSU is funded by the Australian Government Department of Health and Ageing; the Faculty of Medicine, the University of Sydney; and the Division of Paediatrics and Child Health, Royal Australasian College of Physicians.

We acknowledge the important continued contribution of all paediatricians and child health professionals who participate in surveillance studies conducted by the APSU.

References

1. Elliott E, Cronin P, Rose D, Zurynski Y, Eds. APSU Annual Report 2002–2003. Australian Paediatric Surveillance Unit, 2005; ISSN:1443–3524.

2. Elliott E, Cronin P, Zurynski Y. Eds. APSU Annual Report 2004. Australian Paediatric Surveillance Unit, 2005; (in press).

3. Australian Bureau of Statistics. *Australian Demographic Statistics*, 3101.0, March Quarter; 2005.

Table 2. Confirmed cases identified for 2004 and for the total study period

Condition	Date study commenced	Questionnaire response (%) for total study period	Number of confirmed cases for 2004	Number of confirmed cases for total study period	Reported rate (per 10^5 per annum)
Acute flaccid paralysis	March 1995	89	45*	338*	0.87§
Congenital cytomegalovirus	Jan 1999	66	17	48	3.85‖
Congenital rubella (with defects)	May 1993	96	1	50	0.11§
Perinatal exposure to HIV; HIV infection	May 1993	91	24† 0†	253 39	8.37‖ 1.29‖¶
Neonatal herpes simplex virus infection	Jan 1997	96	11	71	4.10‖
Hepatitis C virus infection	Jan 2003	84	12	24	0.30§
Non-tuberculous mycobacterial infection	July 2004	85	20‡	20	–

* All reported cases that have been classified by the Polio Expert Committee were 'non-polio AFP' according to WHO criteria.
† In 2004 all reported cases were perinatal exposures to HIV infection. No new HIV infections were reported.
‖ Based on number of births as estimated by the Australian Bureau of Statistics.[8]
¶ All HIV infections resulted from perinatally acquired HIV.
‡ Includes confirmed and probable cases. Due to the short surveillance period a rate is not reported.
§ Based on population of children aged ≤ 15 years as estimated by the Australian Bureau of Statistics.[8]

4. Morris AMS, Elliott EJ, D'Souza RM, Antony J, Kennett M, Longbottom H. Acute flaccid paralysis in Australian children. *J Paediatr Child Health* 2003; 9:22–26.

5. Trincado DE, Rawlinson WD. Congenital and perinatal infections with cytomegalovirus. *J Paediatr Child Health* 2001;37:187–192.

6. Forrest J, Donovan T, Burgess M. A resurgence of congenital rubella in Australia? *Commun Dis Intell* 2003;27:533–535.

7. McDonald AM, Li Y, Cruickshank MA, Elliott EJ, Kaldor JM, Ziegler JB. Use of interventions for reducing mother-to-child transmission of HIV in Australia. *MJA* 2001;174:449–52.

8. Elliott E, Jones CA, Mews C, Hardiker W, Kesson A, Polis S, Dore G, Kaldor J. Reported risk factors for hepatitis C virus infection in Australian children: results of national surveillance. *J Paediatr Child Health* 2005;41:S3.

A report from the Communicable Diseases Network Australia, 1 July to 30 September 2005

The Communicable Diseases Network Australia (CDNA) consists of communicable disease authorities from various Australian Government agencies and state and territory health authorities, in addition to expert bodies and individuals in the specific areas of communicable disease epidemiology, clinical management, disease control and laboratory diagnosis. The CDNA provides national public health leadership and co-ordination on communicable disease surveillance, prevention and control, and offers strategic advice to governments and other key bodies on public health actions to minimise the impact of communicable diseases in Australia and the region.

Refugee health

CDNA endorsed the *Recommendations for refugee pre-departure assessment/treatment for malaria* which were developed by the International Organisation for Migration based on advice provided by the Australasian Society for Infectious Diseases.

Salmonella control in chicken meat and eggs

CDNA agreed the control of *Salmonella* is an important public health issue, with control of *Salmonella* in eggs and chicken meat a possible means of controlling *Salmonella* in humans. CDNA requested that OzFoodNet collate and summarise available data on *Salmonella* prevalence in both eggs and chicken meat. The Network considered a presentation from OzFoodNet at a face-to-face meeting held in Melbourne and noted Food Standards Australia and New Zealand is currently developing a proposal for primary production processing standards, a risk assessment and a cost benefit analysis. CDNA agreed to initiate dialogue between industry representatives, state and territory health authorities, Animal Health Australia and the Department of Agriculture, Fisheries and Forestry.

Viral haemorrhagic fever

The *Laboratory precautions for samples collected from patients with a suspected viral haemorrhagic fever* has been revised and updated by the Public Health Laboratory Network and was endorsed by CDNA. This paper consists of two parts. Only Part A, *Guidelines for laboratories that are not associated with a designated isolation hospital* was amended. Part B remains unchanged.

Shigella notifications to the National Notifiable Diseases Surveillance System

In response to the increasing number of cases being reported, CDNA requested OzFoodNet provide information on *Shigella* notifications in Australia, in particular subtypes and Indigenous status of notified cases. At the face-to-face meeting held in Melbourne, CDNA considered a paper from OzFoodNet and noted some problems exist with the National Notifiable Diseases Surveillance System (NNDSS) data as states and territories are not coding in a consistent manner when entering notifications data in NNDSS.

Guidelines for the public health management of trachoma in Australia

At the September meeting in Melbourne CDNA endorsed the Guidelines, which had been developed by the CDNA Trachoma Steering Group, as a minimum standard. It was agreed antibiotic resistance surveillance is required, however a mechanism to measure resistance is yet to be determined.

Face-to-face meeting

In September the full CDNA met in Melbourne, along with the CDNA Jurisdictional Executive Group. Further details of the meetings will be reported to *CDI* as outcomes are achieved. In addition to the items previously mentioned, the major items discussed at these meetings were:

- review of the structure of CDNA and its sub committees and working groups;
- CDNA's 2005–06 workplan;
- a review of the meningococcal B vaccination programs in New Zealand;
- development of CDNA working procedures which will result in protocols for all aspects of the CDNA and its subcommittees (including appointment of the CDNA chair and making diseases nationally notifiable);
- nucleic acid detection tests for *Neisseria gonorrhoeae*;
- pandemic influenza planning;
- interpandemic influenza guidelines; and
- reports from the National Centre for Immunisation Research and Surveillance of Vaccine Preventable Diseases and the CDNA sub-committees and working groups.

In addition, representatives from the Department of Immigration and Multicultural and Indigenous Affairs travelled from Canberra to Melbourne to meet with the CDNA Jurisdictional Executive Group. Health policy, the DIMIA Humanitarian Program policy, humanitarian settlement and special health projects in relation to refugees being resettled in Australia were major discussion points.

Supplementary report: surveillance of adverse events following immunisation among children aged less than 7 years in Australia, 1 January to 30 June 2005

Glenda Lawrence,[1] Ian Boyd[2]

This report summarises national passive surveillance data contained in the Adverse Drug Reactions Advisory Committee (ADRAC) database at 30 September 2005 for adverse events following immunisation (AEFI) reported for children aged <7 years who received vaccines between 1 January and 30 June 2005.[1–3] The average annual population-based AEFI reporting rates were calculated using mid-2004 population estimates. Reporting rates per 100,000 doses of vaccine were calculated for eight vaccines that are funded by the National Immunisation Program (NIP) using denominator data from the Australian Childhood Immunisation Register (ACIR). The report includes data for adverse events following receipt of the seven-valent pneumococcal conjugate vaccine (7vPCV), which has been funded under the NIP from 1 January 2005 for all infants at two, four and six months of age, with a catch-up program for children born from 1 January 2003.[4] AEFI reporting rates were not estimated for some vaccines due to lack of reliable denominator data.

The data reported here are provisional only. It is important to note that an AEFI is defined as a medically important event that is temporally associated with immunisation but not necessarily causally associated with immunisation. Readers are referred to previous reports for a description of the national AEFI passive surveillance system,[1] methods used to analyse the data[1–3] and information regarding limitations and interpretation of the data.[2] Often, more than one vaccine is listed as suspected of involvement in the reported adverse event, so the number of vaccines listed will be greater than the number of AEFI records analysed.

1 January to 30 June 2005

There were a total of 254 AEFI records (28.5 per 100,000 population) for children aged <7 years for vaccines administered in the first six months of 2005. This was a 16 per cent increase on the 219 records (24.5 per 100,000 population) for the corresponding six month period in 2004. Thirty-five per cent (n=88) of records were for children aged <1 year, 17 per cent (n=44) for children aged 1 to <2 years and 48 per cent (n=122) for children aged 2 to <7 years. The male to female ratio was 1.6:1.0.

Of the 254 records analysed, 15 (5.9%) had outcomes defined as 'serious' (i.e. recovery with sequelae, hospitalisation, life-threatening event or death), and was lower than previously reported (9%).[2] Serious or potentially life-threatening AEFIs reported included anaphylactic reaction (n=2), seizure (n=5), hypotonic-hyporesponsive episode (HHE) (n=5) and thrombocytopenia (n=3). No deaths were reported. The most common reaction categories were injection site reaction (n=138; 54%), allergic reaction (n=62; 24%) and fever (n=46; 18%).

One or more of the eight vaccines shown in the Table was recorded as suspected of involvement in the reported adverse event for 248 of the 254 records analysed. The six records that listed other suspected vaccines included Bacille Calmette-Guérin (n=1), varicella (n=3) and pneumococcal polysaccharide (n=2) vaccines.

The AEFI reporting rates per 100,000 vaccine doses recorded on the ACIR, both overall and for specific vaccines, were generally similar to those for 2004[2,3] and lower than the average reporting rate

1. National Centre for Immunisation Research and Surveillance of Vaccine Preventable Diseases, University of Sydney and The Children's Hospital at Westmead, Sydney, New South Wales
2. Adverse Drug Reactions Unit, Therapeutic Goods Administration, Canberra, Australian Capital Territory

Corresponding author: Dr Glenda Lawrence, National Centre for Immunisation Research and Surveillance of Vaccine Preventable Diseases, Locked Bag 4001, Westmead NSW 2145. Telephone:+61 2 9845 1433. Facsimile: +61 2 9845 1418.
Email: glendal@chw.edu.au

for vaccines received in the first six months for the four years 2001 to 2004 (Table). The reporting rate for AEFIs with outcomes defined as 'serious' for the eight vaccines decreased from 1.0 to 0.6 per 100,000 doses (Table).

The largest reductions in reporting rates of specific vaccines were for meningococcal C conjugate vaccine and measles-mumps-rubella vaccine (Table). There was an increase in the reporting rate in 2005, compared with 2004, for the combined *Haemophilus influenzae* type b-hepatitis B vaccine (Table) and diphtheria-tetanus-acellular pertussis vaccine for children aged <1 year (16.3 vs 8.0 per 100,000 doses). This may relate to reporting of AEFIs following 7vPCV during 2005 as the vaccines are usually given concurrently at two, four and six months of age.

AEFI reporting rates for January to June 2005 were lower among the 2 to <7 year age group and slightly higher for the <1 and 1 to <2 year age groups compared with January to June 2004 (Table). Much of the increase in reporting for the two younger age groups appears to be related to the commencement, on 1 January 2005, of the universal 7vPCV program for infants and the catch-up program for children born on or after 1 January 2003 (Figure 1).

Table. Reporting rates of adverse events following immunisation (AEFI) per 100,000 vaccine doses,* children aged <7 years, ADRAC database, January to June 2005

	AEFI records‡ (n)	Vaccine doses* (n)	Reporting rate per 100,000 doses§		Ratio of 2005 rate and 4-year mean‖
			Jan-June 2005	Jan-June 2004	
AEFI category†					
Total	248	2,407,362	10.3	12.2	0.6
'Certain' or 'probable' causality rating	114	2,407,362	4.7	4.5	0.6
'Serious' outcome	14	2,407,362	0.6	1.0	0.4
Vaccine					
Diphtheria-tetanus-pertussis	126	263,576	47.8	48.5	0.8
Diphtheria-tetanus-pertussis-hepatitis B	37	224,070	16.5	14.1	0.7
Haemophilus influenzae type b	41	226,782	18.1	20.1	0.6
Haemophilus influenzae type b-hepatitis B	20	129,600	15.4	6.0	1.5
Poliovirus (oral or inactivated)	56	486,445	11.5	10.4	0.9
Pneumococcal conjugate	104	666,299	15.6	–	–
Measles-mumps-rubella	59	254,198	23.2	33.6	0.8
Meningococcal C conjugate	28	156,392	17.9	29.1	0.5
Age group					
<1 year	87	1,395,474	6.2	4.9	0.8
1 to <2 years	40	541,852	7.4	6.7	0.3
2 to <7 years	212	470,036	25.7	32.3	0.9

* Number of vaccine doses recorded on the Australian Childhood Immunisation Register (ACIR) and administered between 1 January and 30 June 2005.

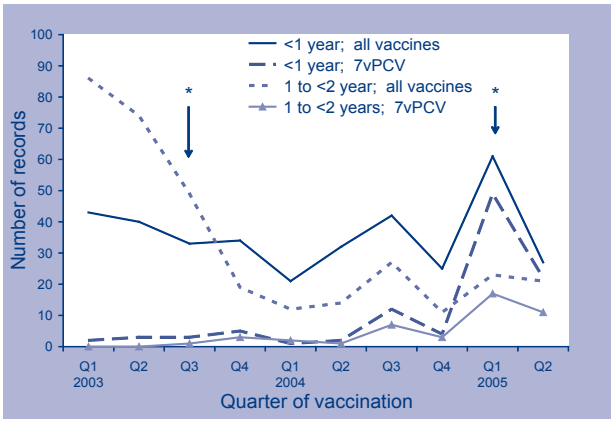
† Records where at least one of the eight vaccines shown in the table was suspected of involvement in the reported adverse event. AEFI category includes all records (i.e. total), those assigned 'certain' or 'probable' causality ratings, and those with outcomes defined as 'serious'. Causality ratings were assigned using the criteria described previously.[1,2] A 'serious' outcome is defined as recovery with sequelae, hospitalisation, life-threatening event or death.[1,2]

‡ Number of AEFI records in which the vaccine was coded as 'suspected' of involvement in the reported adverse event and the vaccination was administered between 1 January and 30 June 2005. More than one vaccine may be coded as 'suspected' if several were administered at the same time.

§ The estimated AEFI reporting rate per 100,000 vaccine doses recorded on the ACIR.

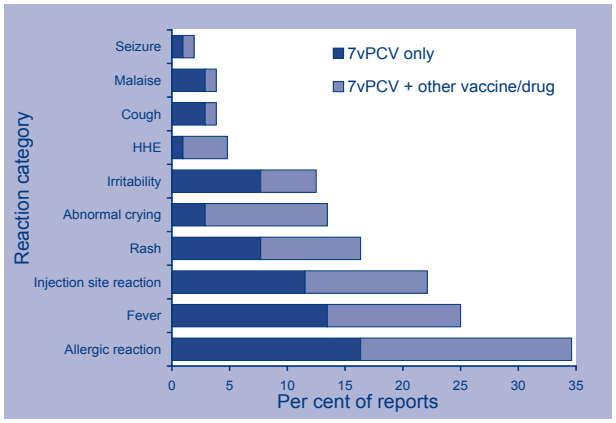
‖ Ratio of the reporting rate for January to June 2005 to the average (mean) reporting rate for January to June of the previous four years (2001–2004) (or 2003–2004 for meningococcal C conjugate vaccine).[2]

Figure 1. Reports of adverse events following immunisation, ADRAC database, 1 January 2003 to 30 June 2005, by age group and suspected vaccine



* Arrows indicate changes to national immunisation policy: (i) in September 2003, the 7-valent pneumococcal conjugate vaccine (7vPCV) was recommended for all children aged <2 years and the fourth dose of diphtheria-tetanus-acellular pertussis vaccine, due at 18 months of age, was removed from the immunisation schedule;[5] (ii) commencement of the nationally funded 7vPCV program for all children from 1 January 2005, with a catch-up program for children born from 1 January 2003.[4]

Figure 2. Frequently reported adverse events following receipt of seven-valent pneumococcal conjugate vaccine, ADRAC database, 1 January to 30 June 2005



HHE Hypotonic-hyporesponsive episode.

Pneumococcal conjugate vaccine

There were 104 reports where 7vPCV was listed as suspected of involvement in the reported AEFI and the vaccine was administered during January to June 2005. The AEFI reporting rate was 15.6 per 100,000 doses of 7vPCV recorded on the ACIR and was similar for the three age groups analysed (16.4, 13.9 and 15.6 per 100,000 doses for the <1 year, 1 to <2 years and 2 to <7 years age groups, respectively). Of the 104 records, 7vPCV was listed as the only suspected vaccine for 45 (43%) records while 10 (9.6%) records listed outcomes defined as 'serious'.

The most frequently reported adverse events following 7vPCV were allergic reaction (35%), fever (25%) and injection site reaction (22%) (Figure 2). More severe AEFIs where 7vPCV was the only suspected vaccine included HHE (n=1), seizure (n=1), severe allergic reaction (n=1) and thrombocytopenia (n=1). The United States of America passive AEFI surveillance system (Vaccine Adverse Events Reporting System) has also received reports of HHE and thrombocytopenia following 7vPCV where it was the only vaccine administered.[6] A causal relationship has not been established between 7vPCV and these AEFIs.

Conclusion

As seen previously, changes in the Australian Standard Vaccination Schedule are reflected in the AEFI surveillance data.[2,3] The increased AEFI reporting rate for children aged <1 year in the first six months of 2005, compared with the same period for 2004, corresponds in time with the introduction of the universal 7vPCV program in January 2005. Previous reports showed changes in AEFI reporting patterns following the commencement of the national meningococcal C program in January 2003 and conclusion of the catch-up component of the program during 2004, and removal from the immunisation schedule of the fourth dose of diphtheria-tetanus-acellular pertussis, due at 18 months of age, in September 2003.[2,3]

Overall, AEFI reporting rates for the first six months of 2005 have decreased among children aged <7 years for most vaccines funded under the NIP compared with average reporting rates for the previous four years. The reporting rate of serious AEFIs among Australian children remains low, with the majority of reported AEFIs being mild, transient events.

Acknowledgment

We thank Nick Wood, Peter McIntyre and David Isaacs for their assistance with this report.

References

1. Lawrence G, Menzies R, Burgess M, McIntyre P, Wood N, Boyd I, Purcell P, Isaacs D. Surveillance of adverse events following immunisation: Australia, 2000–2002. *Commun Dis Intell* 2003; 27:307–323.

2. Lawrence G, Boyd I, McIntyre P, Isaacs D. Annual report: surveillance of adverse events following immunisation in Australia, 2004. *Commun Dis Intell* 2005;29:248–262.

3. Lawrence G, Boyd I. Adverse events following immunisation for children aged less than 7 years, 1 January to 30 June 2004. *Commun Dis Intell* 2004;28:324–323.

4. Australian Government Department of Health and Ageing. National childhood pneumococcal vaccination program – information for immunisation providers. Available from: http://immunise.health.gov.au/universal/providers.htm Accessed on 8 November 2005.

5. National Health and Medical Research Council. *The Australian Immunisation Handbook*. 8th edn. Canberra: Australian Government Publishing Service; 2003.

6. Wise R, Iskander J, Pratt R, Campbell S, Ball R, Pless R, Braun M. Post licensure safety surveillance for 7-valent pneumococcal conjugate vaccine. *JAMA* 2004;292:1702–1710.

Erratum

Table 1 in the adverse events following immunisation annual report published in the last issue of *CDI*, (Lawrence GL, Boyd I, McIntyre PB, Isaacs D. Annual report: surveillance of adverse events following immunisation in Australia, 2004 *Commun Dis Intell* 2005;29:248-262), was incorrect. The rows for the Australian Capital Territory and New South Wales were omitted. The corrected table has been reprinted below.

Table 1. Adverse events following immunisation (AEFI), ADRAC database, 1 January to 31 December 2004, by jurisdiction

Jurisdiction	AEFI records		Annual reporting rate per 100,000 population*			
	n	%	Overall	'Certain' or 'probable' causality rating†	'Serious' outcome‡	Aged <7 years
Australian Capital Territory	116	12	35.9	16.0	1.54	179.6
New South Wales	318	33	4.7	2.3	0.46	18.7
Northern Territory	35	4	17.5	9.0	1.50	74.3
Queensland	170	17	4.4	2.1	0.49	25.4
South Australia	127	13	8.3	4.2	0.39	50.7
Tasmania	6	1	1.2	0.2	0.21	0.0
Victoria	123	13	2.5	1.0	0.18	13.9
Western Australia	59	6	3.0	1.1	0.40	21.5
Other§	21	2	na	na	na	na
Total	975	100	4.8	2.2	0.44	24.5

* Average annual rates per 100,000 population calculated using mid-2004 population estimates (Australian Bureau of Statistics).

† See previous report[7] for criteria used to assign causality ratings.

‡ Adverse event following immunisation records defined as 'serious' (i.e. recovery with sequelae, hospitalisation, life-threatening event or death, Table 2).

§ Records where the jurisdiction in which the AEFI occurred was not reported or was unclear, including AEFIs notified by pharmaceutical companies (n = 17).

na Not available.

Communicable diseases surveillance

Highlights for 3rd quarter, 2005

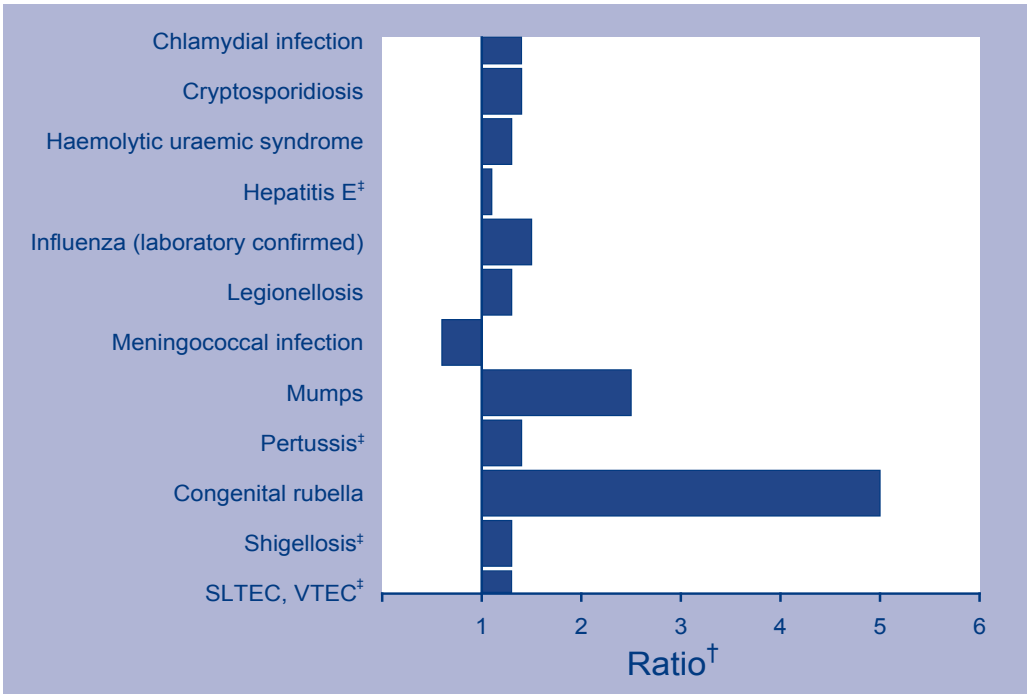
Communicable disease surveillance highlights report on data from various sources, including the National Notifiable Diseases Surveillance System (NNDSS) and several disease specific surveillance systems that provide regular reports to Communicable Diseases Intelligence. These national data collections are complemented by intelligence provided by State and Territory communicable disease epidemiologists and/or data managers. This additional information has enabled the reporting of more informative highlights each quarter.

The NNDSS is conducted under the auspices of the Communicable Diseases Network Australia. NNDSS collates data on notifiable communicable diseases from State or Territory health departments. The Virology and Serology Laboratory Reporting Scheme (LabVISE) is a sentinel surveillance scheme which collates information on laboratory diagnosis of communicable diseases. In this report, data from the NNDSS are referred to as 'notifications' or 'cases', and those from ASPREN are referred to as 'consultations' or 'encounters' while data from the LabVISE scheme are referred to as 'laboratory reports'.

Figure 1 shows the changes in select disease notifications with an onset in the third quarter of 2005 compared with the five-year mean for the same period. The number of notifications received in the quarter was above the five-year mean for hepatitis D, cryptosporidiosis, Shiga-like toxin-producing *Escherichia coli*/verotoxin-producing *E. coli* (STLEC/VTEC), haemolytic uraemic syndrome, hepatitis E, shigellosis, gonococcal infection, chlamydial infection, salmonella infection, laboratory-confirmed influenza, mumps, pertussis, congenital rubella, syphilis, congenital syphilis, Barmah Forest virus infection, Ross River virus infection and legionellosis. The number of notifications received was below the five-year mean for meningococcal infection.

The number of notifications of the following diseases were above the five-year mean plus two standard deviations: hepatitis E, pertussis, shigellosis and STLEC/VTEC (Figure 1).

Figure 1. Selected* diseases from the National Notifiable Diseases Surveillance System, comparison of provisional totals for the period 1 July to 30 September 2005 with historical data†



* Selected diseases are chosen each quarter according to current activity.

† Ratio of current quarter total to mean of corresponding quarter for the previous five years.

‡ Notifications above or below the 5 year mean plus two standard deviations for the same period.

Gastrointestinal illnesses

Botulism

One case of infant botulism in a 5-month-old male from Queensland was reported in this quarter. The source of infection was unknown.

Cryptosporidiosis

There were 311 notifications of cryptosporidiosis during the quarter which is 1.4 times the five-year mean for the same period. The majority of cases were reported by New South Wales and Victoria (87 cases each), and Queensland (64).

Two hundred and thirty-eight notifications (76.5%) were identified as *Cryptosporidium parvum* infection which is the most important species in human disease.[1] There was no species information provided for the remaining 23.5 per cent of cases.

Shiga-like toxin-producing *Esherichia colil* verotoxin-producing *E. coli*

Thirteen notifications of SLTEC/VTEC were received during the quarter, which is 1.3 times the five-year mean for the same period. South Australia notified four cases, Victoria reported three, and New South Wales, Queensland and Western Australia reported two cases each. Of the five cases with serotype information, two were *E. coli* serotype 0157, two were serotype 026 and one was serotype 077.

Hepatitis E

Four notifications of hepatitis E were received in the quarter. There were two cases each from New South Wales and Queensland. Two of the four cases were acquired overseas and the place of acquisition was unknown for the other cases. There have been 28 cases for the year to date in 2005.

Shigellosis

There were 137 notifications of shigellosis during the quarter, which is 1.3 times the five-year mean for the same period. The notifications were from the Northern Territory (36), Western Australia (31), New South Wales (24), and Victoria (21).

Ten per cent (14/137) of cases were reported as imported from overseas, 36 per cent were locally acquired and the place of acquisition of the rest was unknown (54%). Thirty per cent (41/137) of cases occurred in children aged under five years, and 23 per cent (32/137) were in the 15–34 year age range. Of the 137 notifications, 65 (47%) were notifications of *Shigella sonnei*, and 60 (44%) were *Shigella flexneri*. Twelve cases did not have subtyping information.

Sexually transmissible infections

Chlamydial infection

During the quarter there were 9,003 notifications of chlamydial infection received from states and territories, which is 1.4 times the five-year mean for the same period. The majority of these notifications were reported by New South Wales (2,372), Queensland (2,226), Victoria (1,853) and Western Australia (1,377).

Thirty-eight per cent (3,416/9,003) of the total number of infections occurred in the 20–24 year age group and 23 per cent (2,099/9,003) occurred in the 15–19 year age group. The highest rate of chlamydial infection (307 cases per 100,000 population) occurred in females in the 20–24 year age group. The highest rate in males was 179 cases per 100,000 population in the same age range.

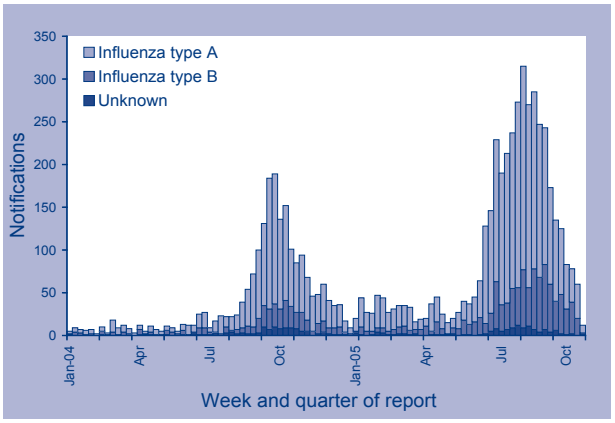
Vaccine preventable diseases

Laboratory confirmed influenza

There were 2,814 cases of laboratory-confirmed influenza in the third quarter of 2005. This was one and a half times the five-year mean for the same period.

One thousand two hundred and fifty-three cases were from Queensland, 603 from New South Wales, 355 from Western Australia and 318 from Victoria. Seventy-four per cent of the national laboratory-confirmed influenza notifications were type A, 22 per cent were type B, one per cent were mixed infections (mainly from New South Wales), and three per cent were of unknown type. During this quarter, influenza notifications peaked for 2005, with 315 notifications in the week ending 9 August. In 2004, the greatest number of notifications was 189 in the week ending 28 September (Figure 2).

Figure 2. Notifications of laboratory confirmed influenza, Australia, 1 January 2004 to 31 October 2005



Mumps

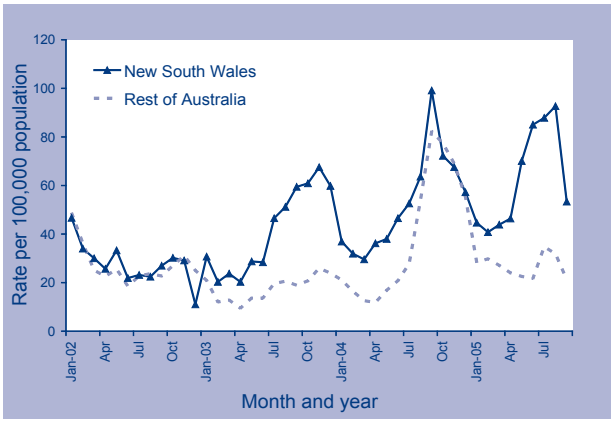
There were 73 notifications of mumps in the third quarter of 2005, which is 2.4 times the five-year mean for the same period. The majority of cases were reported from Queensland (34), New South Wales (21) and Western Australia (10). Of the 73 cases, 41 (56%) cases were reported from the 20–34 year age range.

There were 10 mumps cases notified in the Perth metropolitan area, compared to three in the corresponding period in 2004. Six cases were aged between 19 and 24 years, and four cases were aged over 35 years. Three cases with onset dates within a four week period were students at the same university, two of whom resided at different residential colleges. No epidemiological links could be established between any of the students, however, two had returned from overseas trips recently and hence may have imported the infection coincidentally. Public health response included isolation of the students, contact tracing and promotion of the measles-mumps-rubella vaccination for students residing at the colleges.

Pertussis

For the third quarter, 3,056 pertussis notifications were received, which is 1.4 times the five-year mean for the same period. Of these 1,749 (57%) cases were reported from New South Wales where the pertussis notification rate has remained greater than the overall Australian rate since 2003 and has mirrored national trends (Figure 3).

Figure 3. Notification rates of pertussis, New South Wales compared to the rest of Australia, 1 January 2002 to 30 September 2005



Three per cent (92 cases) of the 3,056 notifications were reported in infants less than one year of age. Rates of pertussis infection were generally highest in the over 55 year age groups, and were generally higher in females than in males.

Congenital rubella

One case of congenital rubella was reported from Victoria this quarter. The child's mother was born overseas, and during pregnancy was found to have no immunity to rubella so was vaccinated after delivery. There are some doubts concerning this diagnosis that will be followed up with further laboratory testing in several months. The last reported case prior to this occurred in March 2004.

Other bacterial infections

Legionellosis

Eighty-one cases of legionellosis were notified this quarter, of which 24 were from Queensland, 21 from New South Wales and 13 each from South Australia and Western Australia. This is 1.3 times the five-year mean for the same period.

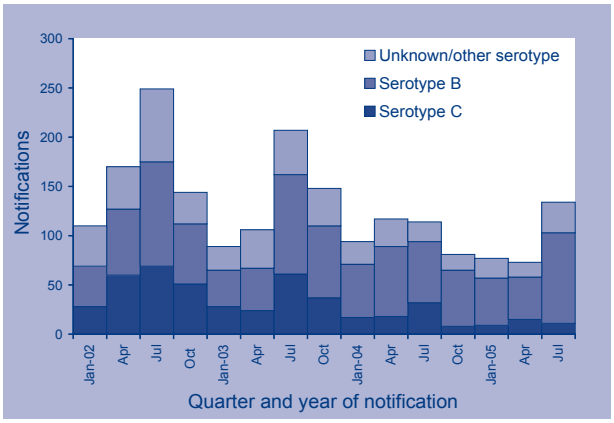
Of the 81 cases, 45 cases (55%) were *Legionella pneumophilia* and 30 were *Legionella longbeachae* (37%). Forty-seven cases (58%) occurred in males giving a 1.3:1 male to female ratio.

Meningococcal infection

There were 135 notifications of meningococcal infection in Australia in the third quarter of 2005. This number is well below the five-year mean for this period (208 notifications) although this is the peak season. There were 38 cases in New South Wales, 36 in Victoria, 20 in Queensland and 19 in Western Australia.

Meningococcal B infection accounted for 92 (68%) notifications this quarter, and 11 (8%) cases were meningococcal C infection. In previous years, the proportion of meningococcal C infections notified in the third quarter has been between 27 per cent and 29 per cent (Figure 4). Twenty per cent of the cases this quarter were not typed. There were four cases of W135 and one case of 29-E, imported from New Zealand. In Australia, the last reported case of 29-E serotype prior to this occurred in 1997.

Figure 4. Notifications of meningococcal disease, Australia, 1 January 2002 to 30 September 2005



In Victoria in August there were three separate instances of epidemiologically-linked cases of invasive meningococcal disease. The first occurred in two children aged two and three years, respectively, who attended the same family day care centre. The first case was laboratory-confirmed as serogroup B while the second case, with a date of onset seven days after the first case, was classified as probable. Both cases had been vaccinated with serogroup C vaccine.

The second outbreak occurred in two vaccinated children aged 8 and 10 years who attended the same small primary school. The first case was laboratory-confirmed as serogroup B and the second, with an onset three days after the first case, was confirmed by serology only as culture and polymerase chain reaction tests were negative.

The third outbreak involved two females aged 17 and 44 years with the same date of onset. Both had attended a family reunion six days earlier and both cases were confirmed as serogroup B.

References

1. Kosek M, Alcantara C, Lima AAM, Guerrant RL. Cryptosporidiosis: an update. *Lancet Infect Dis* 2001:1:262-268.

Acknowledgements

Thanks to James Fielding (DHS, Victoria) and Carolien Giele (WA Health) for their contributions.

Tables

A summary of diseases currently being reported by each jurisdiction is provided in Table 1. There were 27,665 notifications to the National Notifiable Diseases Surveillance System (NNDSS) with a notification date between 1 July and 30 September 2005 (Table 2). The notification rate of diseases per 100,000 population for each State or Territory is presented in Table 3.

There were 7,022 reports received by the Virology and Serology Laboratory Reporting Scheme (LabVISE) in the reporting period, 1 July to 30 September 2005 (Tables 4 and 5).

Table 1. Reporting of notifiable diseases by jurisdiction

Disease	Data received from:
Bloodborne diseases	
Hepatitis B (incident)	All jurisdictions
Hepatitis B (unspecified)	All jurisdictions
Hepatitis C (incident)	All jurisdictions except Qld
Hepatitis C (unspecified)	All jurisdictions
Hepatitis D	All jurisdictions
Gastrointestinal diseases	
Botulism	All jurisdictions
Campylobacteriosis	All jurisdictions except NSW
Cryptosporidiosis	All jurisdictions
Haemolytic uraemic syndrome	All jurisdictions
Hepatitis A	All jurisdictions
Hepatitis E	All jurisdictions
Listeriosis	All jurisdictions
Salmonellosis	All jurisdictions
Shigellosis	All jurisdictions
SLTEC, VTEC	All jurisdictions
Typhoid	All jurisdictions
Quarantinable diseases	
Cholera	All jurisdictions
Plague	All jurisdictions
Rabies	All jurisdictions
Smallpox	All jurisdictions
Tularemia	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 (all)	All jurisdictions
Syphilis < 2 years duration	All jurisdictions
Syphilis > 2 years or unspecified duration	All jurisdictions
Syphilis - congenital	All jurisdictions

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
Vectorborne diseases	
Barmah Forest virus infection	All jurisdictions
Flavivirus infection (NEC)†	All jurisdictions
Dengue	All jurisdictions
Japanese encephalitis virus	All jurisdictions
Kunjin virus	All jurisdictions
Malaria	All jurisdictions
Murray Valley encephalitis virus	All jurisdictions
Ross River virus infection	All jurisdictions
Zoonoses	
Anthrax	All jurisdictions
Australian bat lyssavirus	All jurisdictions
Brucellosis	All jurisdictions
Leptospirosis	All jurisdictions
Lyssaviruses unspecified	All jurisdictions
Ornithosis	All jurisdictions
Q fever	All jurisdictions
Other bacterial infections	
Legionellosis	All jurisdictions
Leprosy	All jurisdictions
Meningococcal infection	All jurisdictions
Tuberculosis	All jurisdictions

* Laboratory confirmed influenza is not notifiable in South Australia but reports are forwarded to NNDSS.

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

Table 2. Notifications of diseases received by State and Territory health authorities in the period 1 July to 30 September 2005, by date of onset*

Disease	State or territory								Total 3rd quarter 2005†	Total 2nd quarter 2005	Total 3rd quarter 2004	Last 5 years mean 3rd quarter	Year to date 2005	Last 5 years YTD mean	Ratio‡
	ACT	NSW	NT	Qld	SA	Tas	Vic	WA							
Bloodborne diseases															
Hepatitis B (incident)	1	6	2	12	1	1	16	5	44	61	78	102.4	179	291.6	0.4
Hepatitis B (unspecified)	20	704	35	285	52	9	417	85	1,607	1,902	1,504	1,811.4	5,390	5,185.2	0.9
Hepatitis C (incident)	0	9	3	NN	7	1	10	20	50	87	128	132.0	211	388.8	0.4
Hepatitis C (unspecified)	47	1,420	59	739	87	45	721	202	3,320	3,754	3,079	4,046.8	10,647	12,437.8	0.8
Hepatitis D	0	7	0	5	0	0	0	0	12	4	10	8.2	20	21.2	1.5
Gastrointestinal diseases															
Botulism	0	0	0	1	0	0	0	0	1	1	0	0.0	3	0.3	0.0
Campylobacteriosis§	68	NN	36	1,124	392	170	1,183	463	3,436	3,521	3,766	3,603.2	11,112	10,776.8	1.0
Cryptosporidiosis‖	1	87	5	64	26	4	87	37	311	826	245	227.3	2,384	1,591.5	1.4
Haemolytic uraemic syndrome	0	1	0	1	0	0	0	1	3	4	4	2.4	11	7.4	1.3
Hepatitis A	0	19	23	8	4	0	15	5	74	86	71	108.4	241	389.2	0.7
Hepatitis E	0	2	0	2	0	0	0	0	4	7	5	3.8	28	13.2	1.1
Listeriosis	2	3	0	0	1	0	1	1	8	14	16	12.6	35	50.0	0.6
Salmonellosis (NEC)	17	277	69	352	96	19	203	142	1,175	1,960	1,171	1,089.4	5,829	5,415.4	1.1
Shigellosis	3	24	36	11	10	1	21	31	137	182	88	103.4	547	388.0	1.3
SLTEC, VTEC¶	0	2	0	2	4	0	3	2	13	30	11	10.4	56	37.4	1.3
Typhoid	0	5	0	0	0	0	2	2	9	10	15	14.2	41	51.8	0.6
Quarantinable diseases															
Cholera	0	0	0	0	0	0	0	1	1	1	2	1.4	4	3.0	0.7
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
Smallpox	0	0	0	0	0	0	0	0	0	0	0	0.0	0	0.0	0.0
Tularemia	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 in the period 1 July to 30 September 2005, by date of onset,* *continued*

Disease	State or territory								Total 3rd quarter 2005†	Total 2nd quarter 2005	Total 3rd quarter 2004	Last 5 years mean 3rd quarter	Year to date 2005	Last 5 years YTD mean	Ratio‡
	ACT	NSW	NT	Qld	SA	Tas	Vic	WA							
Sexually transmissible infections															
Chlamydial infection**	172	2,372	284	2,226	528	191	1,853	1,377	9,003	10,895	8,889	6,469.6	30,203	19,173.6	1.4
Donovanosis	0	0	1	1	0	0	0	0	2	2	3	4.0	9	15.0	0.5
Gonococcal infection	13	333	314	316	63	10	277	347	1,673	2,060	1,685	1,582.0	5,776	4,975.2	1.1
Syphilis (all)	2	224	30	71	1	10	107	58	503	629	589	510.2	1,708	350.2	1.0
Syphilis < two years duration	1	29	16	30	1	2	22	4	105	153	166	NN	405	356.0	NN
Syphilis > two years or unspecified duration	1	195	14	41	0	8	85	54	398	476	423	NN	1,303	962.0	NN
Syphilis - congenital	0	1	0	1	3	0	2	0	7	6	7	3.8	20	10.4	1.8
Vaccine preventable disease															
Diphtheria	0	0	0	0	0	0	0	0	0	0	0	0.0	0	0.2	0.0
Haemophilus influenzae type b	0	0	1	1	0	0	0	1	3	1	6	6.4	11	19.2	0.5
Influenza (laboratory confirmed)‖	27	603	37	1,253	209	12	318	355	2,814	794	1,095	1,931.5	3,989	2,299.3	1.5
Measles	0	1	0	0	0	0	0	1	2	2	10	18.0	9	64.4	0.1
Mumps	0	21	1	34	2	0	5	10	73	74	21	29.0	195	90.2	2.5
Pertussis	68	1,749	15	537	350	3	266	68	3,056	2,522	3,034	2,144.4	7,812	4,576.2	1.4
Pneumococcal disease (invasive)‖	4	213	24	131	44	18	90	51	575	455	961	852.8	1,292	1,701.3	0.7
Poliomyelitis	0	0	0	0	0	0	0	0	0	0	0	0.0	0	0.0	0.0
Rubella	0	1	0	3	0	0	1	1	6	15	5	48.0	27	124.6	0.1
Rubella - congenital	0	0	0	0	0	0	1	0	1	0	0	0.2	1	0.8	5.0
Tetanus	0	0	0	0	0	0	0	0	0	0	0	0.4	0	3.4	0.0
Vectorborne diseases															
Barmah Forest virus infection	0	72	6	105	4	0	5	5	197	435	193	143.2	992	846.2	1.4
Dengue	1	7	1	8	1	0	9	4	31	43	30	30.8	170	289.8	1.0
Flavivirus infection (NEC)	0	0	0	5	0	0	1	0	6	8	6	10.0	23	52.0	0.6
Japanese encephalitis virus‖	0	0	0	0	0	0	0	0	0	0	0	0.0	0	0.4	0.0
Kunjin virus‖	0	0	0	0	0	0	0	0	0	0	1	0.2	1	8.0	0.0
Malaria	2	26	10	60	10	6	23	18	155	167	137	149.8	670	515.6	1.0
Murray Valley encephalitis virus‖	0	0	0	0	0	0	0	0	0	0	0	0.2	2	2.3	0.0
Ross River virus infection	1	71	20	117	9	0	6	19	243	556	152	170.8	1,715	3,036.0	1.4

Table 2. Notifications of diseases received by State and Territory health authorities in the period 1 July to 30 September 2005, by date of onset,* *continued*

Disease	State or territory								Total 3rd quarter 2005†	Total 2nd quarter 2005	Total 3rd quarter 2004	Last 5 years mean 3rd quarter	Year to date 2005	Last 5 years YTD mean	Ratio‡
	ACT	NSW	NT	Qld	SA	Tas	Vic	WA							
Zoonoses															
Anthrax‖	0	0	0	0	0	0	0	0	0	0	0	0.0	0	0.0	0.0
Australian bat lyssavirus‖	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	4	11	7.8	24	20.4	1.0
Leptospirosis	0	4	1	11	0	0	2	2	20	33	29	31.0	93	155.6	0.6
Lyssavirus unspecified‖	0	0	0	0	0	0	0	0	0	0	0	0.0	0	0.0	0.0
Ornithosis	0	21	0	2	0	0	4	1	28	50	54	52.6	115	133.4	0.5
Q fever	0	24	0	16	1	0	9	2	52	113	123	139.4	244	463.0	0.4
Other bacterial infections															
Legionellosis	1	21	1	24	13	0	8	13	81	74	59	64.8	240	260.8	1.3
Leprosy	0	0	0	0	0	0	0	0	0	1	2	1.4	5	5.2	0.0
Meningococcal infection††	3	39	3	20	12	3	36	19	135	77	114	208.0	285	451.2	0.6
Tuberculosis	0	22	1	35	5	4	98	13	178	248	256	252.8	659	720.6	0.7
Total	453	8,392	1,018	7,590	1,935	507	5,800	3,360	29,055	31,714	27,665	26,193.0	93,028	78,731.6	1.1

* Date of onset = the true onset. If this is not available, the 'date of onset' is equivalent to the earliest of two dates: (i) specimen date of collection, or (ii) the date of notification to the public health unit. Hepatitis B and C unspecified were analysed by the date of notification.

† 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.

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

‖ Notifiable from January 2001 only. Ratio and mean calculations are based the last three years.

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

** Includes *Chlamydia trachomatis* identified from cervical, rectal, urine, urethral, throat and eye samples, except for South Australia which reports only genital tract specimens, Northern Territory which excludes 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.

Table 3. Notification rates of diseases, 1 July to 30 September 2005, by state or territory. (Rate per 100,000 population)

Disease*	State or territory								Australia
	ACT	NSW	NT	Qld	SA	Tas	Vic	WA	
Bloodborne diseases									
Hepatitis B (incident)	1.2	0.4	4.0	1.2	0.3	0.8	1.3	1.0	0.9
Hepatitis B (unspecified)	24.7	41.8	70.0	29.4	13.6	7.5	33.5	17.2	32.0
Hepatitis C (incident)	0.0	0.5	6.0	NN	1.8	0.8	0.8	4.0	1.2
Hepatitis C (unspecified)	58.0	84.4	118.1	76.1	22.7	37.3	58.0	40.8	66.0
Hepatitis D	0.0	0.4	0.0	0.5	0.0	0.0	0.0	0.0	0.2
Gastrointestinal diseases									
Botulism	0.0	0.0	0.0	0.1	0.0	0.0	0.0	0.0	0.0
Campylobacteriosis†	83.9	NN	72.0	115.8	102.2	141.0	95.2	93.4	102.7
Cryptosporidiosis	1.2	5.2	10.0	6.6	6.8	3.3	7.0	7.5	6.2
Haemolytic uraemic syndrome	0.0	0.1	0.0	0.1	0.0	0.0	0.0	0.2	0.1
Hepatitis A	0.0	1.1	46.0	0.8	1.0	0.0	1.2	1.0	1.5
Hepatitis E	0.0	0.1	0.0	0.2	0.0	0.0	0.0	0.0	0.1
Listeriosis	2.5	0.2	0.0	0.0	0.3	0.0	0.1	0.2	0.2
Salmonellosis (NEC)	21.0	16.5	138.1	36.3	25.0	15.8	16.3	28.7	23.4
Shigellosis	3.7	1.4	72.0	1.1	2.6	0.8	1.7	6.3	2.7
SLTEC, VTEC‡	0.0	0.1	0.0	0.2	1.0	0.0	0.2	0.4	0.3
Typhoid	0.0	0.3	0.0	0.0	0.0	0.0	0.2	0.4	0.2
Quarantinable diseases									
Cholera	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.2	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
Smallpox	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
Tularemia	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§	212.3	141.0	568.2	229.4	137.7	158.5	149.1	277.9	179.1
Donovanosis	0.0	0.0	2.0	0.1	0.0	0.0	0.0	0.0	0.0
Gonococcal infection	16.0	19.8	628.3	32.6	16.4	8.3	22.3	70.0	33.3
Syphilis (all)	0.6	3.3	14.9	1.8	0.1	2.1	2.1	2.9	2.5
Syphilis < 2 years duration	0.3	0.4	8.0	0.8	0.1	0.4	0.4	0.2	0.5
Syphilis > 2 years or unspecified duration	0.3	2.9	7.0	1.0	0.0	1.7	1.7	2.7	2.0
Syphilis - congenital	0.0	0.0	0.0	0.0	0.2	0.0	0.0	0.0	0.0

Table 3. Notification rates of diseases, 1 July to 30 September 2005, by state or territory. (Rate per 100,000 population), *continued*

Disease*	State or territory								Australia
	ACT	NSW	NT	Qld	SA	Tas	Vic	WA	
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	2.0	0.1	0.0	0.0	0.0	0.2	0.1
Influenza (laboratory confirmed)	33.3	35.8	74.0	129.1	54.5	10.0	25.6	71.6	56.0
Measles	0.0	0.1	0.0	0.0	0.0	0.0	0.0	0.2	0.0
Mumps	0.0	1.2	2.0	3.5	0.5	0.0	0.4	1.6	1.4
Pertussis	83.9	103.9	30.0	55.3	91.2	2.5	21.4	13.7	60.8
Pneumococcal disease (invasive)	4.9	12.7	48.0	13.5	11.5	14.9	7.2	10.3	11.4
Poliomyelitis	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
Rubella	0.0	0.1	0.0	0.3	0.0	0.0	0.1	0.2	0.1
Rubella - congenital	0.0	0.0	0.0	0.0	0.0	0.0	0.1	0.0	0.0
Tetanus	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
Vectorborne diseases									
Barmah Forest virus infection	0.0	4.3	12.0	10.8	1.0	0.0	0.4	1.0	3.9
Dengue	1.2	0.4	2.0	0.8	0.3	0.0	0.7	0.8	0.6
Flavivirus infection (NEC)	0.0	0.0	0.0	0.5	0.0	0.0	0.1	0.0	0.1
Japanese encephalitis virus	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
Kunjin virus	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
Malaria	2.5	1.5	20.0	6.2	2.6	5.0	1.9	3.6	3.1
Murray Valley encephalitis virus	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
Ross River virus infection	1.2	4.2	40.0	12.1	2.3	0.0	0.5	3.8	4.8
Zoonoses									
Anthrax	0.0	0.0	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	0.0	0.7	0.0	0.0	0.0	0.0	0.2
Leptospirosis	0.0	0.2	2.0	1.1	0.0	0.0	0.2	0.4	0.4
Lyssavirus unspecified	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
Ornithosis	0.0	1.2	0.0	0.2	0.0	0.0	0.3	0.2	0.6
Q fever	0.0	1.4	0.0	1.6	0.3	0.0	0.7	0.4	1.0
Other bacterial infections									
Legionellosis	1.2	1.2	2.0	2.5	3.4	0.0	0.6	2.6	1.6
Leprosy	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
Meningococcal infection‖	3.7	2.3	6.0	2.1	3.1	2.5	2.9	3.8	2.7
Tuberculosis	0.0	1.3	2.0	3.6	1.3	3.3	7.9	2.6	3.5

* 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* (SLTEC/VTEC).

§ Includes *Chlamydia trachomatis* identified from cervical, rectal, urine, urethral, throat and eye samples, except for South Australia which reports only genital tract specimens, Northern Territory which excludes 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.

Table 4. Virology and serology laboratory reports by state or territory* for the reporting period 1 July to 30 September 2005, and total reports for the year†

	State or territory								This period 2005	This period 2004	Year to date 2005	Year to date 2004
	ACT	NSW	NT	Qld	SA	Tas	Vic	WA				
Measles, mumps rubella												
Measles virus	–	–	–	1	–	–	–	–	1	8	4	21
Mumps virus	–	–	–	2	2	–	2	1	7	2	25	5
Rubella virus	–	–	–	2	–	–	1	–	3	3	10	11
Hepatitis viruses												
Hepatitis A virus	–	3	–	5	12	–	–	1	21	17	38	36
Hepatitis D virus	–	–	–	1	4	–	1	–	6	4	12	7
Hepatitis E virus	–	–	–	2	–	–	–	–	2	2	11	13
Arboviruses												
Ross River virus	–	–	9	21	13	–	3	5	51	26	333	707
Barmah Forest virus	–	1	–	21	6	–	–	–	28	42	158	168
Flavivirus (unspecified)	–	–	–	7	–	–	2	–	9	16	29	94
Adenoviruses												
Adenovirus type 1	–	–	–	–	–	–	5	–	5		6	
Adenovirus not typed/pending	–	74	–	26	100	–	11	1	212	344	497	793
Herpesviruses												
Cytomegalovirus	1	85	4	13	192	2	20	–	317	215	728	614
Varicella-zoster virus	3	43	1	211	110	3	9	–	380	664	1,110	1,517
Epstein-Barr virus	–	27	27	209	187	–	8	101	559	687	1,540	1,855
Other DNA viruses												
Poxvirus group not typed	–	–	–	–	–	–	1	–	1		2	2
Parvovirus	–	3	–	20	14	–	7	–	44	166	123	284
Picornavirus family												
Coxsackievirus A16	–	2	–	–	–	–	–	–	2		5	5
Echovirus type 7	–	1	–	–	–	–	–	–	1		7	1
Echovirus type 11	–	1	–	–	–	–	–	–	1	8	4	14
Echovirus type 18	–	3	–	–	–	–	–	–	3	2	13	5
Echovirus type 30	1	11	–	–	–	–	–	–	12	2	31	6
Poliovirus type 1 (uncharacterised)	–	15	–	–	–	–	–	–	15	9	19	15
Poliovirus type 2 (uncharacterised)	–	12	–	–	–	–	–	–	12	5	18	13
Poliovirus type 3 (uncharacterised)	–	4	–	–	–	–	–	–	4	5	6	6
Rhinovirus (all types)	–	63	–	–	11	–	1	–	75	242	246	429
Enterovirus type 71 (BCR)	–	1	–	–	–	–	–	–	1	1	3	3
Enterovirus not typed/pending	1	57	–	8	7	1	2	–	76	46	141	133

Table 4. Virology and serology laboratory reports by state or territory* for the reporting period 1 July to 30 September 2005, and total reports for the year,† *continued*

	State or territory								This period 2005	This period 2004	Year to date 2005	Year to date 2004
	ACT	NSW	NT	Qld	SA	Tas	Vic	WA				
Ortho/ paramyoxviruses												
Influenza A virus	–	136	2	75	244	–	57	–	514	284	634	355
Influenza A virus H3N2	–	1	–	–	–	–	1	–	2		2	
Influenza B virus	–	30	–	18	80	–	16	–	144	93	226	130
Parainfluenza virus type 1	–	8	–	1	5	–	3	–	17	34	45	131
Parainfluenza virus type 2	–	6	–	–	6	–	1	–	13	5	46	11
Parainfluenza virus type 3	–	58	–	5	100	1	10	–	174	230	272	427
Respiratory syncytial virus	1	279	–	87	230	34	82	1	714	1,092	1,545	2,485
Other RNA viruses												
HTLV-1	–	–	–	–	3	–	–	–	3	7	6	13
Rotavirus	–	327	1	–	352	5	69	–	754	451	992	621
Norwalk agent	–	–	–	–	–	–	68	–	68	294	163	491
Other												
Chlamydia trachomatis not typed	–	170	4	611	433	12	6	–	1,236	1,583	3,775	4,015
Chlamydia pneumoniae	–	2	–	–	–	–	–	–	2	3	6	7
Chlamydia psittaci	–	1	–	–	1	–	6	–	8	29	38	138
Mycoplasma pneumoniae	–	40	7	170	100	22	54	30	423	382	925	1,043
Mycoplasma hominis	–	2	–	–	–	–	–	–	2	3	4	4
Coxiella burnetii (Q fever)	1	2	–	7	11	–	13	–	34	42	120	122
Rickettsia prowazeki	–	–	–	–	62	–	–	–	62	33	116	33
Orientia tsutsugamushi	–	–	–	–	27	–	–	–	27	21	46	22
Rickettsia - spotted fever group	–	–	–	–	80	1	–	–	81	50	178	50
Streptococcus group A	–	3	–	144	–	–	33	–	180	137	422	360
Brucella abortus	–	1	–	–	1	–	–	–	2	1	3	5
Brucella species	–	4	–	2	–	–	–	–	6	2	9	5
Bordetella pertussis	1	30	1	48	257	–	65	–	402	491	1,153	759
Bordetella parapertussis	–	–	–	–	–	–	2	–	2		2	1
Legionella pneumophila	–	1	–	–	1	–	1	–	3	12	17	65
Legionella longbeachae	–	–	–	–	16	–	2	–	18	21	37	59
Cryptococcus species	–	–	–	2	2	–	–	–	4	9	29	32
Leptospira species	–	1	–	5	1	–	–	–	7	3	23	19

Table 4. Virology and serology laboratory reports by state or territory* for the reporting period 1 July to 30 September 2005, and total reports for the year,† *continued*

	State or territory								This period 2005	This period 2004	Year to date 2005	Year to date 2004
	ACT	NSW	NT	Qld	SA	Tas	Vic	WA				
Treponema pallidum	–	59	2	110	83	–	–	–	254	295	835	910
Entamoeba histolytica	–	–	–	2	–	–	2	–	4	4	12	9
Toxoplasma gondii	–	7	–	2	2	–	–	–	11	10	31	27
Echinococcus granulosus	–	–	–	–	3	–	–	–	3	5	8	12
Total	9	1,574	58	1,838	2,758	81	564	140	7,022	8,142	16,839	19,118

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

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

– No data received this period.

Table 5. Virology and serology reports by laboratories for the reporting period 1 July to 30 September 2005*

State or territory	Laboratory	July 2005	August 2005	September 2005	Total this period
Australian Capital Territory	The Canberra Hospital	–	–	–	–
New South Wales	Institute of Clinical Pathology and Medical Research, Westmead	153	148	133	434
	New Children's Hospital, Westmead	167	162	131	460
	Repatriation General Hospital, Concord	–	–	–	–
	Royal Prince Alfred Hospital, Camperdown	–	–	–	–
	South West Area Pathology Service, Liverpool	190	207	229	626
Queensland	Queensland Medical Laboratory, West End	566	738	630	1,934
	Townsville General Hospital	–	–	–	–
South Australia	Institute of Medical and Veterinary Science, Adelaide	786	1,066	904	2,756
Tasmania	Northern Tasmanian Pathology Service, Launceston	37	22	20	79
	Royal Hobart Hospital, Hobart	–	–	–	–
Victoria	Monash Medical Centre, Melbourne	89	32	36	157
	Royal Children's Hospital, Melbourne	52	82	81	215
	Victorian Infectious Diseases Reference Laboratory, Fairfield	68	38	78	184
Western Australia	PathCentre Virology, Perth	–	–	–	–
	Princess Margaret Hospital, Perth	–	–	–	–
	Western Diagnostic Pathology	26	103	48	177
Total		2,134	2,598	2,290	7,022

* The complete list of laboratories reporting for the 12 months, January to December 2005, 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 Sentinel Practice Research Network

The Research and Health Promotion Unit of the Royal Australian College of General Practitioners operates the Australian Sentinel Practice Research Network (ASPREN). ASPREN is a network of general practitioners who report presentations of defined medical conditions each week. The aim of ASPREN is to provide an indicator of the burden of disease in the primary health setting and to detect trends in consultation rates.

There are currently about 50 general practitioners participating in the network from all states and territories. Seventy-five per cent of these are in metropolitan areas and the remainder are rural based. Between 4,000 and 6,000 consultations are recorded each week.

The list of conditions is reviewed annually by the ASPREN management committee and an annual report is published.

In 2005, eight conditions are being monitored, four of which are related to communicable diseases. These include influenza, gastroenteritis, varicella and shingles. There are two definitions for influenza for 2005. A patient may be coded once or twice depending on their symptoms. The definition for influenza 1 will include more individuals. Definitions of these conditions were published in Commun Dis Intell *2005;29:91.*

Data from 1 January to 30 September 2005 compared with 2004 are shown as the rate per 1,000 consultations in Figures 5 and 6.

Figure 5. Consultation rates for influenza-like illness, ASPREN, 1 January to 30 September 2005, by week of report

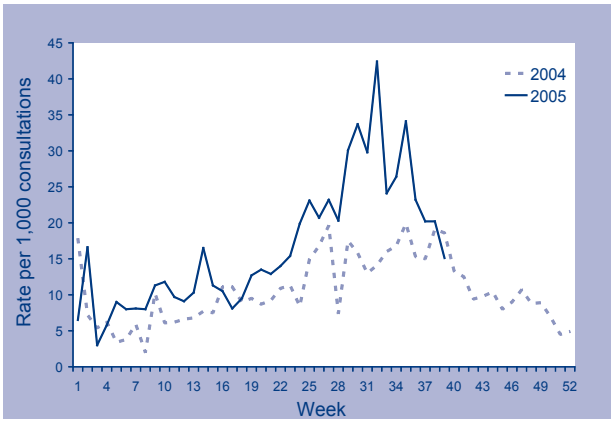
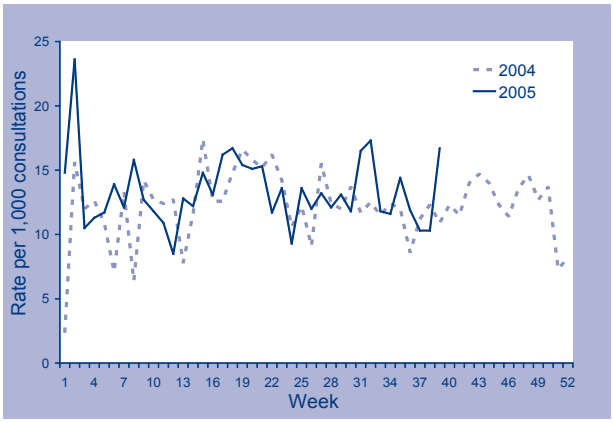


Figure 6. Consultation rates for gastroenteritis, ASPREN, 1 January to 30 September 2005, by week of report



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 *2005;29:93.*

Laboratory confirmed cases of invasive meningococcal disease for the period 1 July to 30 September 2005, are included in this issue of Communicable Diseases Intelligence *(Table 6).*

Table 6. Number of laboratory confirmed cases of invasive meningococcal disease, Australia, 1 July to 30 September 2005, by jurisdiction and serogroup*

Jurisdiction	Year	Serogroup													
		A		B		C		Y		W135		ND		All	
		Q3	ytd	Q3	ytd	Q3	ytd	Q3	ytd	Q3	ytd	Q3	ytd	Q3	ytd
Australian Capital Territory	05			2	*3*	1	*3*			0	*1*			3	*7*
	04			0	*3*	3	*7*							3	*10*
	03			2	*3*	2	*2*							4	*5*
New South Wales	05			27	*60*	4	*13*	0	*3*	4	*7*	2	*3*	37	*86*
	04			22	*60*	6	*15*	1	*3*	2	*4*	3	*14*	34	*96*
	03			38	*75*	19	*32*	1	*4*	0	*1*	3	*15*	61	*127*
Northern Territory	05			2	*5*	0	*2*			0				2	*7*
	04			0	*5*	0	*0*			0	*1*			0	*6*
	03			3	*9*	0	*0*			1	*1*			4	*10*
Queensland	05	0	*0*	13	*34*	5	*7*	0	*0*	0	*0*	0	*0*	18	*45*
	04	0	*1*	13	*36*	8	*20*	0	*1*	1	*2*	0	*2*	22	*62*
	03	1	*1*	17	*34*	16	*31*	1	*1*	0	*0*	0	*8*	35	*75*
South Australia	05			9	*13*	1	*3*							10	*16*
	04			2	*11*	1	*1*							3	*12*
	03			7	*15*	1	*2*	1	*1*	1	*1*			10	*19*
Tasmania	05			4	*6*	0	*0*							4	*6*
	04			3	*6*	5	*5*			0	*1*	1	*3*	9	*15*
	03			3	*3*	4	*5*							7	*8*
Victoria	05			26	*41*	3	*6*	1	*1*	1	*3*	2	*3*	33	*55*
	04			17	*45*	3	*12*	0	*3*	2	*2*	1	*3*	23	*65*
	03			22	*35*	17	*39*	2	*2*	0	*1*	1	*6*	42	*83*
Western Australia	05			20	*29*	0	*0*	0	*2*		0			20	*31*
	04			11	*23*	2	*4*			1	*1*			14	*28*
	03			11	*22*	2	*5*	0	*1*					13	*28*
Total	05	0	*1*	103	*191*	14	*38*	1	*6*	5	*11*	4	*6*	127	*253*
	04	0	*1*	68	*189*	28	*64*	1	*7*	6	*11*	5	*22*	108	*294*
	03	1	*1*	103	*196*	61	*116*	5	*9*	2	*4*	4	*29*	176	*355*

* Numbers of laboratory-confirmed diagnoses of invasive meningococcal disease made in the same period in 2004 and 2003 are also shown.

Q3 3rd quarter.

ytd Year to 30 September 2005.

Gonococcal surveillance

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

The Australian Gonococcal Surveillance Programme (AGSP) reference laboratories in the various States and Territories report data on sensitivity to an agreed 'core' group of antimicrobial agents quarterly. The antibiotics currently routinely surveyed are penicillin, ceftriaxone, ciprofloxacin and spectinomycin, all of which are administered as single dose regimens and currently used in Australia to treat gonorrhoea. When in vitro resistance to a recommended agent is demonstrated in 5 per cent or more of isolates from a general population, it is usual to remove that agent from the list of recommended treatment.[1] Additional data are also provided on other antibiotics from time to time. At present all laboratories also test isolates for the presence of high level (plasmid-mediated) resistance to the tetracyclines, known as TRNG. Tetracyclines are however, not a recommended therapy for gonorrhoea in Australia. Comparability of data is achieved by means of a standardised system of testing and a program-specific quality assurance process. Because of the substantial geographic differences in susceptibility patterns in Australia, regional as well as aggregated data are presented. For more information see Commun Dis Intell *2005;29:92-93.*

Reporting period 1 April to 30 June 2005

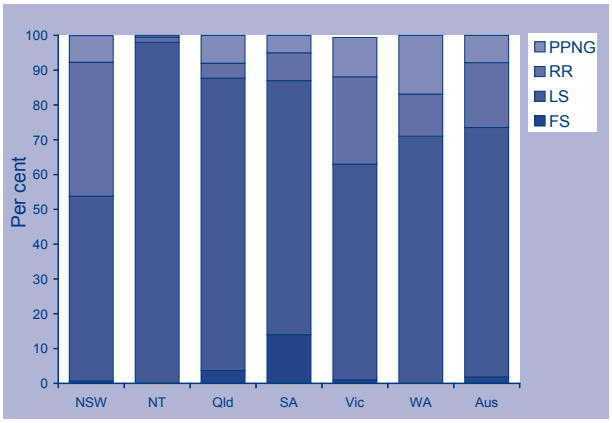
The AGSP laboratories received a total of 1,028 isolates in this quarter of which 1,008 underwent susceptibility testing. This was about 15 per cent more than the 873 gonococci reported for the same period in 2004 and approximates the 980 seen in 2003. Approximately 28 per cent of this total was from New South Wales, 19 per cent each from Victoria and the Northern Territory, 16 per cent from Queensland, 11 per cent from Western Australia and six per cent from South Australia. Small numbers of isolates were also received from Tasmania and the Australian Capital Territory.

Penicillins

In this quarter 267 (26.5%) of all isolates examined were penicillin resistant by one or more mechanisms. Seventy-nine (7.8%) were penicillinase producing *Neisseria gonorrhoea* (PPNG) and 188 (18.7%) resistant by chromosomal mechanisms, (CMRNG). The proportion of all strains resistant to the penicillins by any mechanism ranged from two per cent in the Northern Territory to 46 per cent in New South Wales. High rates of penicillin resistance were also found in Victoria (36%) and Western Australia (29%).

Figure 7 shows the proportions of gonococci fully sensitive (MIC ≤0.03 mg/L), less sensitive (MIC 0.06–0.5 mg/L), relatively resistant (MIC ≥1 mg/L) or else PPNG, aggregated for Australia and by state or territory. A high proportion of those strains classified as PPNG or else resistant by chromosomal mechanisms fail to respond to treatment with penicillins (penicillin, amoxycillin, ampicillin) and early generation cephalosporins.

Figure 7. Categorisation of gonococci isolated in Australia, 1 April to 30 June 2005, by penicillin susceptibility and region



FS Fully sensitive to penicillin, MIC ≤0.03 mg/L.

LS Less sensitive to penicillin, MIC 0.06–0.5 mg/L.

RR Relatively resistant to penicillin, MIC ≥1 mg/L.

PPNG Penicillinase producing *Neisseria gonorrhoeae.*

In New South Wales most of the penicillin resistance was due to CMRNG (111, 38.5%) with 22 PPNG (7.6%). The proportion of CMRNG in Victoria (25%) was less than in New South Wales and that of PPNG higher (11.3%) and in Western Australia PPNG were more prominent (17% of all isolates) with 12 per cent CMRNG. The proportion of PPNG in Queensland was eight per cent and in South Australia five per cent. CMRNG were present in Queensland (4.3% of isolates there), South Australia (5%) and in three (1.5%) gonococci from Darwin. No PPNG or CMRNG were reported from Tasmania or the Australian Capital Territory.

Ceftriaxone

Fifteen isolates with decreased susceptibility to ceftriaxone (MIC range 0.06–0.12 mg/L) were detected. Fourteen were found in New South Wales and one in Queensland. All 15 isolates were penicillin resistant, 14 by chromosomal mechanisms (CMRNG) and one was a PPNG. All 15 were also quinolone resistant (ciprofloxacin MICs 16 mg/L or more). It is emphasised that no treatment failures have been documented locally when a 250 mg IM dose of ceftriaxone has been used.

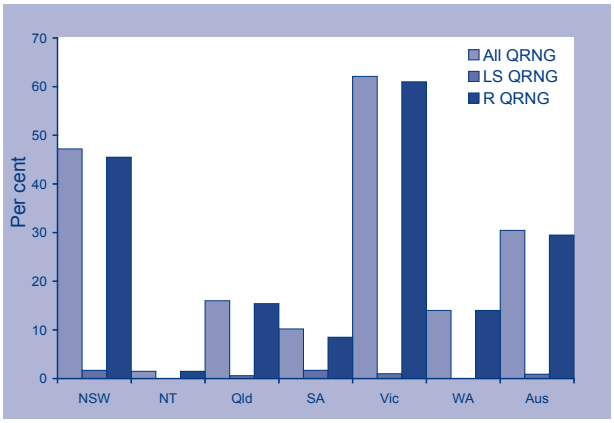
Spectinomycin

All isolates were susceptible to this injectable agent.

Quinolone antibiotics

The total number (307) and proportion (30%) of quinolone resistant *N. gonorrhoeae* (QRNG) were both substantially higher than the corresponding figures in the second quarter of 2004 (172 QRNG, 20%) and 2003 (135 isolates, 14%) (Figure 8). The majority of QRNG (298 of 307, 97%) exhibited higher-level resistance. QRNG are defined as those isolates with an MIC to ciprofloxacin equal to or greater than 0.06 mg/L. QRNG are further subdivided into less sensitive (ciprofloxacin MICs 0.06–0.5 mg/L) or resistant (MIC ≥ 1 mg/L) groups.

Figure 8. The distribution of quinolone resistant isolates of *Neisseria gonorrhoeae* in Australia, 1 April to 30 June 2005, by jurisdiction



LS QRNG Ciprofloxacin MICs 0.06–0.5 mg/L.
R QRNG Ciprofloxacin MICs ≥1 mg/L.

QRNG were again widely distributed and were detected in all states and territories with the exception of Tasmania and the Australian Capital Territory. The highest proportion of QRNG was found in Victoria where 121 QRNG represented 62 per cent of isolates. In New South Wales there were 136 QRNG (47%), in Queensland 26 (16%), in Western Australia 15 (14%) and in South Australia 6 (10%). Three QRNG were detected in the Northern Territory.

High level tetracycline resistance

The number (131) and proportion (13%) of gonococci showing high level plasmid mediated tetracycline resistance (TRNG) detected were similar to the 2004 (121, 14%) figures. TRNG were found in most states and territories and represented between 0.5 per cent (Northern Territory) and 38 per cent of isolates (Victoria).

Reference

1. Management of sexually transmitted diseases. World Health Organization 1997; Document WHO/GPA/TEM94.1 Rev.1 p 37.

Childhood immunisation coverage

Tables 7, 8 and 9 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 April and 30 June 2004, at 24 months of age for the cohort born between 1 April and 30 June 2003, and at 6 years of age for the cohort born between 1 April and 30 June 1999 according to the Australian Standard Vaccination Schedule.

For information about the Australian Childhood Immunisation Register see Surveillance systems reported in CDI, *published in* Commun Dis Intell *2005;29:94 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.

Immunisation coverage for children 'fully immunised' at 12 months of age for Australia did not change from the last quarter, remaining at 91.0 per cent (Table 7). There was a substantial decrease in 'fully immunised' coverage by state and territory in only one jurisdiction, the Australian Capital Territory, but despite this decrease of 2.1 per cent, the Australian Capital Territory had coverage of 93.6 per cent.

There was a small increase of 0.4 per cent in coverage for children 'fully immunised' at 24 months of age for Australia, bringing the coverage to 93.7 per cent (Table 8). Coverage for individual vaccines remained largely unchanged in most jurisdictions and was greater than 95 per cent in almost all jurisdictions for all vaccines, except Hib and MMR.

Table 9 shows immunisation coverage estimates for children 'fully immunised' and for individual vaccines at six years of age for Australia and by state or territory. This was unchanged in most jurisdictions, apart from increases in Tasmania and the Australian Capital Territory. Coverage for vaccines assessed at six years is at or near 85 per cent in most jurisdictions, but Western Australia and Queensland still remain below this.

Table 7. Percentage of children immunised at 1 year of age, preliminary results by vaccine and state or territory for the birth cohort 1 April to 30 June 2004; assessment date 30 September 2005

Vaccine	State or territory								
	ACT	NSW	NT	Qld	SA	Tas	Vic	WA	Australia
Total number of children	952	20,807	900	12,736	4,229	1,305	14,930	6,321	62,180
Diphtheria, tetanus, pertussis (%)	94.0	92.2	92.3	92.0	92.2	93.1	93.6	90.9	92.4
Poliomyelitis (%)	94.0	92.2	91.9	91.9	92.1	93.0	93.5	90.6	92.3
Haemophilus influenzae type b (%)	96.1	93.9	95.2	93.9	94.7	94.9	95.1	93.5	94.3
Hepatitis B (%)	95.8	95.0	95.6	94.6	94.7	94.8	94.9	93.2	94.7
Fully immunised (%)	93.6	90.6	91.7	90.8	91.2	92.0	92.1	89.2	91.0
Change in fully immunised since last quarter (%)	-2.1	+0.0	-0.3	-0.0	+0.1	+0.8	+0.3	-0.8	-0.0

Table 8. Percentage of children immunised at 2 years of age, preliminary results by vaccine and state or territory for the birth cohort 1 April to 30 June 2003, assessment date 30 September 2005

Vaccine	State or territory								
	ACT	NSW	NT	Qld	SA	Tas	Vic	WA	Australia
Total number of children	983	21,749	926	13,035	4,442	1,372	15,394	6,261	64,162
Diphtheria, tetanus, pertussis (%)	97.5	95.1	97.0	94.9	95.8	96.9	95.7	94.6	95.3
Poliomyelitis (%)	97.6	95.1	96.8	94.8	95.7	96.8	95.7	94.5	95.2
Haemophilus influenzae type b (%)	95.5	93.0	95.9	93.7	94.4	94.2	94.1	92.2	93.5
Measles, mumps, rubella (%)	95.7	93.1	96.8	93.6	94.5	94.2	94.3	92.8	93.7
Hepatitis B(%)	96.6	95.9	98.4	95.4	96.4	97.0	96.2	95.4	95.9
Fully immunised (%)	94.2	91.6	95.0	92.0	93.0	93.2	92.9	90.7	92.1
Change in fully immunised since last quarter (%)	+2.6	+0.4	+1.4	+0.3	+2.4	-1.4	+0.1	+0.7	+0.4

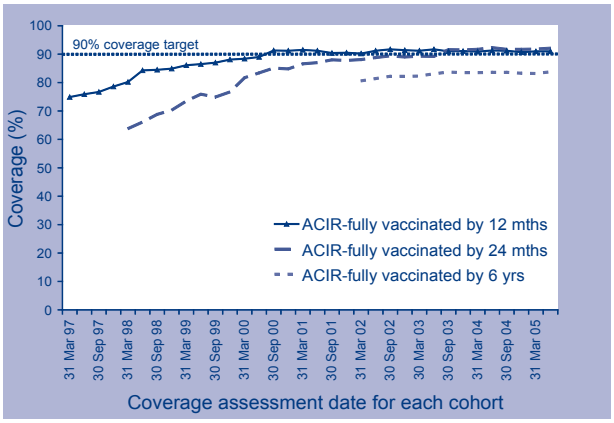
* The 12 months age data for this cohort was published in *Commun Dis Intell* 2004;28:546.

Table 9. Percentage of children immunised at 6 years of age, preliminary results by vaccine and state or territory for the birth cohort 1 April to 30 June 1999; assessment date 30 September 2005

Vaccine	State or territory								
	ACT	NSW	NT	Qld	SA	Tas	Vic	WA	Australia
Total number of children	1,091	22,073	846	13,502	4,777	1,598	15,596	6,884	66,367
Diphtheria, tetanus, pertussis (%)	90.7	85.4	83.6	81.7	84.2	86.1	87.7	82.1	84.8
Poliomyelitis (%)	90.8	85.5	84.9	82.0	84.6	86.5	87.9	82.4	85.1
Measles, mumps, rubella (%)	90.9	85.3	84.5	81.9	84.5	86.1	87.9	82.3	84.9
Fully immunised (%)[1]	90.5	84.3	82.7	80.5	83.3	85.2	86.9	80.5	83.8
Change in fully immunised since last quarter (%)	+2.6	+0.5	-1.5	+0.6	+0.5	+3.3	+0.7	-0.3	+0.5

Figure 9 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 six years, although the rate of increase has slowed over the past two years for all age groups. The Figure shows that there have now been eight consecutive quarters where 'fully immunised' coverage at 24 months of age has been greater than 'fully immunised' coverage at 12 months of age, following the removal of the requirement for the 18 month DTPa vaccine. However, both measures have been above 90 per cent for this 24-month period and show levels of high coverage for the vaccines included maintained over a significant period of time. Currently, coverage for the more recent vaccines, meningococcal C conjugate at 12 months and pneumococcal conjugate at two, four, and six months, are not included in the 12 or 24 months coverage data.

Figure 9. Trends in vaccination coverage, Australia, 1997 to 2005, by age cohorts



Acknowledgement: These figures were provided by Medicare Australia, to specifications provided by the Australian Government Department of Health and Ageing. For further information on these figures or data on the Australian Childhood Immunisation Register please contact the Immunisation Section of Medicare Australia: Telephone: +61 2 6124 6607.

HIV and AIDS surveillance

National surveillance for HIV disease is coordinated by the National Centre in HIV Epidemiology and Clinical Research (NCHECR), in collaboration with State and Territory health authorities and the Commonwealth of Australia. Cases of HIV infection are notified to the National HIV Database on the first occasion of diagnosis in Australia, by either the diagnosing laboratory (Australian Capital Territory, New South Wales, Tasmania, Victoria) or by a combination of laboratory and doctor sources (Northern Territory, Queensland, South Australia, Western Australia). Cases of AIDS are notified through the State and Territory health authorities to the National AIDS Registry. Diagnoses of both HIV infection and AIDS are notified with the person's date of birth and name code, to minimise duplicate notifications while maintaining confidentiality.

Tabulations of diagnoses of HIV infection and AIDS are based on data available three months after the end of the reporting interval indicated, to allow for reporting delay and to incorporate newly available information. More detailed information on diagnoses of HIV infection and AIDS is published in the quarterly Australian HIV Surveillance Report, and annually in 'HIV/AIDS, viral hepatitis and sexually transmissible infections in Australia, annual surveillance report'. The reports are available from the National Centre in HIV Epidemiology and Clinical Research, 376 Victoria Street, Darlinghurst NSW 2010. Internet: http://www.med.unsw.edu.au/nchecr. Telephone: +61 2 9332 4648. Facsimile: +61 2 9332 1837. For more information see Commun Dis Intell *2005;29:91–92.*

HIV and AIDS diagnoses and deaths following AIDS reported for 1 April to 30 June 2005, as reported to 30 September 2005, are included in this issue of Communicable Diseases Intelligence *(Tables 10 and 11).*

Table 10. New diagnoses of HIV infection, new diagnoses of AIDS and deaths following AIDS occurring in the period 1 April to 30 June 2005, by sex and state or territory of diagnosis

	Sex	State or territory								Totals for Australia			
		ACT	NSW	NT	Qld	SA	Tas	Vic	WA	This period 2005	This period 2004	YTD 2005	YTD 2004
HIV diagnoses	Female	0	6	0	0	2	0	8	3	19	33	45	71
	Male	0	106	0	41	16	0	56	12	231	175	430	390
	Not reported	0	0	0	1	0	0	0	0	1	1	1	1
	Total*	0	112	0	42	18	0	64	15	251	209	476	463
AIDS diagnoses	Female	0	1	0	0	0	0	3	0	4	7	12	11
	Male	0	11	0	7	3	0	10	1	32	37	62	82
	Total*	0	12	0	7	3	0	13	1	36	44	74	94
AIDS deaths	Female	0	0	0	0	0	0	0	0	0	2	2	4
	Male	0	4	0	3	1	0	3	1	12	23	24	38
	Total*	0	4	0	3	1	0	3	1	12	25	26	42

* Totals include people whose sex was reported as transgender.

Table 11. Cumulative diagnoses of HIV infection, AIDS and deaths following AIDS since the introduction of HIV antibody testing to 30 June 2005, by sex and state or territory

	Sex	State or territory								
		ACT	NSW	NT	Qld	SA	Tas	Vic	WA	Australia
HIV diagnoses	Female	30	800	18	238	87	8	326	174	1,681
	Male	248	12,911	123	2,513	863	89	4,859	1,124	22,730
	Not reported	0	234	0	1	0	0	22	0	257
	Total*	278	13,972	141	2,761	951	97	5,226	1,305	24,731
AIDS diagnoses	Female	9	234	2	66	31	4	102	35	483
	Male	92	5,205	42	991	392	48	1,895	415	9,080
	Total*	101	5,455	44	1,059	424	52	2,007	452	9,594
AIDS deaths	Female	6	131	1	41	20	2	59	24	284
	Male	71	3,529	26	645	270	32	1,376	290	6,239
	Total*	77	3,670	27	688	290	34	1,443	315	6,544

* Totals include people whose sex was reported as transgender.

National Enteric Pathogens Surveillance System

The National Enteric Pathogens Surveillance System (NEPSS) collects, analyses and disseminates data on human enteric bacterial infections diagnosed in Australia. These pathogens include Salmonella, E. coli, Vibrio, Yersinia, Plesiomonas, Aeromonas *and* Campylobacter. Communicable Diseases Intelligence *NEPSS quarterly reports include only* Salmonella.

Data are based on reports to NEPSS from Australian laboratories of laboratory-confirmed human infection with Salmonella. Salmonella *are identified to the level of serovar and, if applicable, phage-type. Infections apparently acquired overseas are included. Multiple isolations of a single* Salmonella *serovar/phage-type from one or more body sites during the same episode of illness are counted once only. The date of the case is the date the primary diagnostic laboratory isolated a* Salmonella *from the clinical sample.*

Note that the historical quarterly mean counts should be interpreted with caution, and are affected by surveillance artefacts such as newly recognised and incompletely typed Salmonella.

NEPSS is operated by the Microbiological Diagnostic Unit, Public Health Laboratory, Department of Microbiology and Immunology, University of Melbourne; and is overseen by a Steering Committee of state, territory and commonwealth stakeholders. NEPSS can be contacted at the above address or by telephone: +61 3 8344 5701, facsimile: +613 8344 7833 or email joanp@unimelb.edu.au

Reports to the National Enteric Pathogens Surveillance System of Salmonella infection for the period 1 July to 30 September 2005 are included in Tables 12 and 13. Data include cases reported and entered by 25 October 2005. Counts are preliminary, and subject to adjustment after completion of typing and reporting of further cases to NEPSS. For more information see Commun Dis Intell *2005;29:93–94.*

Third quarter 2005

The total number of reports to NEPSS of human *Salmonella* infection fell to 1,206 in the third quarter of 2005, 33 per cent less than in second quarter of 2005, consistent with the usual seasonal nadir of salmonellosis in Australia. However, the third quarter count was four per cent more than the comparable third quarter of 2004 and 17 per cent greater than the ten-year historical mean for this period. Much of this increase is accounted for by reports of common salmonellae, in particular *Salmonella* Typhimurium phage types 135, 170 and 197, and the typically overseas-acquired *S*. Enteritidis phage types 1b and 6a.

During the third quarter of 2005, the 25 most common *Salmonella* types in Australia accounted for 724 cases, 60 per cent of all reported human *Salmonella* infections. Twenty of the 25 most common *Salmonella* infections in the third quarter of 2005 were among the 25 most commonly reported in the second quarter of 2005.

S. Typhimurium PT 135 was the most common serovar/phage type. Reports of *S*. Typhimurium PT 170 declined, manifesting a pattern seen over several years of more marked seasonal variation than most of the other common salmonellae.

Reports of other salmonellae with counts well above historical averages include *S*. Infantis, *S*. Hvittingfoss, and *S*. Typhimurium phage types 12 and U307, and (usually overseas-acquired) *S*. Corvallis.

Acknowledgement: We thank scientists, contributing laboratories, state and territory health departments, and the Australian Government Department of Health and Ageing for their contributions to NEPSS.

Table 12. Reports to the National Enteric Pathogens Surveillance System of *Salmonella* isolated from humans during the period 1 July to 30 September 2005, as reported to 25 October 2005

	ACT	NSW	NT	Qld	SA	Tas	Vic	WA	Australia
Total all *Salmonella* for quarter	24	290	61	312	133	20	214	152	1,206
Total contributing *Salmonella* types	16	99	32	87	48	10	78	69	212

Table 13. Top 25 *Salmonella* types identified in Australia, 1 July to 30 September 2005, by state or territory

National rank	*Salmonella* type	State or territory								Total 3rd quarter 2005	Last 10 years mean 3rd quarter	Year to date 2005	Year to date 2004
		ACT	NSW	NT	Qld	SA	Tas	Vic	WA				
1	*S.* Typhimurium PT 135	6	33	0	39	5	7	35	9	134	81	374	449
2	*S.* Saintpaul	0	7	7	29	2	0	3	5	53	44	317	288
3	*S.* Typhimurium PT 170	0	28	0	9	0	2	12	1	52	21	403	414
4	*S.* Typhimurium PT 64	1	0	0	0	44	0	0	0	45	48	52	8
5	*S.* Typhimurium PT 9	2	19	0	3	5	0	13	1	43	75	341	286
6	*S.* Infantis	0	7	4	4	12	2	5	2	36	21	132	111
7	*S.* Typhimurium PT 197	0	12	0	15	1	0	6	2	36	12	489	205
8	*S.* Enteritidis PT 1b	0	10	0	0	0	0	8	8	26	2	35	26
9	*S.* Chester	0	3	4	7	5	0	1	5	25	22	145	155
10	*S.* Typhimurium RDNC	0	9	1	3	3	0	9	0	25	15	84	81
11	*S.* Enteritidis PT 6a	0	5	0	2	2	0	4	12	25	6	72	50
12	*S.* Virchow PT 8	1	1	1	17	1	1	1	0	23	22	187	270
13	*S.* Birkenhead	0	5	0	18	0	0	0	0	23	22	154	200
14	*S.* Agona	1	6	0	3	2	1	4	4	21	12	52	63
15	*S.* Hvittingfoss	0	6	2	12	0	0	1	0	21	10	156	116
16	*S.* Typhimurium PT 12	0	6	0	1	0	0	9	3	19	6	97	203
17	*S.* Stanley	3	7	0	0	2	0	1	2	15	15	51	60
18	*S.* Anatum	0	0	1	4	2	0	2	5	14	11	52	70
19	*S.* Weltevreden	0	4	4	1	0	0	3	2	14	8	43	58
20	*S.* Corvallis	0	3	0	2	2	0	0	7	14	1	63	28
21	*S.* Newport	0	2	0	0	1	0	7	3	13	8	26	24
22	*S.* Typhimurium PT U307	0	2	0	5	0	0	6	0	13	2	18	11
23	*S.* Aberdeen	0	2	0	10	0	0	0	0	12	12	124	91
24	*S.* Waycross	0	2	0	9	0	0	1	0	12	9	90	99
25	*S.* Ball	0	0	10	0	0	0	0	0	10	7	41	49

Overseas briefs

World Health Organization Disease Outbreak News

This material has been summarised from information provided by the World Health Organization (http://www.who.int).

Poliomyelitis

Indonesia

30 September 2005

Eleven new poliovirus cases were confirmed today from Banten, Lampung and Central Java provinces. This brings the total number of poliovirus cases to 251.

Lampung and Central Java provinces were not included in two emergency vaccination campaigns held on 31 May and 28 June. The 1st round of the National Immunisation Days (NIDs) was held on 30 August; the 2nd round was conducted on 27 September, each time targeting 24.4 million children less than five years of age throughout the country.

Prior to this outbreak (caused by an importation of type 1 wild poliovirus), Indonesia had not had a wild poliovirus case since 1995.

Somalia

13 September 2005

One case of poliomyelitis has been reported in Mogadishu, Somalia where a 15-month-old girl had onset of paralysis on 12 July 2005. Genetic sequencing is ongoing to determine the origin of the virus. Somalia had been polio-free since 2002.

The Global Polio Eradication Initiative – spearheaded by the World Health Organization (WHO), Rotary International, the US Centers for Disease Control and Prevention and UNICEF launched an ambitious new series of polio immunisation campaigns to prevent the re-establishment of the disease in the Horn of Africa.

Ethiopia

6 September 2005

As of 31 August 2005, two new polio cases were reported in Ethiopia, bringing the total number of cases associated with this outbreak to 15 in 2005. One of the two new cases occurred in Oromia Province, in the centre of the country, near the border with Somalia and had onset of paralysis on 1 July 2005. The second case, in Amhara region, the north-western part of Ethiopia, had onset of paralysis on 16 July. Both cases occurred after the National Immunisation Days (NIDs) held on 23 May.

Discussions were ongoing to advance the next NIDs to early October. Ethiopia had been polio-free since January 2001, before a poliovirus was imported into the country from neighbouring Sudan in December 2004.

Angola

25 August 2005

As of 23 August, seven cases of polio have been reported. Five provinces have been affected: the most recent case, in Benguela, had an onset of 12 July. This case occurred before the first National Immunisation Day (NID) campaign on 29 July.

A second NID was to be held on 26 August. In order to ensure rapid interruption of the virus transmission in Angola, a third round of NIDs was planned for late September 2005. The partners in the Polio Eradication Initiative in Angola have asked the international community for financial support to help them carry out this campaign.

Avian influenza

Indonesia

29 September 2005

The Ministry of Health in Indonesia has today confirmed another fatal human case of H5N1 avian influenza. The patient, a 27-year-old woman from Jakarta, developed symptoms on 17 September, was hospitalised on 19 September, and died on 26 September. Confirmatory testing was conducted at a WHO reference laboratory in Hong Kong.

Initial investigation has revealed that the woman had direct contact with diseased and dying chickens in her household shortly before the onset of illness. The woman is the fourth laboratory-confirmed case of H5N1 infection in Indonesia. Three of these cases were fatal.

As a result of intensified surveillance and heightened public concern, growing numbers of people with respiratory symptoms or possible exposure to the virus are being admitted to hospital for observation and, when appropriate, treatment. Until a conclusive diagnosis is made, these patients are classified by the Ministry of Health as suspect cases. While many do not have symptoms compatible with a diagnosis of H5N1 infection, screening of patient samples is being undertaken in national laboratories as part of efforts to ensure that no new cases are missed.

Laboratory testing to confirm human infection with H5N1 avian influenza is technically difficult; some tests produce inconclusive or unreliable results. To ensure a reliable assessment of the situation in Indonesia, authorities are, after initial screening, continuing to send samples from people considered likely to have H5N1 infection to WHO reference laboratories for diagnostic confirmation.

According to the Food and Agriculture Organization (FAO), highly pathogenic H5N1 avian influenza is now endemic in poultry in many parts of Indonesia. As influenza virus activity in Indonesia may increase during the wet season, from November to April, human exposure to animal virus could be greater during the coming months. Further sporadic human cases can be anticipated.

Viet Nam

19 September 2005

The Ministry of Health in Viet Nam has retrospectively confirmed an additional fatal case of H5N1 infection that dates back to July. The case, in a 35-year-old male farmer from Ben Tre Province, developed symptoms on 25 July and died on 31 July.

The newly confirmed case brings the total in Viet Nam since mid-December 2004 to 64 cases, of which 21 were fatal.

Geographical spread of H5N1 avian influenza in birds

Situation assessment and implications for human health

18 August 2005

Beginning in late July 2005, official reports to the OIE from government authorities indicate that the H5N1 virus has expanded its geographical range. Both Russia and Kazakhstan reported outbreaks of avian influenza in poultry in late July, and confirmed H5N1 as the causative agent in early August. Deaths in migratory birds, infected with the virus, have also been reported. Outbreaks in both countries have been attributed to contact between domestic birds and wild waterfowl via shared water sources.

These are the first outbreaks of highly pathogenic H5N1 avian influenza recorded in the two countries. Both countries were previously considered free of the virus.

In early August, Mongolia issued an emergency report following the death of 89 migratory birds at two lakes in the northern part of the country. Avian influenza virus type A has been identified as the cause, but the virus strain has not yet been determined. Samples have been shared with WHO reference laboratories and are currently being investigated. Also in early August, an outbreak of H5N1 in poultry was detected in Tibet, China.

In all of these recent outbreaks, authorities have announced control measures in line with FAO and OIE recommendations for highly pathogenic avian influenza. To date, no human cases have been detected, vigilance is high, and rumours are being investigated by local authorities.

Very large die-offs of migratory birds from avian influenza, such as the one detected at the end of April at Qinghai Lake in central China, in which more than 6,000 birds died, are considered unusual. Research published in July indicates that H5N1 viruses in that outbreak are similar to viruses that have been circulating in South East Asia for the last two years.

Analyses of viruses from the Russian outbreak, recently published on the OIE website, show apparent similarity to viruses isolated from migratory birds during the Qinghai Lake outbreak. Specimens from the Mongolian outbreak in migratory birds should also prove useful in shedding light on these recent developments. Monitoring the spread and evolution of avian H5N1 viruses in birds and rapidly comparing these results with previously characterised H5N1 viruses is an essential activity for assessing the risk of pandemic influenza.

Implications for human health

The poultry outbreaks in Russia and Kazakhstan are caused by a virus that has repeatedly demonstrated its ability, in outbreaks in Hong Kong in 1997, in Hong Kong in 2003, and in South East Asia since early 2004, to cross the species barrier to infect humans, causing severe disease with high fatality. A similar risk of human cases exists in areas newly affected with H5N1 disease in poultry.

Experience in South East Asia indicates that human cases of infection are rare, and that the virus does not transmit easily from poultry to humans. To date, the majority of human cases have occurred in rural areas. Most, but not all, human cases have been linked to direct exposure to dead or diseased poultry, notably during slaughtering, de-feathering, and food preparation. No cases have been confirmed in poultry workers or cullers. No cases have been linked to the consumption of properly cooked poultry meat or eggs.

Pandemic risk assessment

The possible spread of H5N1 avian influenza to poultry in additional countries cannot be ruled out. WHO recommends heightened surveillance for outbreaks in poultry and die-offs in migratory birds, and rapid introduction of containment measures, as recommended by FAO and OIE. Heightened vigilance for cases of respiratory disease in persons

with a history of exposure to infected poultry is also recommended in countries with known poultry outbreaks. The provision of clinical specimens and viruses, from humans and animals, to WHO and OIE/FAO reference laboratories allows studies that contribute to the assessment of pandemic risk and helps ensure that work towards vaccine development stays on course.

The expanding geographical presence of the virus is of concern as it creates further opportunities for human exposure. Each additional human case increases opportunities for the virus to improve its transmissibility, through either adaptive mutation or reassortment. The emergence of an H5N1 strain that is readily transmitted among humans would mark the start of a pandemic.

Japanese encephalitis in India

13 September 2005

One thousand one hundred and forty-five cases of Japanese encephalitis have been reported from 14 districts of Uttar Pradesh Province, India from 29 July to 30 August 2005. About one-fourth of these (296) have died. Ninety cases from the adjoining districts of Bihar have also been admitted to the hospitals in Uttar Pradesh.

The majority of the affected villages have reported only single cases. Entomological surveys in the affected villages have revealed high density of *Culex tritaeniorhynchus* and *Culex vishnui* group – the vectors of Japanese encephalitis.

Outbreak associated with Streptococcus suis *in pigs in China*

3 August 2005

To date, the Ministry of Health in China has reported 206 cases of human disease associated with an outbreak of *Streptococcus suis* in pigs. Of these human cases, 38 have been fatal and 18 patients are critically ill.

Virtually all cases have occurred in Sichuan Province, where infections with *Streptococcus suis* have been detected in pigs in a concurrent outbreak. The province has one of the largest pig populations in China.

Investigation and containment of the outbreak have been given high priority by Chinese authorities. The country's ministries of health and agriculture are working in close collaboration, and WHO and FAO are being promptly informed of new developments.

Investigations conducted by Chinese epidemiologists indicate that the first human cases occurred at the end of June in Ziyang City, Sichuan Province. Cases have since been reported in 11 prefectures in Sichuan Province. Most cases reported have occurred in adult male farmers. Information reported to WHO suggests that close contact with diseased or dead pigs is the principal source of human infection.

Symptoms reported by local clinicians include high fever, malaise, nausea, and vomiting, followed by meningitis, subcutaneous haemorrhage, toxic shock, and coma in severe cases. The incubation period is short and disease progression is rapid.

Local experts are conducting active searches for further cases. To date, Chinese authorities say they have found no evidence of human-to-human transmission.

The outbreak in humans has some unusual features and is being closely followed by WHO. Diagnostic testing to further characterise the causative agent is recommended as an essential part of ongoing efforts to understand this outbreak, ensure its rapid containment, and prevent further deaths.

Pro-MED-mail

This material has been summarised from information provided by ProMED-mail (www.promedmail.org).

Poliomyelitis – Yemen

Source: Adnkronos International *22 August 2005 [edited]*

Health officials in Yemen say they have detected around 50 new cases of polio, on top of the 369 cases already confirmed in the country.

The new cases were discovered in three provinces where parents refused to let their children be vaccinated because of a lack of confidence and lack of awareness about how dangerous the disease is. Many families are sceptical because the outbreak of the potentially fatal disease began following a routine national immunisation programme.

The Ministry of Public Health and Population launched a 2-day campaign, sending out 34,580 health workers to go door-to-door, immunising some 4 million children aged under five years. Each house where children have been immunised will be marked, and special ink will be put on the fingernails of every immunised child.

The World Health Organization declared Yemen polio-free in 1996. Only six countries in the world are considered polio-endemic: Nigeria, India, Pakistan, Niger, Afghanistan and Egypt. It is thought the disease may have spread from Nigeria to 12 other African countries, including Sudan.

Meanwhile, Saudi Arabia is putting stricter measures into place ahead of the Hajj pilgrimage in January, to stop the disease spreading there. All children under 15 travelling to the holy city of Mecca

from the 19 countries considered most 'at risk' will be immunised on the spot. Every year more than 2 million pilgrims travel to Saudi Arabia to take part in the pilgrimage, which all fit, healthy Muslims are expected to undertake at least once in their lives.

More than 1,050 children are reported to have been paralysed by polio so far in 2005 alone, mostly in Nigeria. According to the most recent information available on the polio eradication website, 453 of the 1,053 cases reported as of 16 August 2005 were from Nigeria, representing 43 per cent of cases reported this year.

Creutzfeldt-Jakob disease (new variant) update 2005

United Kingdom: vCJD monthly statistics

Source: UK Department of Health, Monthly Creutzfeldt-Jakob Disease Statistics, Press release number 2005/0310, 5 September 2005 [edited]

The Department of Health issued the latest information about the number of known cases of Creutzfeldt-Jakob disease. This includes cases of variant Creutzfeldt-Jakob disease. The position is as follows:

Summary of vCJD cases – deaths

Deaths from definite vCJD (confirmed): 108

Deaths from probable vCJD
(without neuropathological confirmation): 42

Deaths from probable vCJD (neuropathological confirmation pending): 0

Number of deaths from definite or probable vCJD (as above): 150

Summary of vCJD cases – alive

Number of probable vCJD cases still alive: 7

Total number of definite or probable vCJD cases (dead and alive): 157

Since the previous monthly statistics were released on 2 August 2005, the total number of deaths from definite or probable vCJD remains unchanged at 150. The number of probable vCJD cases still alive remains unchanged at seven cases. Therefore, the overall total number of definite or probable vCJD cases (dead and alive) remains unchanged at 157.

These data are consistent with the view that the vCJD outbreak in the United Kingdom (UK) is now in decline. The number of deaths due to definite or probable vCJD in the UK during the first eight months of 2005 remains at two. The number of deaths was 28 in the year 2000, followed by 20 in 2001, 17 in 2002, 18 in 2003, and 9 in 2004.

The number of definite or probable vCJD cases in the UK is now 157, followed by France with 13, Ireland 2, and Italy, the Netherlands, Portugal and Spain with single cases. Single cases have been confirmed also in Canada, Ireland, Japan and the USA, involving patients with periods of residence in the UK and who are presumed to have contracted infection in the UK. The Japanese case is exceptional in that the patient may have spent less than one month in the UK.

In addition, the US National Prion Disease Pathology Surveillance Centre confirmed a vCJD diagnosis by analysing a brain biopsy sample from a 33-year-old Saudi man admitted to a hospital in Saudi Arabia. Although detailed information on this patient was not available, he may have visited the United Kingdom, if at all, only for several days. Thus, the patient may have contracted the disease in Saudi Arabia after eating BSE-contaminated cattle products imported from the United Kingdom.

The occurrence of the first suspected human case of vCJD in Spain is not unexpected, since the risk of contracting vCJD appears to be linked to the amount of bovine meat consumed or extent of exposure to bovine products. According to the latest figure compiled by the European Union and the OIE, Spain ranks fifth in the world in terms of the number of BSE-affected cattle (532), after France with 946, Portugal with 949, Ireland with 1,470, and the UK with 184,138. However, in 2005, Spain, with 42 cases, ranks second after the UK with 126 cases, followed by Ireland with 29, Portugal with 17, and France with two cases of BSE.

Influenza viruses, drug resistance

Source: Reuters UK, 30 September 2005 [edited]

A strain of the H5N1 avian influenza virus that may unleash the next global flu pandemic is showing resistance to Tamiflu, the antiviral drug that countries around the world are now stockpiling to fend off the looming threat. Experts in Hong Kong said that the human H5N1 strain which surfaced in northern Viet Nam this year had proved to be resistant to Tamiflu, a powerful antiviral drug.

They urged drug manufacturers to make more effective versions of Relenza, another antiviral that is also known to be effective in battling the much-feared H5N1. Relenza is inhaled, whereas Tamiflu is taken orally. Viral resistance to Tamiflu is growing in Japan, where doctors prescribe the drug to fight common influenza.

Reports in *The Lancet* this month said that resistance to anti-influenza drugs was growing worldwide. In places such as China, drug resistance exceeded 70 per cent, suggesting that drugs like amantadine and rimantadine will probably no longer be effective for treatment or as a preventive in a pandemic outbreak of flu, the reports said.

CDI subject index, 2005

C

D

E

I

J

K

L

M

N

O

P

Q

R

S

T

V

W

Y

Z

CDI author index, 2005

CDI reviewers 2005

The *CDI* staff wish to thank the following reviewers for their valued assistance throughout the year.

Annette Broom, Avner Misrachi, Basil Donovan, Colin Masters, David Lyle, David Smith, Hazel Clothier, John Kaldor, John Mathews, Lance Sanders, Leslee Roberts, Michael Lindsay, Peter Markey, Peter Ryan, Reg Butler, Richard Russell, Robert Menzies, Robyn Hardyman, Scott Bowden, William Rawlinson.