



Department of Health

Government of Western Australia

**Gastrointestinal illness in Western Australia: Incidence,
notifications and outbreaks.**

Annual report of the WA OzFoodNet site, 2005

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1.0 Executive summary

In 2005, the overall number of notifications of gastrointestinal infections in WA remained very stable when compared to the mean of the previous four years. The crude notification rate for most of the enteric diseases also remained fairly stable, with the exception of Shiga toxin producing *E. coli* (STEC) infections, which showed a large increase in 2005. The increased detection was attributable to the introduction of a more sensitive test that detects STEC genes by polymerase chain reaction (PCR). The number of *Campylobacter* and *Salmonella* notifications also increased when compared to the previous two years, the latter partly attributable to a large *Salmonella* Oranienburg outbreak in the last quarter of the year.

The largest number of enteric infections reported continued to be *Campylobacter*, *Salmonella* and *Giardia* infections, which together constituted 90% of all notifiable enteric infections reported to the Department of Health in 2005. No significant change was observed in the epidemiology of the major enteric diseases in Western Australia in 2005. The largest burden of disease for most enteric pathogens was borne by young children, particularly in Aboriginal populations. There was no real disparity by sex for the enteric diseases except *Campylobacter*, for which there was a significantly higher notification rate for males. The northern parts of the State (Kimberley and Pilbara/Gascoyne) had the highest notification rates for most diseases geographically.

Salmonella Typhimurium phage type 135/135A was the most common serovar in WA in 2005, followed by *Salmonella* Oranienburg and *Salmonella* Enteritidis phage type 6A. With the exception of *S. Oranienburg* infections (which were directly related to an outbreak), these two serovars have predominated in WA in previous years. The largest increase in specific *Salmonella* serovars was observed with *Salmonella* Typhimurium phage type 12, *Salmonella* Corvallis and *Salmonella* Oranienburg.

During 2005, 37 outbreaks of gastrointestinal illness were reported to the Department of Health. The majority of these were in aged care facilities and appeared to be caused by norovirus. There were five foodborne outbreaks. For four of these outbreaks the responsible agent was not identified but illness was associated with consumption of food at particular venues. The fifth outbreak was caused by alfalfa sprouts contaminated with *Salmonella* Oranienburg, and resulted in 126 notified cases spread throughout Western Australia.

Cluster investigations were carried out when notification rates were above expected for *Salmonella* Typhimurium phage type 12, *Salmonella* Bovismorbificans, *Salmonella* Virchow and *Salmonella* Typhimurium phage type 44. These investigations did not identify a source of infection.

In 2005, the WA OzFoodNet site continued to participate in two national multi-centre case control studies on locally acquired *Salmonella* Enteritidis and STEC to identify risk factors for these infections.

2.0 Introduction

Western Australia is divided into two metropolitan – North and South - and seven non-metropolitan population health regions – Kimberley, Pilbara and Gascoyne, Midwest and Murchison, Coastal and Wheatbelt, Goldfields, SouthWest, and Great Southern (Figure 1). Prior to July 2005, the metropolitan region was divided into three population health regions – North, South and East. The three metropolitan health regions have been replaced by two Area Health Services, the North Metropolitan and South Metropolitan Area Health Services. The North Metropolitan Area Health Service is a combination of the old North and East metropolitan health regions.

Each region is administered by a Population Health Unit (PHU) responsible for public health activities, including communicable disease control. Disease control activities for the metropolitan area are administered by the Communicable Disease Control Directorate (CDCD) in collaboration with the two metropolitan population health units. The CDCD also maintains and coordinates the notifiable disease surveillance system and provides specialist clinical, public health and epidemiological advice to all PHUs. The West Australian notifiable diseases surveillance system relies on the mandatory reporting by doctors of the 17 notifiable enteric diseases. Prior to 2000, notifications were purely doctor-based. Laboratory reporting, although not mandatory in Western Australia, does occur informally. The inclusion of laboratory data from the beginning of 2000 introduces a bias to both West Australian and national data when comparing data with previous years – notification data now reflect actual incidence more closely.

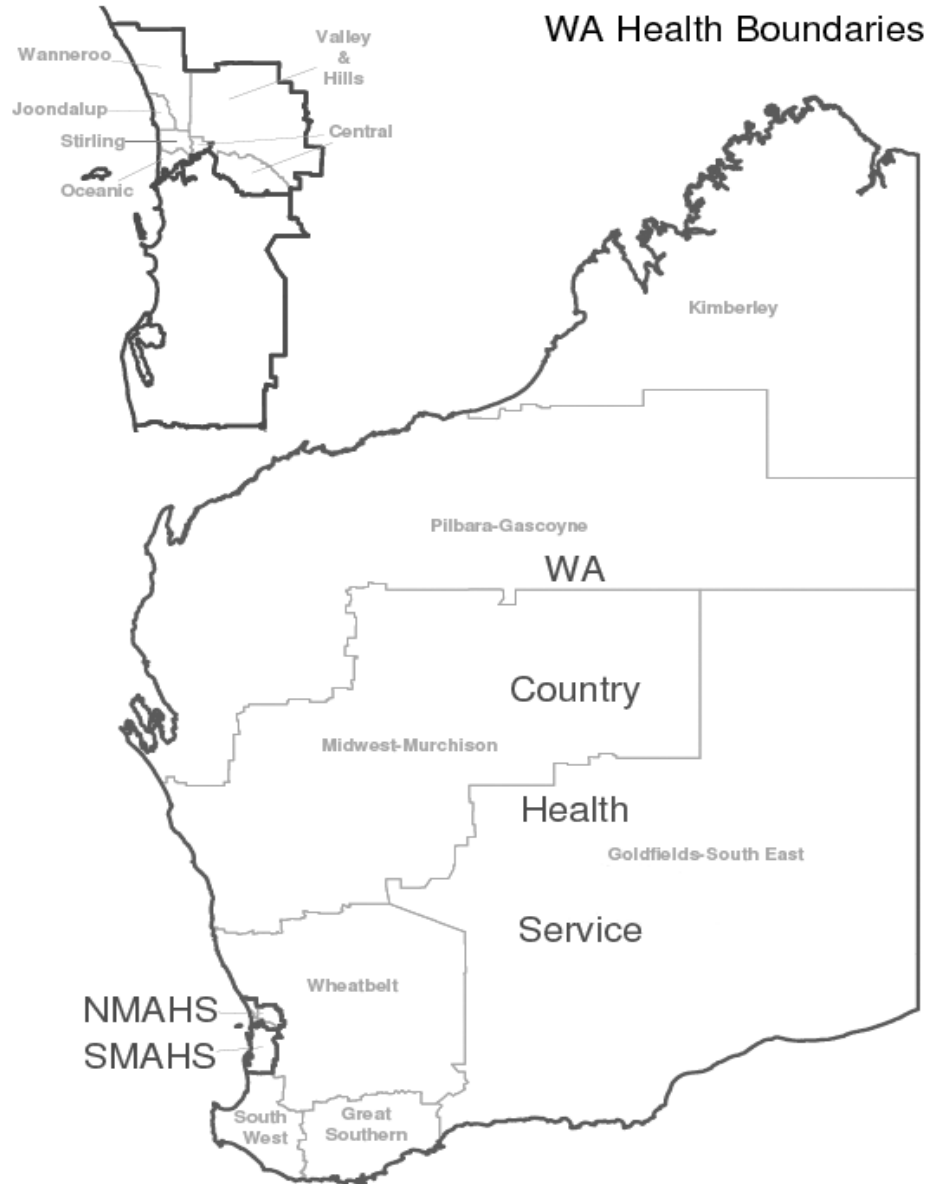
The OzFoodNet site in Western Australia encompasses residents within the whole state - total population 2.0 million. The mission of OzFoodNet is to enhance surveillance of foodborne illness and to conduct applied research into associated risk factors. A full-time epidemiologist coordinates activities in Western Australia, which are overseen by a coordinating national epidemiologist. Collaboration between states is facilitated by monthly teleconferences, face-to-face meetings three times a year, and through the informal network. This network also includes communication and consultation with Food Standards Australia New Zealand (FSANZ), the Commonwealth Department of Health and Ageing (DOHA), the National Centre for Epidemiology and Population Health (NCEPH), the Communicable Diseases Network of Australia (CDNA) and the Public Health Laboratory Network (PHLN).

The primary objectives of OzFoodNet nationally are to:

- Determine the frequency and burden of foodborne disease in Australia,
- Identify the causes and contributing factors to foodborne disease in Australia;
- Provide epidemiological information to inform prevention efforts, and
- Describe the epidemiology of new and emerging foodborne pathogens.

On a local level, the OzFoodNet epidemiologist regularly liaises with staff at the Food Safety Branch of the Environmental Health Directorate of the Department of Health, the Enteric, Food Hygiene, and Diagnostic and Molecular Epidemiology laboratories at PathWest Laboratory Medicine (formerly the West Australian Centre for Pathology and Medical Research, PathCentre), and regional and metropolitan population health units.

This report summarises the surveillance and research activities of the West Australian OzFoodNet sentinel site for the year 2005.



NMAHS: North Metropolitan Area Health Service
SMAHS: South Metropolitan Area Health Service

Figure 1: Map showing location of population health regions in Western Australia.

3.0 Incidence of Foodborne Disease

3.1 Methods

3.1.1 Population under surveillance

Estimated resident population figures for Western Australia for calculation of notification and age-specific rates were obtained from the Rates Calculator version 9.1.2 designed by Dr. Jim Codde at the Health Information Centre of the Department of Health, Government of Western Australia. The Rates Calculator provides population estimates by age, sex, Aboriginality, year and area of residence, and is based on population figures calculated from the 2001 census. The population for WA in 2005 was 2,000,459 persons.

3.1.2 Data sources

3.1.2.1 Rates of notified infections

Data for Western Australia were obtained from cases notified to the Department of Health, Government of Western Australia, and maintained in the Western Australian Notifiable Infectious Diseases Database (WANIDD). Notifications received for salmonellosis, campylobacteriosis, listeriosis, shiga-toxin producing *E. coli* (STEC) infection, shigellosis, yersiniosis, typhoid fever, paratyphoid fever, cholera, Haemolytic Uraemic Syndrome (HUS), cryptosporidiosis, hepatitis A and E, giardiasis, amoebiasis, *Vibrio parahaemolyticus* infection and botulism were collated and analysed in Microsoft® Excel 2002. Data were analysed by date of receipt of notification.

Data on *Salmonella* serovars were obtained from PathWest Laboratory Medicine, the reference laboratory for *Salmonella* isolates in the State. Phage typing data were obtained from the Microbiological Diagnostic Unit (MDU), University of Melbourne, the Institute of Medical and Veterinary Science (IMVS) the National Enteric Pathogens Surveillance Scheme (NEPSS) and the Australian Salmonella Reference Laboratory (ASRL).

Cryptosporidiosis and infections due to Shiga-toxin producing *E. coli* became notifiable in January 2001, therefore data for years prior to 2001 are not available for these infections.

To compare current disease rates to historical totals, crude numbers and rates of notification for 2005 have been compared to the mean values for the previous four years. Where available, numbers and rates of notifications for specific sub-types of infecting organisms were compared to notifications for the previous year.

Age-specific rates were calculated for the major notified enteric diseases. This includes salmonellosis, campylobacteriosis, shigellosis, giardiasis, cryptosporidiosis and hepatitis A. Rates were calculated by region, sex and Aboriginality.

3.1.2.2 Gastrointestinal and foodborne disease outbreaks

Information was collected on gastrointestinal and foodborne disease outbreaks that were reported in WA during 2005. This report collates summary information about the setting where the outbreak occurred, the month the outbreak occurred, the aetiological agent, the number of persons affected, the type of investigation conducted, the level of evidence obtained and the food vehicle responsible (if identified).

3.1.3 Limitations of the data

The number of notifications and crude notification rates reported here do not represent illness from foodborne sources exclusively, and include illness acquired through non-foodborne routes (eg. through contaminated water, person-to-person contact and direct animal exposure).

As with other surveillance data, these data are limited to diagnosed and reported illnesses. The majority of foodborne illness is unreported and undiagnosed, and the rate of diseases reported here is an under-estimation of the true incidence of foodborne illness. This underestimation was calculated to range from a notification to disease ratio of 0.5 for severe illnesses to 0.07 for moderate illnesses (DOHA, 2005). Importantly, some of the most common enteric pathogens are not notifiable, particularly Norovirus. These organisms may be notified as the cause of outbreaks, but not individual cases of disease. Surveillance data are also inherently biased and require careful interpretation. These biases include certain population groups having a higher likelihood of being tested as well as variation in laboratory testing regimes between States and Territories resulting in different levels of detection of disease. In Western Australia, laboratory notification is not mandatory under legislation and although most laboratories do notify the Department of Health by agreement, laboratory notification data is incomplete. In addition to the above limitations, some of the numbers of disease reports are small, as are the underlying populations in some jurisdictions. This can make rates of notification unstable and meaningful interpretation difficult.

The recording of Aboriginality data is notoriously poor. In 2005, for the 17 notifiable enteric diseases these data were missing for approximately 41% of cases, a significant proportion of notified cases. The proportion of cases where indigenous status was unknown varied from region to region and by disease, ranging from 0% - 70%. Consequently, for this report, the rate of infection for specific diseases has been reported "unadjusted" for indigenous and non-indigenous cases, along with a total rate of infection.

3.2 Results

3.2.1 Rates of notified infections

In 2005, WA reported a total of 4415 notifications for 16 notifiable enteric diseases. This equated to a rate of 220.7 per 100 000 population. The rate for 2005 was a 13% increase from the rate of 194.8 per 100 000 for 2004, but very similar to the mean rate for 2001 to 2004 of 218.3 cases per 100 000 population. A summary of the number of cases and notification rates for each enteric disease is shown in Appendix 1.

3.2.2 Salmonellosis

Salmonella was the second most frequently notified cause of enteric infection in WA after *Campylobacter* (Appendix 1). In 2005 in WA there were 791 reported cases of *Salmonella* infection which equated to a rate of 39.5 cases per 100 000 population. This was a 26.6% increase from the rate of 31.2 cases per 100 000 reported in 2004, and 8.2% higher than the mean rate of 36.5 per 100 000 for the previous four years.

In 2005 higher numbers of *Salmonella* cases were reported in the late summer with lowest numbers through the late winter, which is a pattern that was observed in previous years (Figure 2). The number of notified *Salmonella* cases increased towards the end of the year which was also observed in previous years.

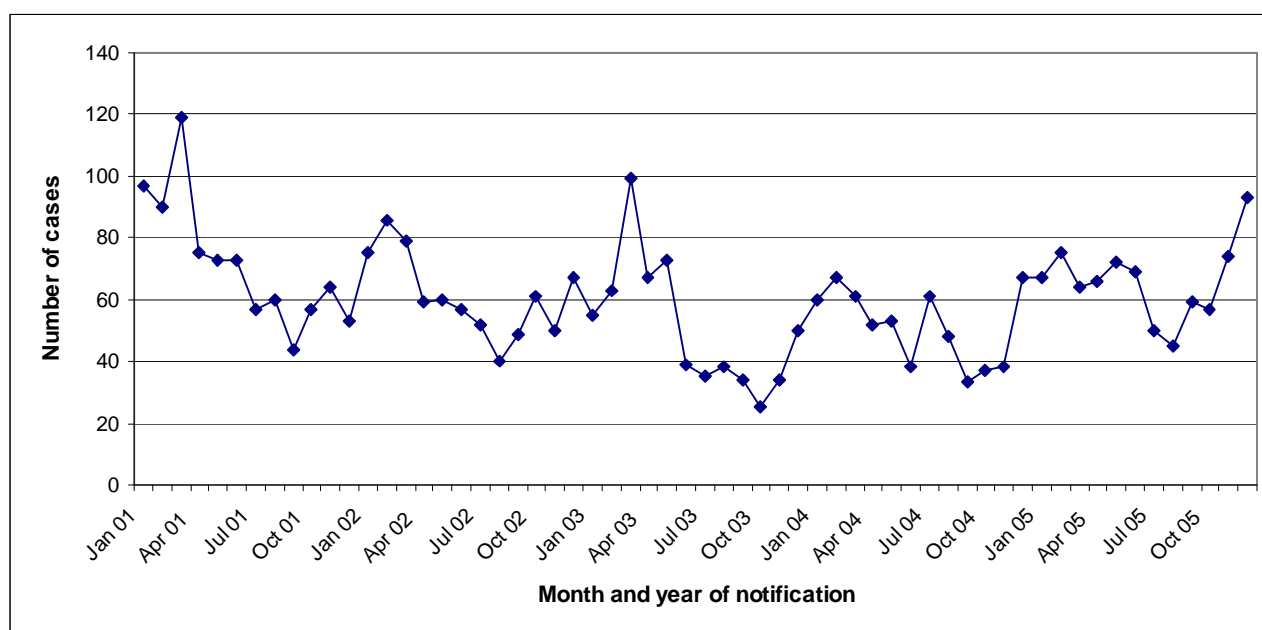


Figure 2: Number of cases of salmonellosis by month and year of notification, WA, 2001 –2005

The mean notification rate for all ages was greater for females (40.5 cases per 100,000) than males (33.6 cases per 100,000), but the difference was not significant ($z=1.36$, $p=0.087$) (Figure 3). The highest age-specific notification rate was for the 0-4 age group with 184 notifications per 100 000 population. The second highest notification rate was for the 20-24 year age group with 45 notifications per 100 000 population.

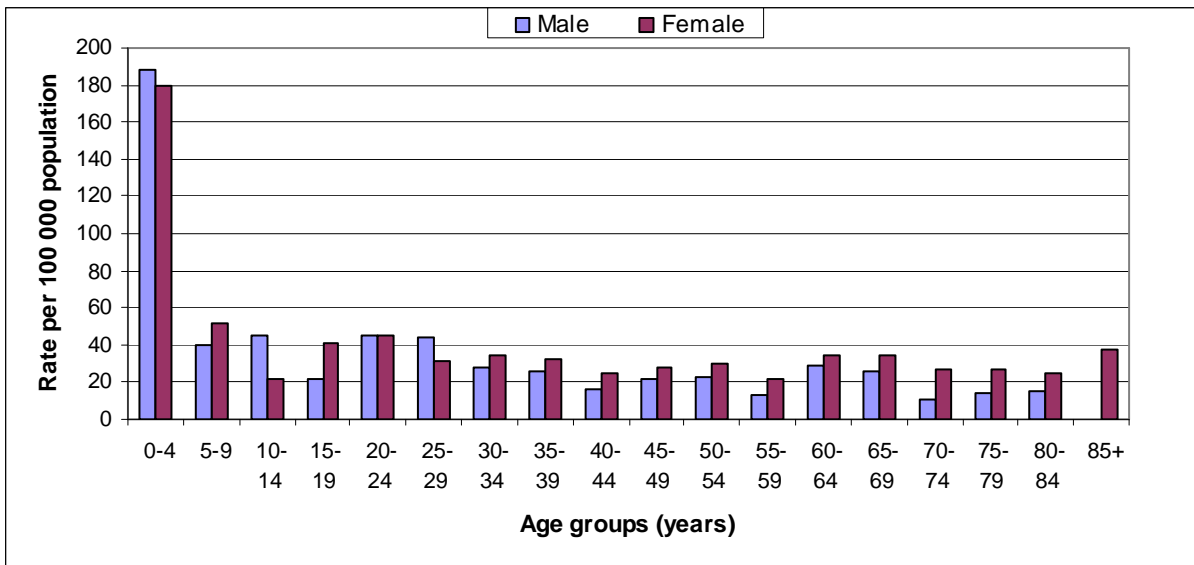


Figure 3: Age-specific notification rates of salmonellosis by sex, WA, 2005

Notification rates for *Salmonella* infection in Aboriginal people were greater than for non-Aboriginal people over all age groups, except for those age groups where no cases for Aboriginal people were notified (Figure 4). The mean notification rate over all age groups for Aboriginal people was 151.4 per 100 000 population and for non-Aboriginal people 21.0 per 100 000 population, which is a ratio of 7.2 times greater for Aboriginal versus non-Aboriginal people. For Aboriginal children in the 0-4 age group, the group with the highest incidence of infection, the notification rate was 814 cases per 100 000 population, which was 11.6 times greater than the notification rate for non-Aboriginal children in this age group of 70.1 per 100 000 population. The notification rate for Aboriginal people was also high for the 75-89 year age group but this represents only two cases from a small population, resulting in a high degree of uncertainty with these results.

The population health region in WA with the highest notification rate was the Kimberley region with 261.5 notifications per 100 000 population (Figure 5). Notification rates for both Aboriginal and non-Aboriginal people were highest in the Kimberley. The second highest notification rate was for the Pilbara and Gascoyne region with 113.8 notifications per 100 000 population.

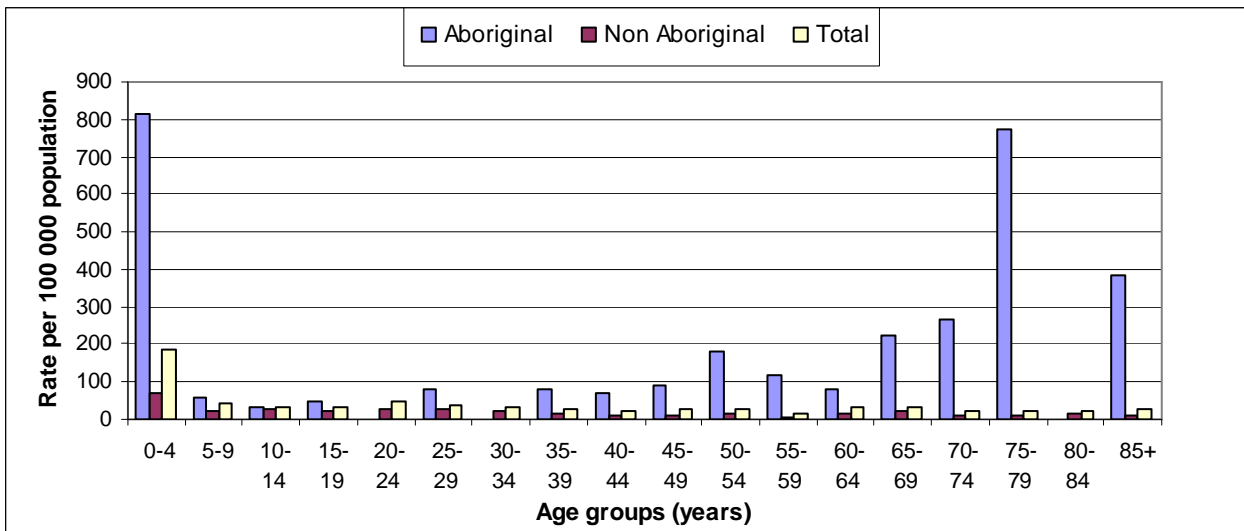


Figure 4: Age-specific notification rates of salmonellosis by Aboriginality, WA, 2005

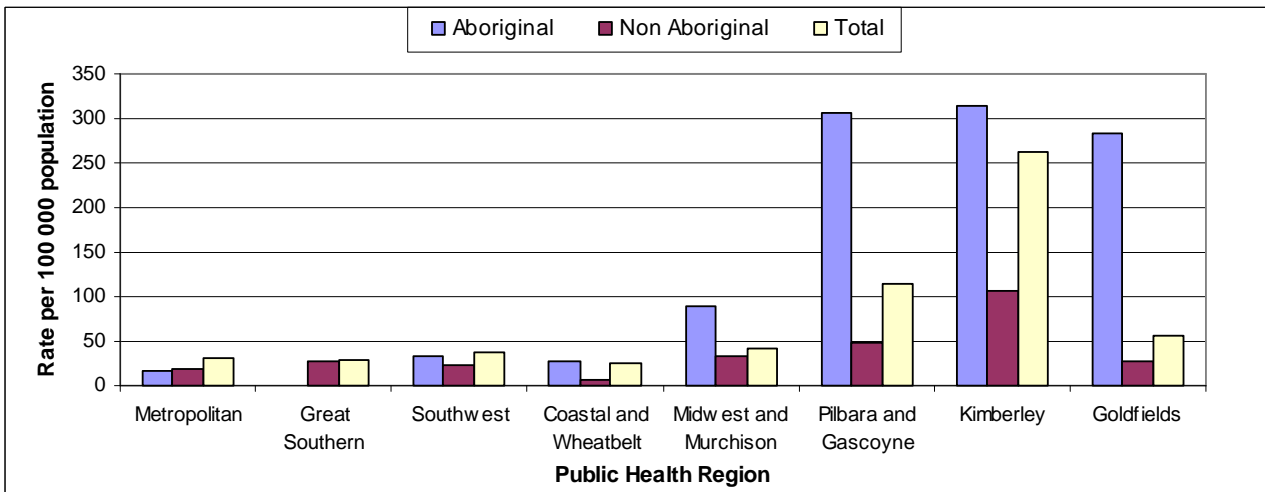


Figure 5: Crude notification rates of salmonellosis by population health region, WA, 2005

The ten most commonly isolated *Salmonella* serotypes or phage types in WA in 2005 are shown in Table 1. The most commonly reported phage type was *Salmonella* Typhimurium phage type 135/135A with 69 notified cases, which comprised 8.7% of *Salmonella* infections. This was slightly lower than for 2004 when 74 cases were notified, and a decrease from the 2001-2004 mean of 87.2 notified cases. The second most common serotype was *Salmonella* Oranienburg with 63 cases notified in 2005. This was a large increase above previous years with a mean of 11 cases over the years 2001 to 2004 and a ratio of 5.7 for cases in 2005 compared to the previous four year mean. There were 35 cases of *Salmonella* Enteritidis phage type 6A, making it the third most commonly notified *Salmonella* in WA in 2005. The fourth most common *Salmonella* was *Salmonella* Saintpaul with 32 cases. This was a decrease from the previous year when there were 46 notified cases and from the four yearly mean of 40.8 notified cases.

Table 1: Number and proportion of the top 10 serotypes or phage types of *Salmonella* infections notified in WA, 2005.

<i>Salmonella</i> type (serotype/phage type)	2005 n	Rank	Proportion % [‡]	Mean 2001- 2004	Ratio [§]
Typhimurium 135/135A	69	1	8.7	87.2	0.8
Oranienburg	63	2	8.0	11	5.7
Enteritidis 6A	35	3	4.4	12	2.9
Saintpaul	32	4	4.0	40.8	0.8
Muenchen	30	5	3.8	26.3	1.1
Chester	29	6	3.7	31.5	0.9
Typhimurium 12	28	7	3.5	0.3	112.0
Anatum	18	8	2.3	14.3	1.3
Corvallis	17	9	2.2	1.8	9.7
Senftenberg	15	10	1.9	14.5	1.0

[‡] Proportion of total *Salmonella* cases notified in 2005.

[§] Ratio of the number of reported cases in 2005 compared to the four year mean of 2001 – 2004.

3.2.3 *Campylobacteriosis*

Campylobacter was the most commonly reported enteric pathogen in WA in 2005 with 2422 notified cases, which equated to a rate of 121.1 cases per 100 000 population (Appendix 1). This was a 21% increase above the 2004 notification rate of 99.8 per 100 000, and an 8.3% increase above the mean rate from 2001 to 2004 of 112.0 per 100 000 population.

During the years 2001 to 2005 *Campylobacter* notifications showed a seasonal pattern and this is shown in Figure 6. *Campylobacter* notifications were higher in the summer months and lowest in the late spring or winter. In 2005 the greatest number of notifications was in October, November and December and the lowest number of notifications in April.

In WA in 2005 the mean notification rate for *Campylobacter* was higher for males than females, with a rate of 134.7 per 100 000 for males and 107.3 per 100 000 for females. There was a significant difference between notification rates for males and females (two tailed z test for proportions, $z=5.60$, $p.<0.001$) with a male: female notification ratio of 1.25:1. Male notification rates were greater in all but two age groups, the 20-24 age group and 85+ age group (Figure 7). The highest notification rate by age was for the 0-4 age group with a rate of 230.9 per 100 000, followed by the 25-29 age group with a notification rate of 171.4 per 100 000. This same pattern was observed in 2004.

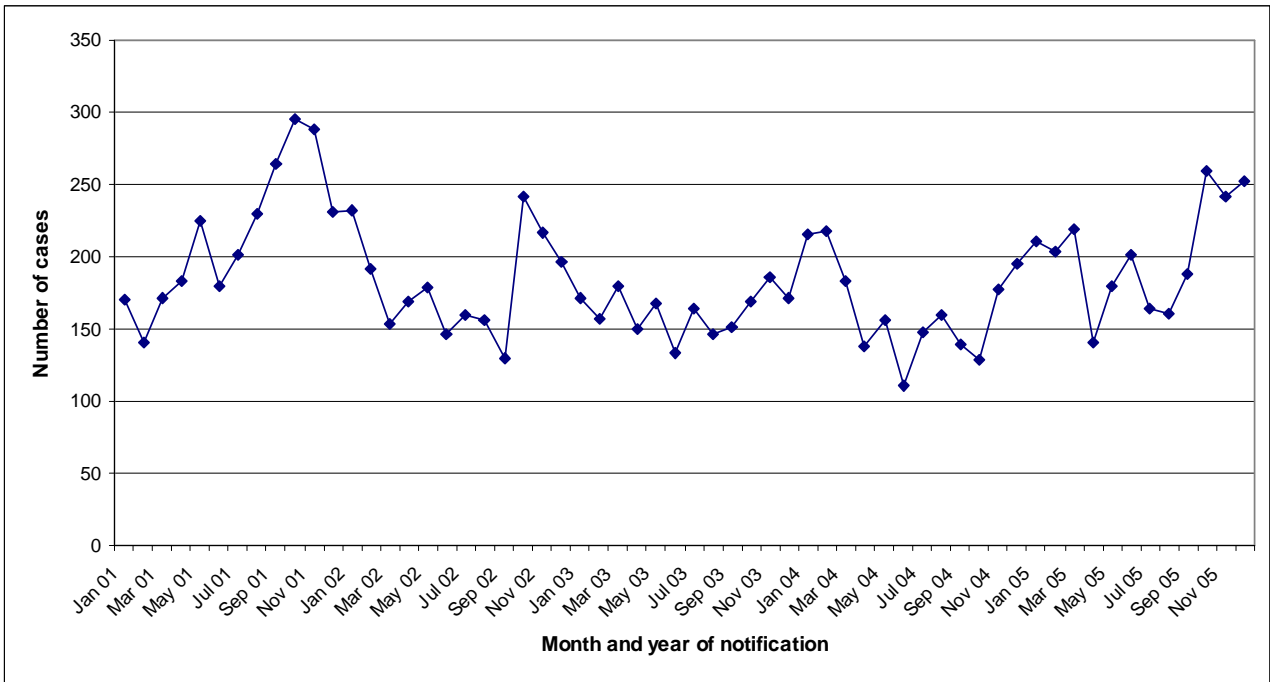


Figure 6: Number of cases of campylobacteriosis by month and year of notification, WA, 2001 – 2005

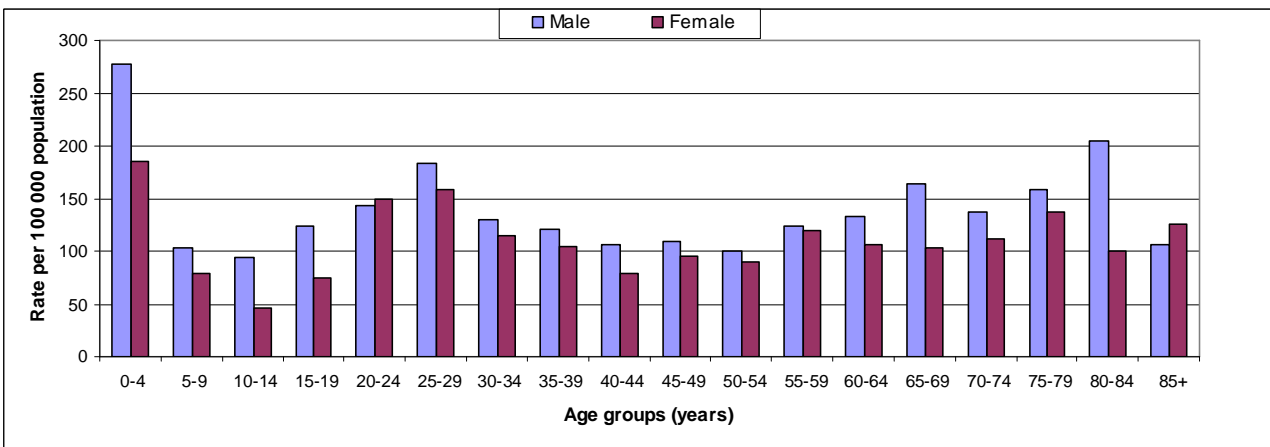


Figure 7: Age-specific notification rates of campylobacteriosis by sex, WA, 2005

Mean *Campylobacter* notification rates over all age groups for Aboriginal and non-Aboriginal people were similar in 2005. The mean rate for Aboriginal people was 67.0 per 100 000 population and for non-Aboriginal people 61.1 per 100 000 population. For the age group with the highest notification rates, the 0-4 age group, the rate for Aboriginal children was six fold greater than the rate for non-Aboriginal children (632.4 per 100 000 vs 107.8 per 100 000) (Figure 8).

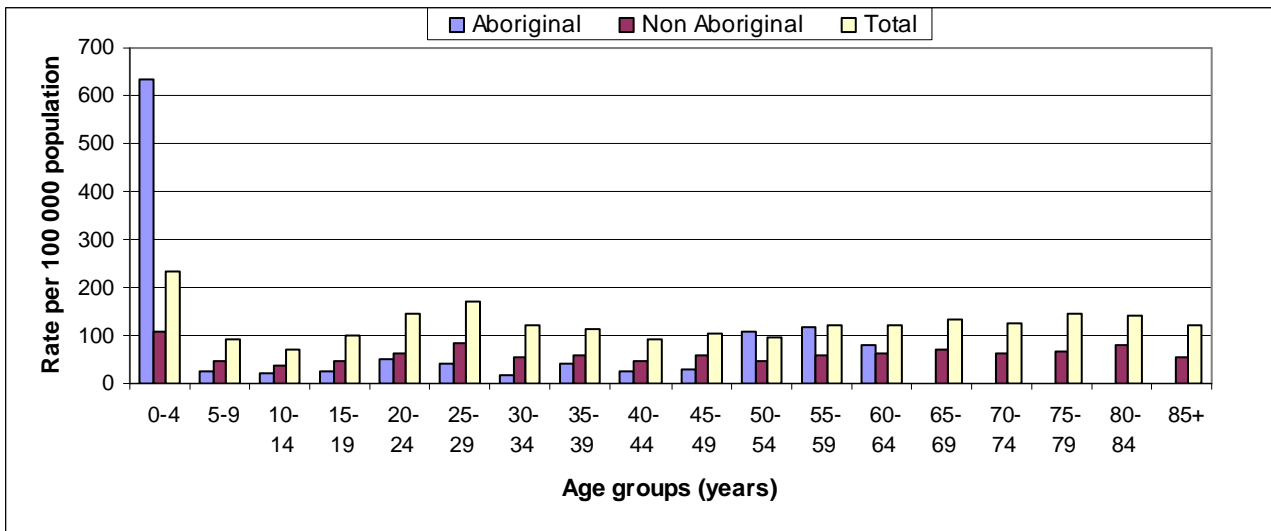


Figure 8: Age-specific notification rates of campylobacteriosis by Aboriginality, WA, 2005

Campylobacter notification rates were not markedly different between the different population health regions (Figure 9). Notification rates varied between 181.7 per 100 000 for the Southwest region and 75.5 per 100 000 for the Midwest and Murchison. For Aboriginal people the region with the highest rate of notifications was the Pilbara and Gascoyne with a rate of 203.0 per 100 000. There were no *Campylobacter* notifications for Aboriginal people in the Southwest region.

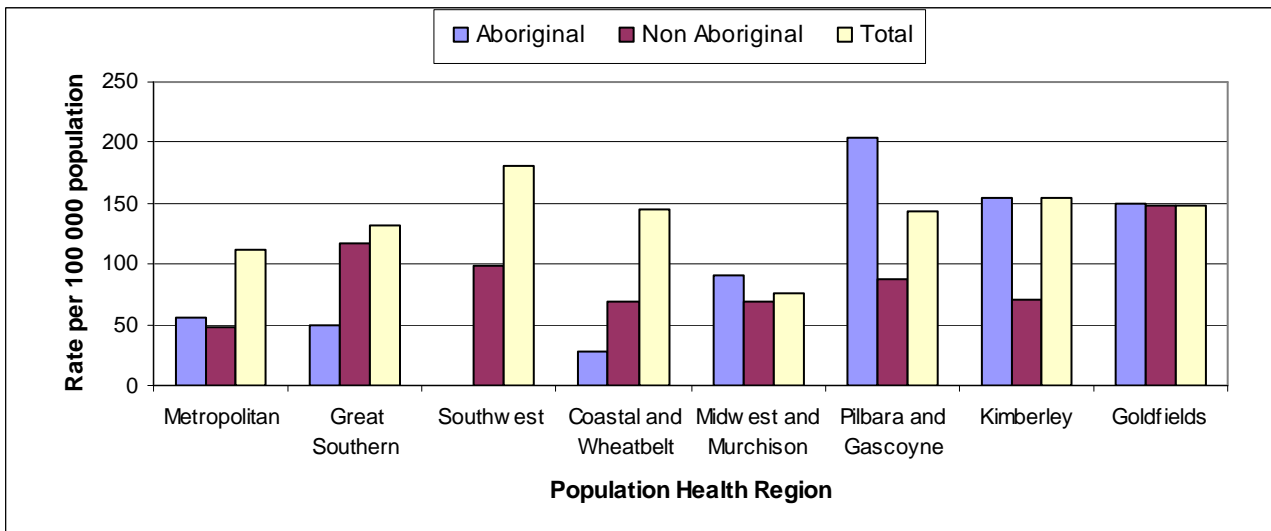


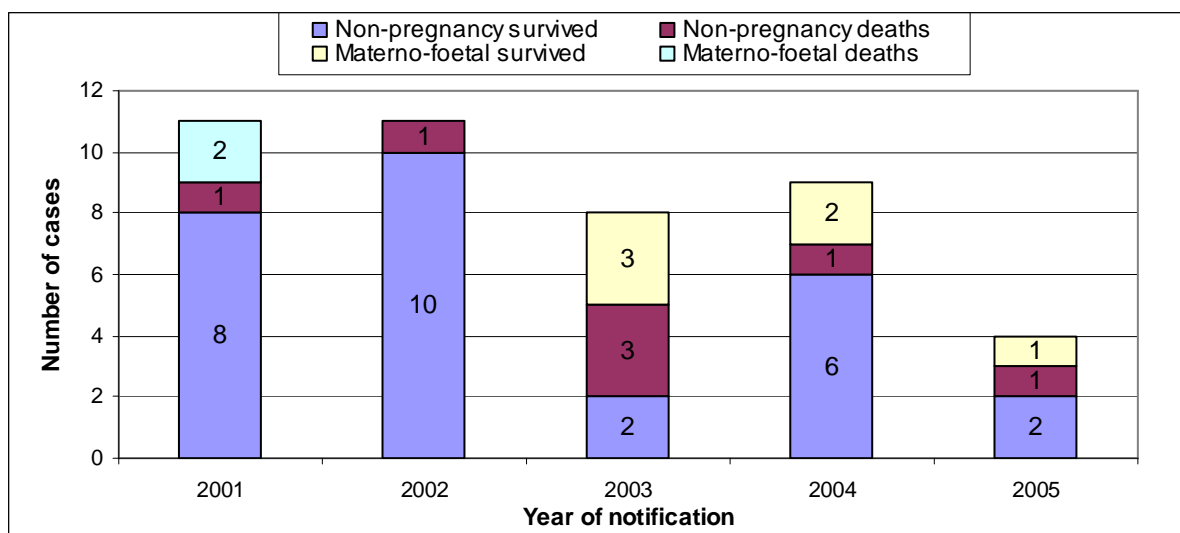
Figure 9: Crude notification rates of campylobacteriosis by population health region, WA, 2005

3.2.4 Listeriosis

In 2005 there were four reported cases of listeriosis (Figure 10). This was lower than the four yearly mean of 10 cases for the years 2001 to 2004.

In 2005 there was one pregnancy related case. The infection was diagnosed from the blood culture of the mother, who was 35 weeks pregnant with twins. Twins were delivered two days after hospital admission. Blood and gastric aspirate samples were negative for both of the twins. Mother and twins survived.

The other infections were for three males aged 66, 82 and 83 years. All the cases had a history of immunosuppressive illnesses. Amongst these three cases there was one death and one of the infections appeared to be acquired in hospital.



Numbers in the bars indicate number of cases

Figure 10: Notifications of listeriosis showing non-pregnancy related infections and deaths and materno-foetal infections and deaths, WA, 2001 to 2005

3.2.5 Shigellosis

There were 151 notified cases of shigellosis in WA in 2005, with a notification rate of 7.5 cases per 100 000 population (Appendix 1). This was greater than the mean notification rate for the previous four years of 5.6 per 100 000 population.

There did not appear to be a seasonal pattern for *Shigella* notifications (Figure 11). In 2005 the greatest number of notified cases was for the month of March and the lowest number for July, but in previous years highest and lowest numbers of notifications were at other times of the year.

The majority of isolates (54%) were *Shigella flexneri* (n=84), of which *Shigella flexneri* 4 was the most common subtype (n=33, 22%), followed by *Shigella flexneri* 2A (n=24, 16%). *Shigella sonnei* constituted 42% of isolates (n=63), with the majority of these (n=43) being classified as *Shigella sonnei* Biotype A.

The *Shigella* notification rate in WA in 2005 was slightly higher for females than for males (8.0 per 100 000 population vs 7.1 per 100 000 population) but this was not significant (z=0.727, p=0.236). The 0-4 year age

group had the highest notification rate with 33 per 100 000 for males and 25 per 100 000 for females (Figure 12).

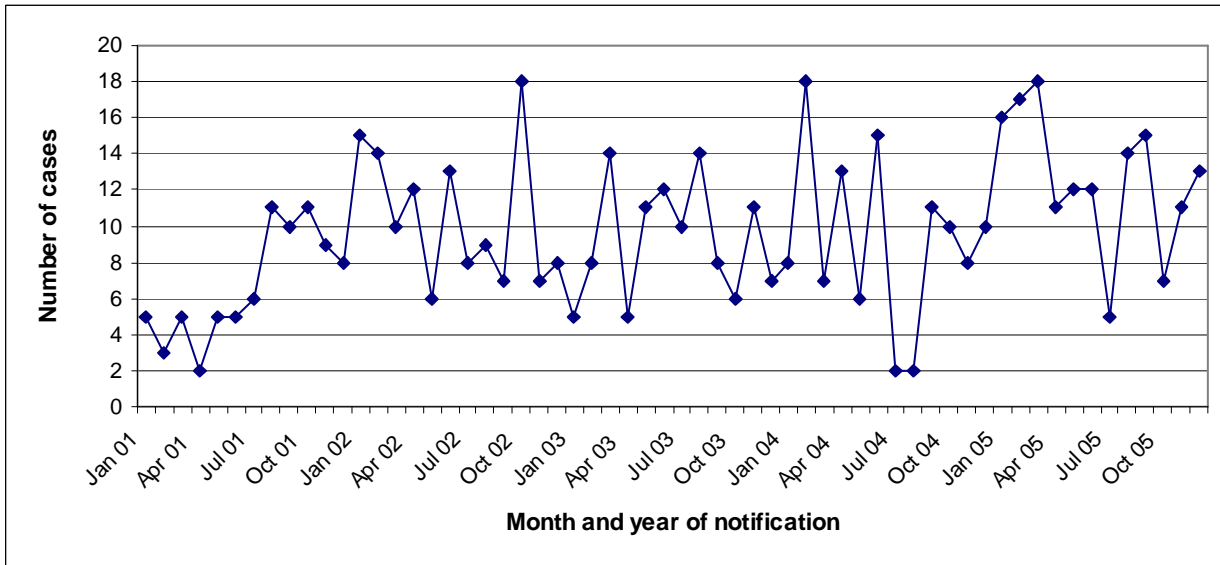


Figure 11: Number of cases of shigellosis by month and year of notification, WA, 2001 – 2005

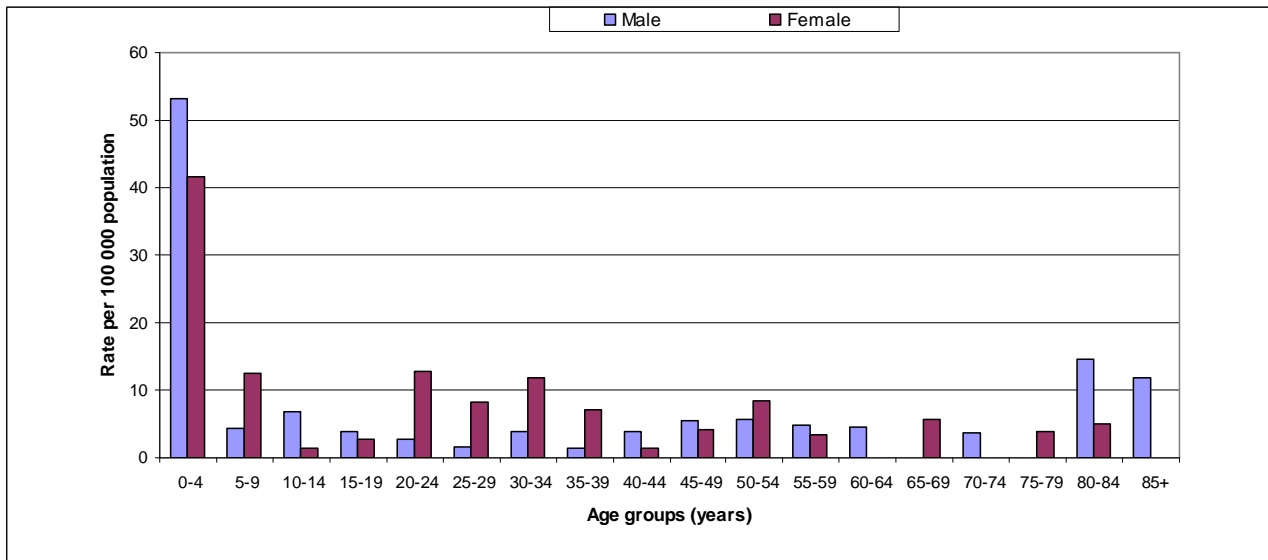


Figure 12: Age-specific notification rates of shigellosis by sex WA, 2005

The notification rate for *Shigella* in Aboriginal people was 103.7 per 100 000 population (Figure 13). This was 64.4 times greater than the notification rate for non-Aboriginal people, which was 1.6 per 100 000 population. For the population as a whole the highest notification rate was for the 0-4 age group with a notification rate of 47.5 per 100 000 population. For this age group the notification rate for Aboriginal children was 474.3 cases per 100 000 population. Rates were also high for Aboriginal people in the 75-79,

80-84 and 85+ age groups but these represented only one case in each group, so are associated with a high degree of uncertainty.

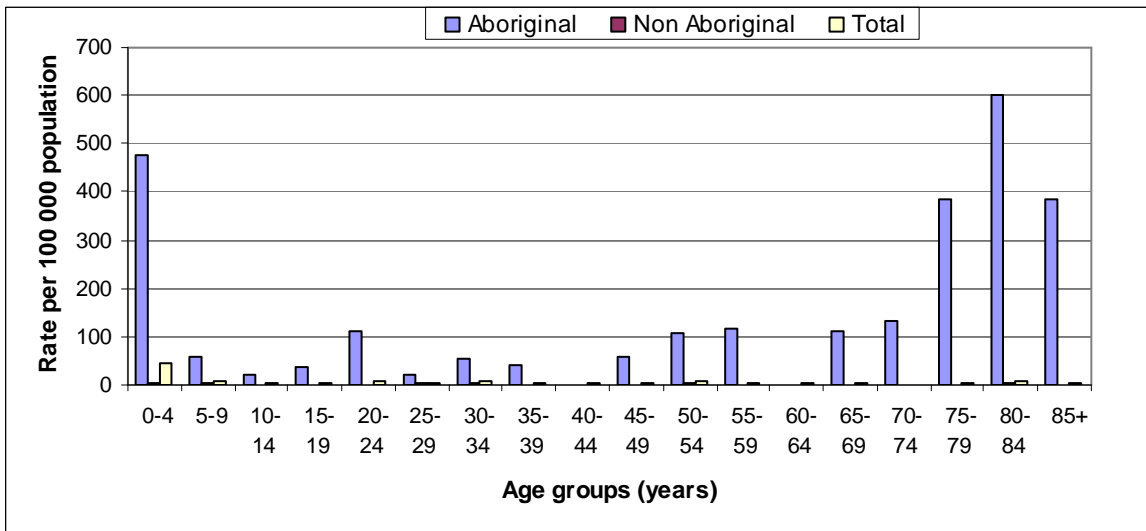


Figure 13: Age-specific notification rates of shigellosis by Aboriginality, WA, 2005

Shigella notification rates varied greatly between the different population health regions (Figure 14). The region with the highest notification rate for *Shigella* was the Kimberley region with a rate of 106 cases per 100 000 population. This contrasts with the Metropolitan region which had a rate of 2.7 cases per 100 000 population. Notification rates were higher for Aboriginal than non-Aboriginal people in all regions. The region with the highest notification rate for Aboriginal people was the Goldfields. In this region the notification rate for Aboriginal people was 216 per 100 000 population and for non-Aboriginal people 6.3 per 100 000 population.

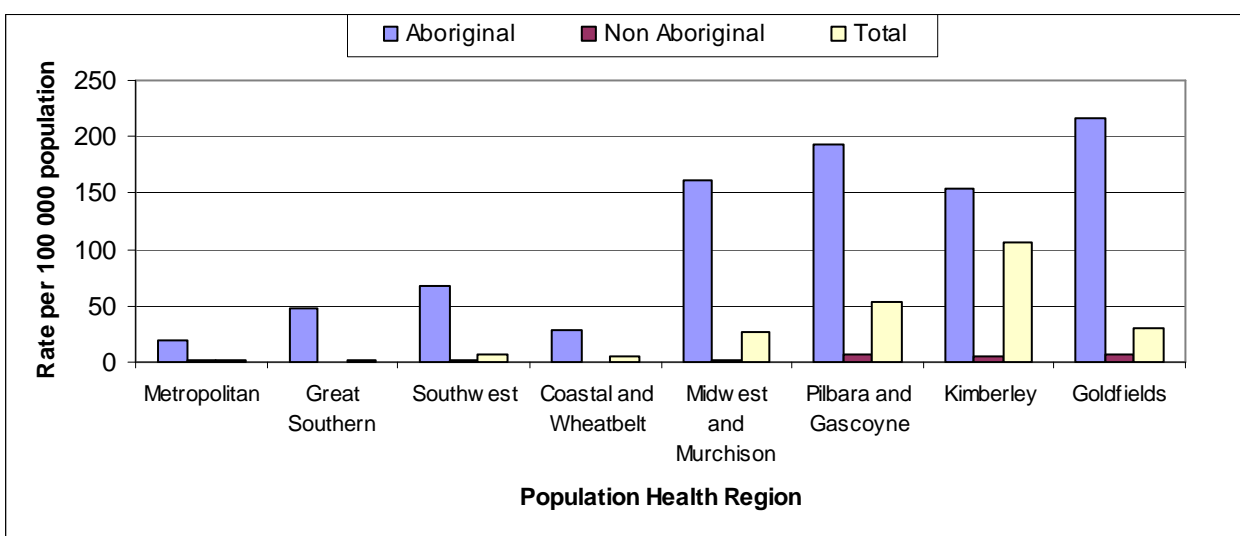


Figure 14: Crude notification rates of shigellosis by population health region, WA, 2005

3.2.6 *Yersiniosis*

Two cases of *Yersinia enterocolitica* were notified in 2005 in an 86 year female and a one year old male. Both cases were non Aboriginal and from the metropolitan region.

3.2.7 *Typhoid and paratyphoid fever*

There were seven cases of typhoid fever and four cases of paratyphoid fever in 2005 (Table 2). All of the paratyphoid cases and 10 of 11 typhoid cases were acquired overseas. The sole locally acquired case acquired her infection from a close family member who was a long term asymptomatic carrier and had had an infection several years earlier in India.

Table 2: Travel status for typhoid and paratyphoid cases, WA, 2005

Organism	Country	Number of cases	Phage type
Salmonella Paratyphi A	Bangladesh	1	13
	Cambodia	1	6
	India	1	2
	Vietnam	1	2
Salmonella Typhi	Australia	1	UNTY
	Bangladesh	1	E7
	China	1	D2
	Guinea	1	A
	India	1	UNTY
	Indonesia	1	A
	Tanzania	1	A
	Total		11

3.2.8 *Cholera*

There was one case of *Vibrio cholerae* serotype O1 Ogawa infection in a 49 year old female who had travelled to Bali, Indonesia in the two weeks prior to the onset of her infection. The isolate was positive for *ctx A* gene by PCR (i.e. a toxin producer) conducted at the MDU, University of Melbourne.

3.2.9 *Shiga toxin producing E. coli infections (STEC)*

Twelve cases of STEC were notified in 2005, resulting in a crude notification rate of 0.6 per 100,000 population. The notification rate was a six fold increase on the mean rate for the previous four years, and arose as a result of an enhanced screening trial at PathWest using a more sensitive PCR test to detect shiga toxin genes, instead of the conventional faecal culture. The cases ranged from 3 – 79 years of age. Two isolates detected by culture methods were able to be typed as serogroup O157:H-, and one sample was serotyped as O111 by PCR. All other samples were serotype non O157, non O111 by PCR. A public health follow-up was conducted with seven of the 10 cases. Two cases were not followed up as they were tested retrospectively and notification of the positive result was so delayed as to make follow-up of little value, and

one case refused to be interviewed. Of the 12 cases reported, five cases had either travelled overseas, were overseas visitors to WA, or immigrants / refugees. Another six cases either lived on rural properties, spent time on hobby farms or came from Aboriginal communities. No risk factors were identified in one case. Exposure to farm mammals, particularly cattle, and their faeces are known risk factors for infection with toxigenic *E. coli* (Parry et al. 1998).

3.2.10 Haemolytic uraemic syndrome (HUS)

There was one case reported in a one year old male who developed HUS secondary to an invasive lower respiratory tract *Streptococcus pneumoniae* infection.

3.2.11 Giardiasis

There were 747 cases of giardiasis notified in WA in 2005 which equated to a notification rate of 37.3 per 100 000 population (Appendix 1). This was lower than notification rates for the previous four years, which had a four year mean notification rate of 47.0 per 100 000.

There was no clear seasonal pattern in monthly *Giardia* notifications over the five years 2001 to 2005 (Figure 15).

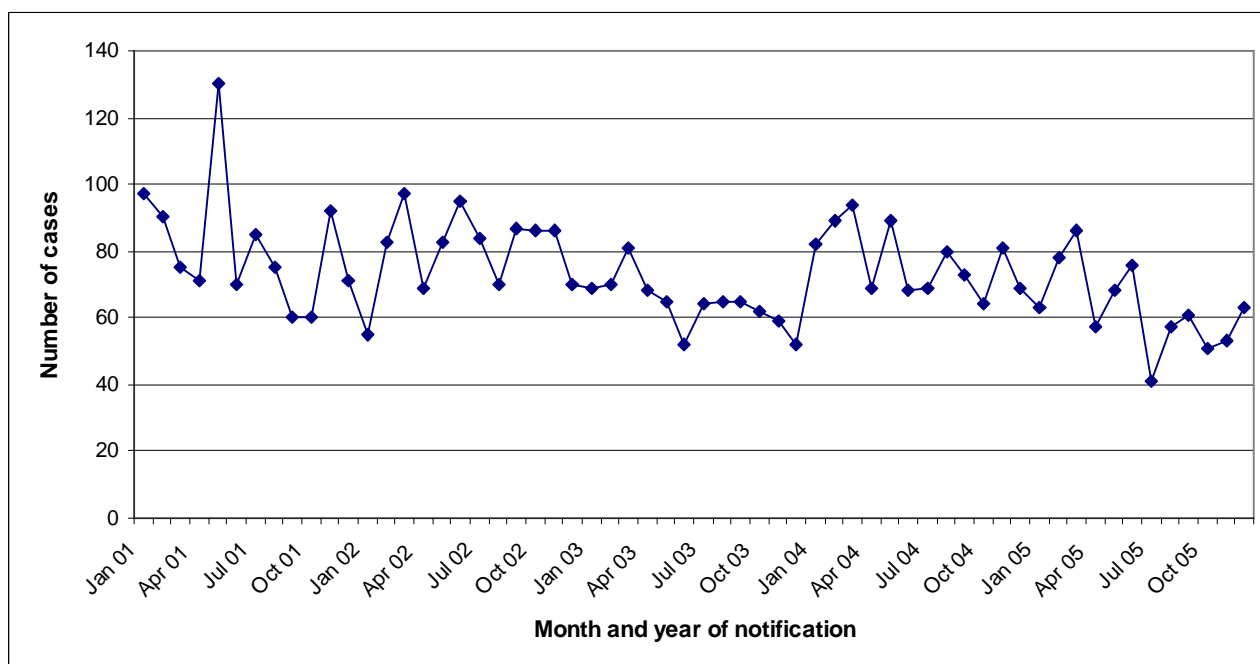


Figure 15: Number of cases of giardiasis by month and year of notification, WA, 2001 – 2005

Overall *Giardia* notification rates for males (38.5 per 100 population) and females (36.1 per 100 000 population) were similar in 2005, with no significant difference between them ($z=0.305$, $p=0.195$). The age group with the highest notification rates was the 0-4 age group with notification rates of 275.8 per 100 000 for males and 252.6 for females (Figure 16).

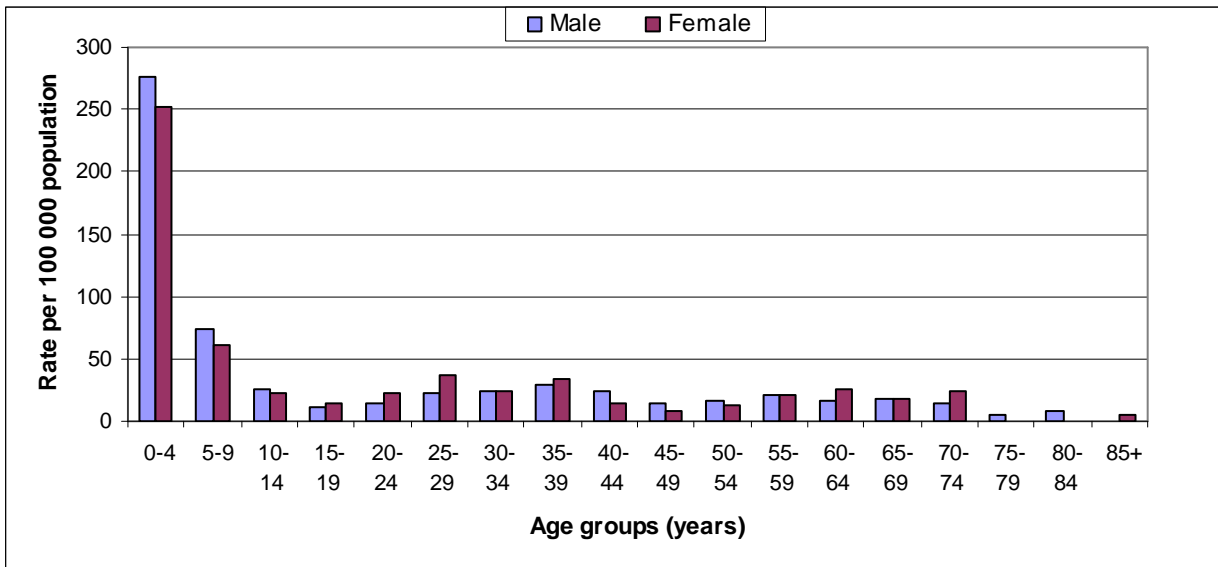


Figure 16: Age-specific notification rates of giardiasis by sex, WA, 2005

The overall notification rate for *Giardia* for Aboriginal people was 238.3 per 100 000. This was 14 times greater than the notification rate for non-Aboriginal people of 16.9 per 100 000. The highest notification rate for Aboriginal people was for the 0-4 age group with a notification rate of 1727 per 100 000 population (Figure 17). This was 22 times greater than the notification rate of 78.0 per 100 000 for non Aboriginal children in this age group.

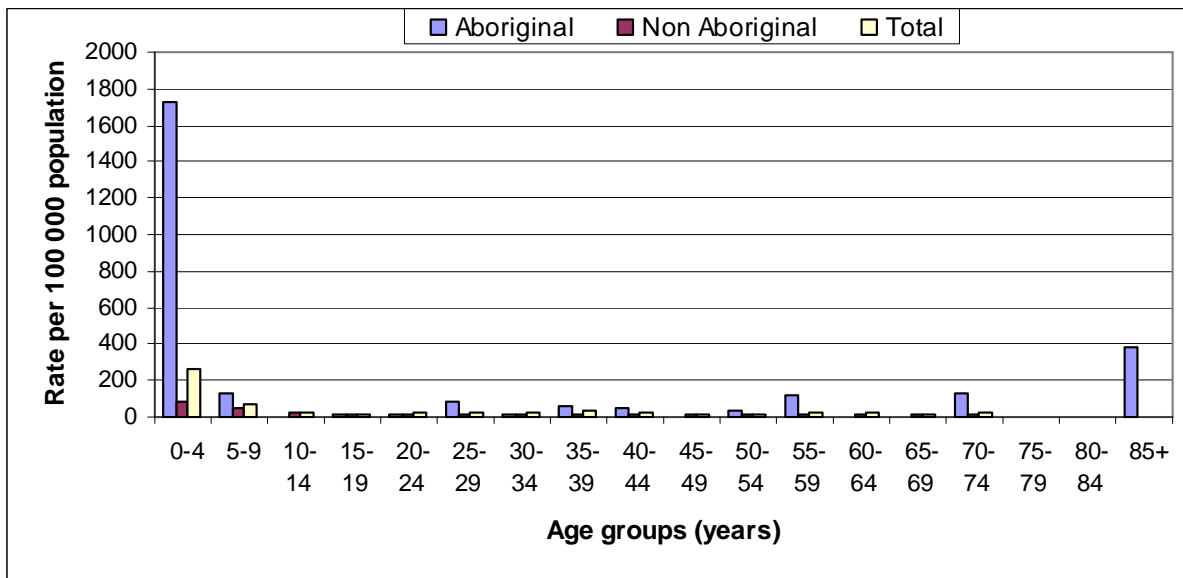


Figure 17: Age specific notification rates of giardiasis by Aboriginality, WA, 2005

There was a large variation in *Giardia* notification rates between the population health regions in 2005 (Figure 21). The region with the highest notification rates was the Kimberley with a total notification rate of 384 cases per 100 000 population. The region with the lowest *Giardia* notification rate was the Great Southern with a rate of 23.8 cases per 100 000 population. Notification rates for Aboriginal people were higher than for non-Aboriginal people over all population health regions with the exception of the Great Southern, where there were no notifications for Aboriginal people.

3.2.12 *Cryptosporidiosis*

In 2005 in WA there were 189 notified cases of cryptosporidiosis (Appendix 1). This equated to a rate of 9.4 per 100 000 population which was a 35% increase on the rate for 2004 of 6.1 cases per 100 000 population. The rate for 2005 was lower than the mean rate from 2001-2004 of 12.3 cases per 100 000 population.

Cryptosporidiosis notifications in WA showed a seasonal pattern with the highest number of cases in late summer/autumn and lower numbers of cases through the winter and spring. In 2005 the highest number of cases was notified in May with 25 cases (Figure 18). The lowest number of cases was in November with 6 cases. The large increase in case numbers in 2003 was discussed in the 2003 OzFoodNet WA Annual Report (Sarna 2003)

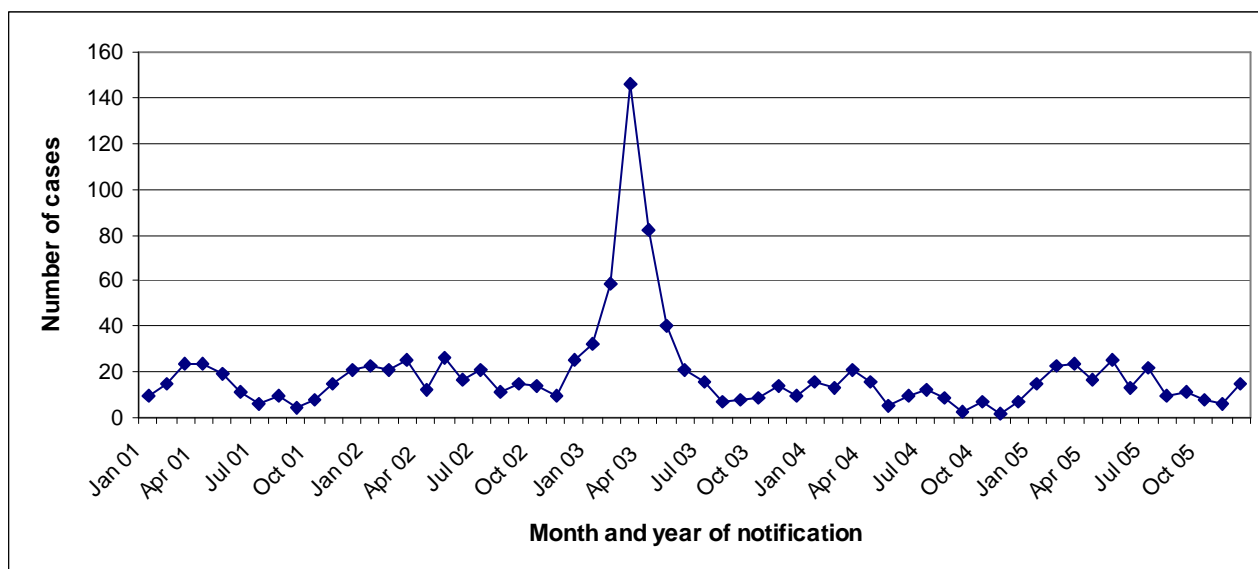


Figure 18: Number of cases of cryptosporidiosis by month and year of notification, WA, 2001 – 2005

In WA in 2005 the overall cryptosporidiosis notification rates were similar for males and females, with no significant difference between them ($z=0.364$, $p=0.136$). The notification rate for males was 10.2 per 100 000 and for females 8.7 per 100 000 (Figure 19). The age group with the highest notification rate was the 0-4 age group, with a notification rate of 106 per 100 000 for males and 96.4 for females.

The overall notification rate for cryptosporidiosis in Aboriginal people was 116 cases per 100 000 population, which was 37 times greater than the rate of 3.1 per 100 000 population for non-Aboriginal people (Figure 20). The highest notification rate was for Aboriginal children in the 0-4 age group, with a rate of 972 cases per 100 000 population. This was 101 times greater than the notification rate for non-Aboriginal children in the 0-4 age group of 16.6 cases per 100 000 population.

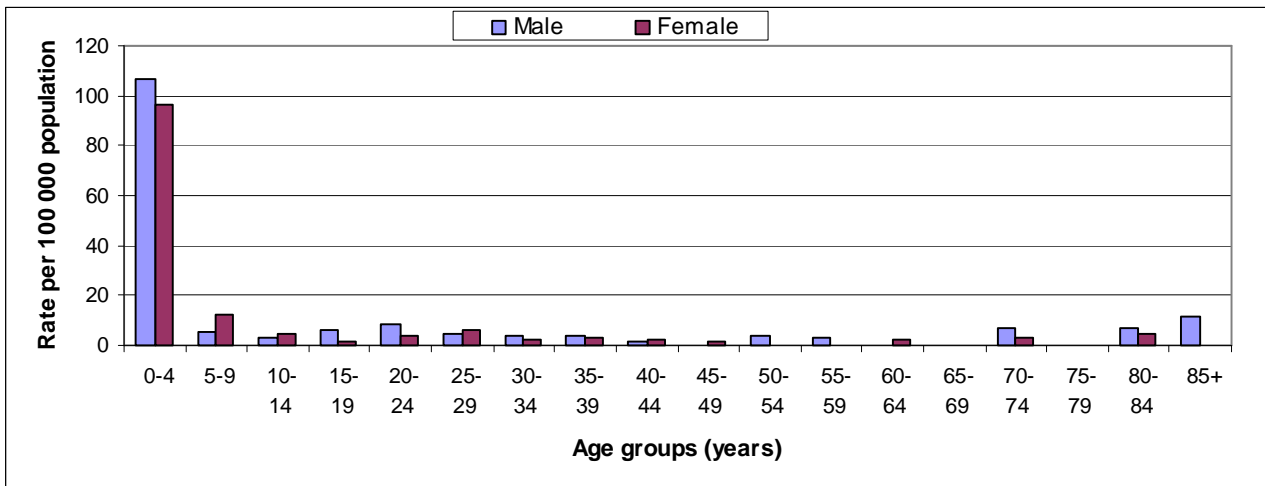


Figure 19: Age-specific notification rates of cryptosporidiosis by sex, WA, 2005

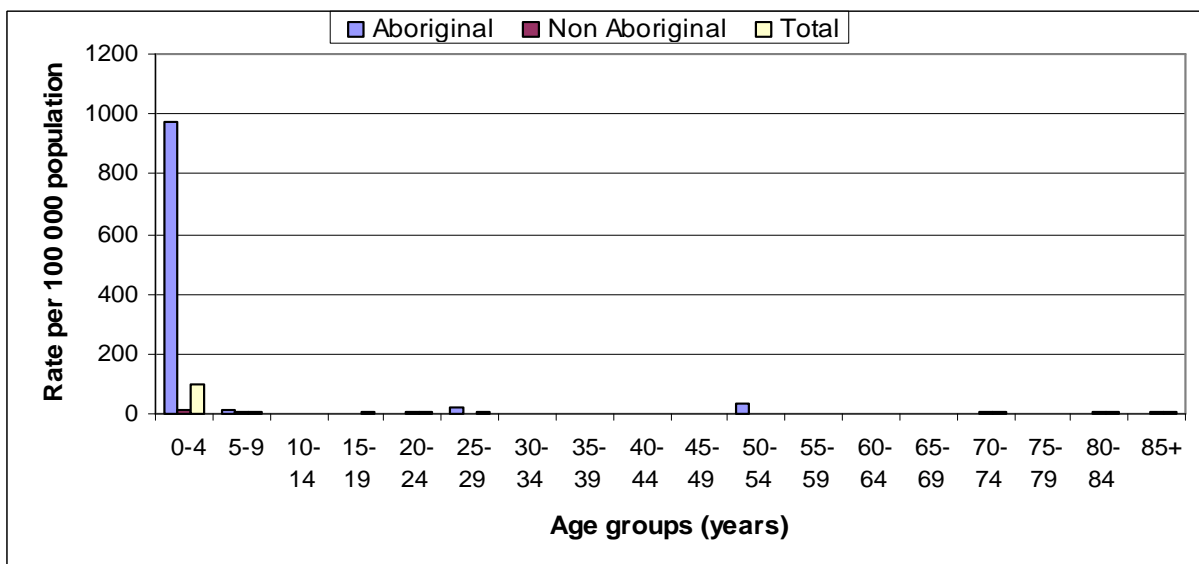


Figure 20: Age-specific notification rates of cryptosporidiosis by Aboriginality, WA, 2005

There was a large difference in notification rates between the population health regions in WA. The Kimberley region had the highest cryptosporidiosis notification rate in WA in 2005 with a rate of 196 cases per 100 000 population (Figure 21). The Metropolitan, Great Southern and Wheatbelt regions all had notification rates that were less than 10 per 100 000 population. The region with the highest notification rate for Aboriginal people was the Kimberley with a rate of 296 per 100 000 population.

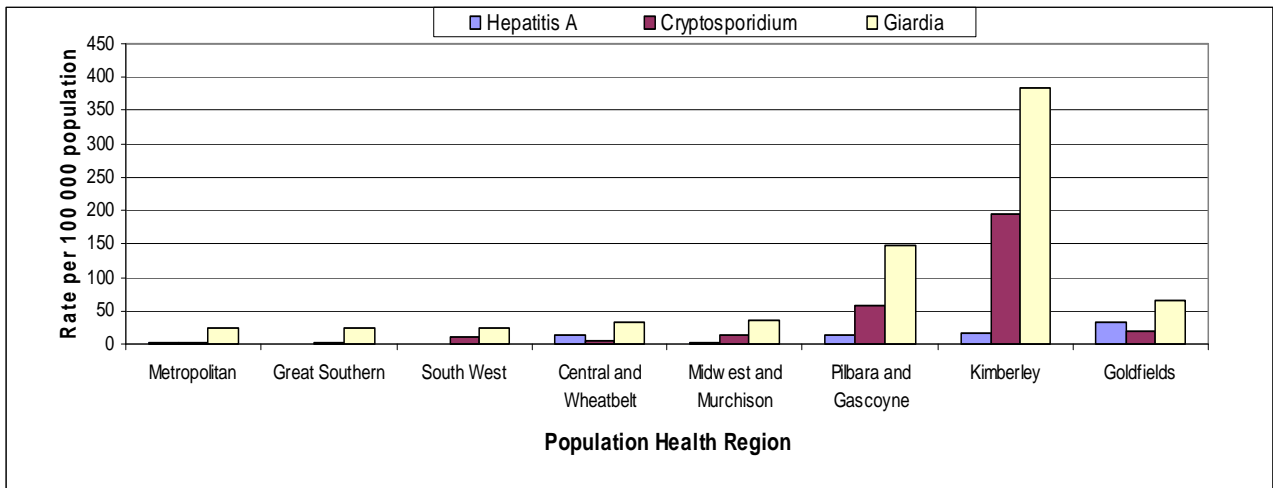


Figure 21: Crude notification rates of hepatitis A, cryptosporidiosis and giardiasis by population health region, WA, 2005

3.2.13 Hepatitis A and E

A total of 73 Hepatitis A infections were notified in 2005, giving a crude notification rate of 3.6 cases per 100,000. This was an increase in the number of cases reported in 2004 (58 cases), and an increase of 30% above the mean rate for the previous four years (Appendix 1).

Increase numbers of notifications were seen in March and May of 2005 (Figure 22). The increased number of cases in May was predominantly in regional areas of the state. The increased number of notifications in May was largely due to transmission of the virus within family/friends groups.

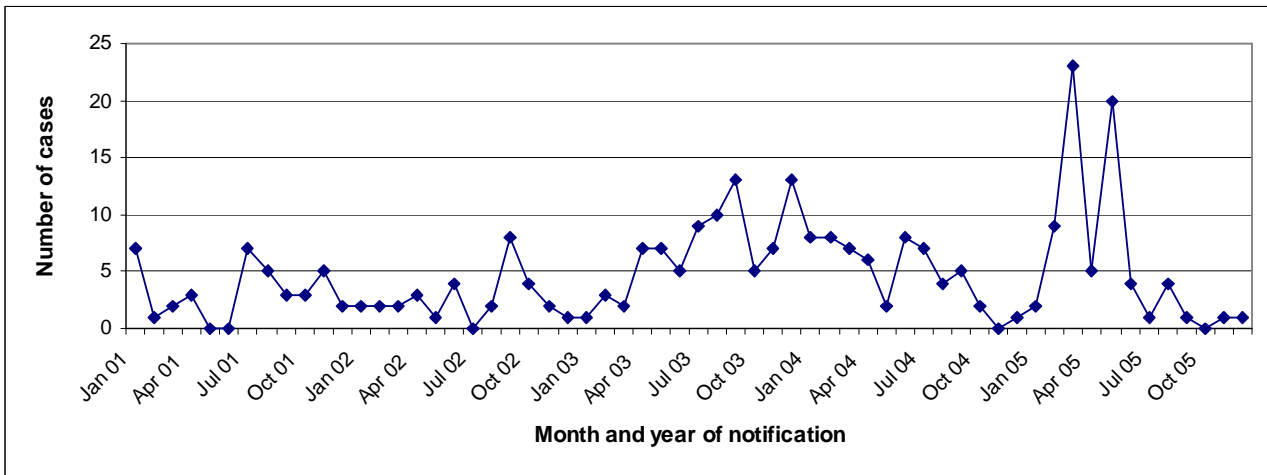


Figure 22: Number of cases of hepatitis A by month and year of notification, WA, 2001 – 2005

The number of cases in each of the age groups was small and varied from 0 to 10 cases, making meaningful interpretation of age-specific rates difficult. However, the greatest number of cases of hepatitis A occurred in the 5-9 year age group and the majority of cases were diagnosed in individuals less than 39 years of age (Figure 23).

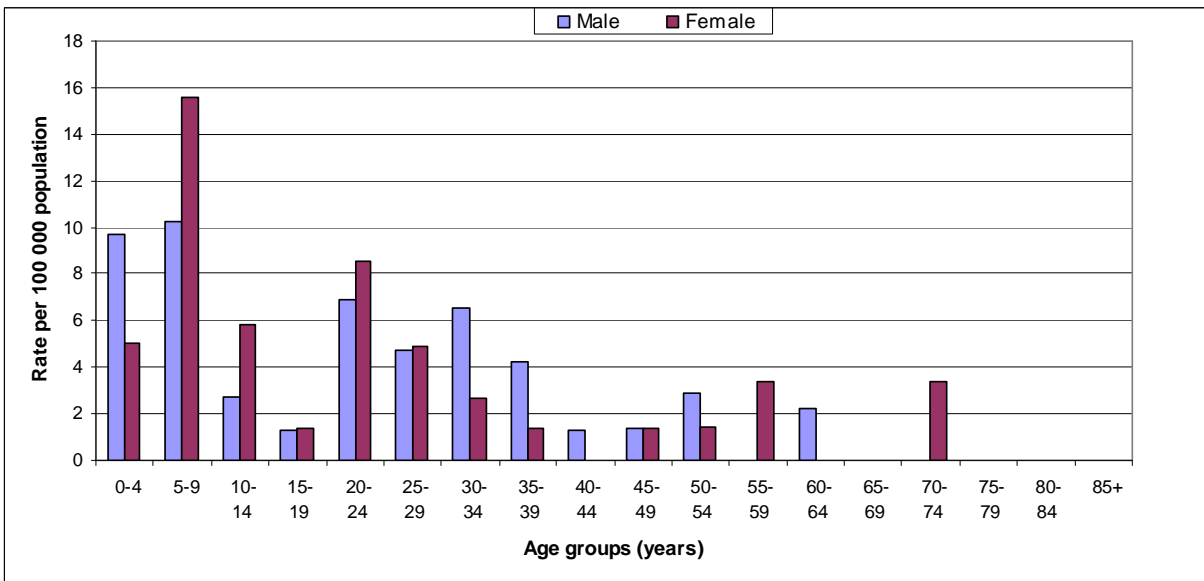


Figure 23: Age-specific notification rates of hepatitis A by sex, WA, 2005

In the Aboriginal population, cases were only reported in individuals 39 years and under, reflecting the general population distribution within this group. The number of cases in these age groups ranged from 1 to 9 cases (rate of infection for these age groups ranging from 16-104 cases per 100,000 people). In contrast, there were cases of hepatitis A in every age group for non-Aboriginals, however these rates were similarly

low (Figure 24). Hepatitis A vaccination for Aboriginal children less than 5 years of age was introduced on 1 Nov 2005 so this may affect Hepatitis A notification rates in future years.

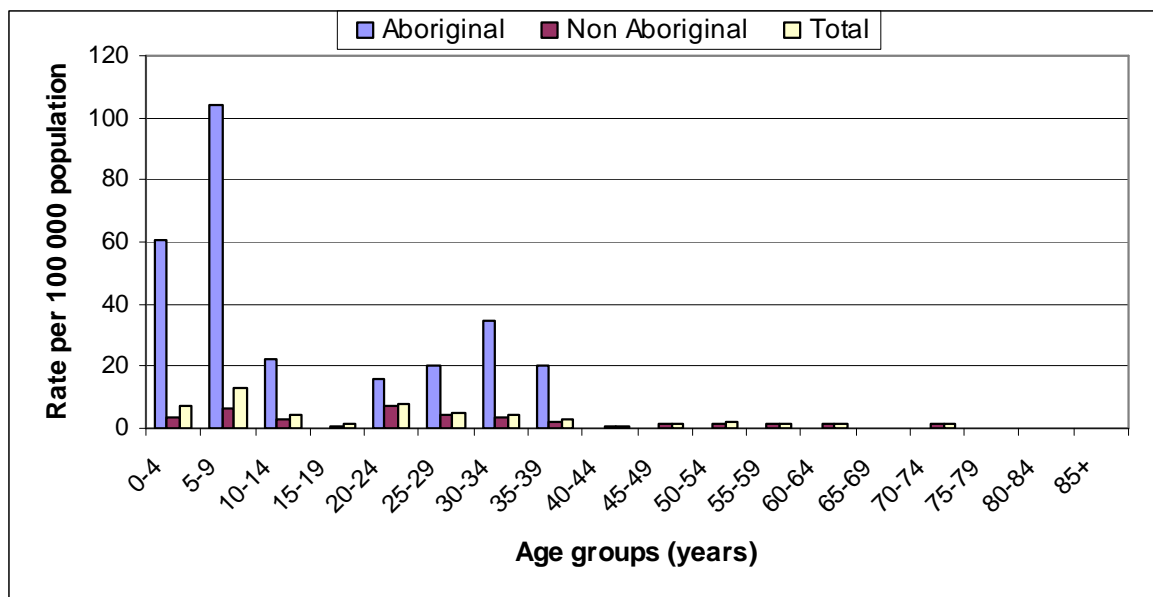


Figure 24: Age-specific notification rates of hepatitis A by Aboriginality, WA, 2005

There were three cases of Hepatitis E notified in 2005, in a 33 year old male and 49 and 23 year old females. Two of the cases had travelled to India and the third had travelled to Indonesia in the month prior to the onset of their infection.

3.2.14 *Vibrio parahaemolyticus* and Botulism

There were no cases of *Vibrio parahaemolyticus* infection notified in 2005. There were no notifications of botulism in 2005, and there had been none in the previous four years.

3.2.15 *Amoebiasis*

Nine cases of *Entamoeba histolytica* infection were reported in 2005 of which six were acquired overseas. The source of infection for three of the cases was not determined. Six males and three females were affected, with a median age 33 years (range 10 – 64 years).

3.3 Discussion

In 2005, the overall number of notifications of gastrointestinal infections in WA remained very stable compared to previous years, with the notification rate very similar to the historical mean (Appendix 1). The crude notification rate for most of the enteric diseases also remained fairly stable, with the exception of STEC which showed a large increase in 2005 attributable to increased detection of STEC genes by PCR using a more sensitive test (see section 5.3). The number of *Campylobacter* and *Salmonella* notifications

also increased when compared to the previous two years, the latter partly attributable to a large *S. Oranienburg* outbreak in the last quarter of the year.

The largest number of enteric infections reported continued to be *Campylobacter*, *Salmonella* and *Giardia* infections, which together constituted 90% of all notifiable enteric infections reported to the Department of Health in 2005. No significant change was observed in the epidemiology of the major enteric diseases in Western Australia in 2005. Notification rates for *Campylobacter*, *Salmonella* and *Cryptosporidium* showed a seasonal pattern with higher numbers of cases in the summer and lower numbers in the winter. This pattern has been observed previously for bacterial enteric pathogens in other countries (Eyles et al. 2003, Gillespie et al. 2005) with a possible link to increased growth of pathogens in food at higher ambient temperatures (Lee and Middleton 2003). Higher rates of Cryptosporidiosis notifications in the summer has been found in other countries (Laupland and Church 2005) with a postulated link to increased use of communal swimming venues (Hlavsa et al. 2005). *Campylobacter* infection was the only disease that showed a difference in notification rates related to sex, with males having a significantly higher number of reported cases than females. This higher notification rate for males was observed in previous years. The largest burden of disease for most enteric pathogens was borne by young children, particularly in Aboriginal populations. Notification rates were also higher for young adults. Notification rates were higher for Aboriginal people as compared to non-Aboriginal people for all enteric infections where number of cases was high enough for valid comparison, with the exception of *Campylobacter*. The greatest difference in notification rates was for *Shigella*, with notification rates 64 times greater for Aboriginal people as compared to non-Aboriginal people. The northern parts of the State (Kimberley and Pilbara/Gascoyne) had the highest notification rates for most diseases geographically.

S. Typhimurium 135/135A was the most common *Salmonella* serotype in WA in 2005, followed by *S. Oranienburg* and *S. Enteritidis* phage type 6A. With the exception of *S. Oranienburg* infections (which were directly related to the outbreak), these two serovars have predominated in WA in previous years. The largest increase in specific *Salmonella* serovars was observed with *S. Typhimurium* PT 12, *S. Corvallis* and *S. Oranienburg*.

4.0 Gastrointestinal and foodborne disease outbreaks

During 2005, 37 outbreaks of gastrointestinal illness were reported to the Department of Health, affecting 929 people. Thirty-two (86%) outbreaks appeared to be due to person-to-person transmission, affecting 681 people (Table 3). The majority of these outbreaks occurred in aged care facilities (n=24), and symptomatically appeared to be due to norovirus, with this confirmed in 11 of the outbreaks. Twenty-one people were hospitalised and there were no deaths.

4.1 Foodborne disease outbreaks

In 2005, five foodborne disease outbreaks affected 201 people (Table 4). A cohort study was conducted in two outbreaks, and a case control study in a third outbreak. Due to the delay in notification of the other two outbreaks, only descriptive information was collected and an environmental investigation conducted. An

agent was only identified in one outbreak (*Salmonella* Oranienburg) associated with alfalfa sprout consumption. A summary of each of the outbreaks is given below.

An outbreak occurred at a youth camp catered for by a commercial caterer in April. Fourteen out of 25 people interviewed (attack rate 56%) (total group 55) were affected with symptoms of nausea, vomiting and diarrhoea. Illness appeared to be of short onset and followed the consumption of salad rolls 8-10 hours prior. The only significant association found was with those individuals who ate a roll for lunch, which interestingly all contained salad (relative risk = 3.0, $p < 0.05$). There were no significant associations with the meat fillings of the roll. However, there were also reports of a person vomiting on the day before the salad rolls were consumed. No faecal or food specimens were collected and thus no pathogen was identified.

The second outbreak occurred in June at a staff lunch with food prepared at individual private residences and brought to the lunch. Eighteen out of 30 (attack rate 60%) people reported symptoms of abdominal pain and diarrhoea approximately 11 hours after consumption of food. No environmental investigation was conducted as the food was not prepared on site. One faecal specimen was tested for bacterial and viral pathogens and was negative. One staff member suggested the chicken curry had been cooked the night before and left out at room temperature overnight. Symptoms were certainly consistent with a *Clostridium perfringens* type intoxication.

Two outbreaks occurred in October, both in restaurant settings and both at the same premises. The first outbreak occurred at a wedding reception where 95 people attended. Twenty-one of 49 people interviewed (attack rate 45%) were ill with symptoms of nausea, vomiting and diarrhoea. Four faecal specimens were tested for bacterial and viral pathogens but were negative. No food specimens were available for testing. Weak associations were observed for some food items, including the chicken roulade (relative risk 1.76), table vegetables (relative risk 1.82) and table salad (relative risk 1.77). Inspection of the premises revealed two concerns – overloading of the fridge and separation of raw and cooked food. The onset times of cases and the lack of secondary cases suggested a point source foodborne outbreak.

The second outbreak in November occurred at a staff development workshop, where 42 work colleagues attended. Fifteen of 31 interviewed were ill (attack rate 48%) with diarrhoea, vomiting and nausea, with a duration of illness from 1 – 6 days and a median of 2 days. No faecal specimens were collected from attendees. Three staff members from the resort also reported illness. Four faecal specimens collected from staff members identified Norovirus in a chef at the venue. The majority of ill people consumed a cooked breakfast on the second day of the workshop, however the staff member with a positive specimen did not work that morning. No food was left over for testing and no significant food associations were found.

4.1.1 *Salmonella* Oranienburg investigation

In mid-November in 2005 laboratory staff at PathWest alerted staff at the Department of Health to an increase in the number of isolations of *Salmonella* Oranienburg. *S.* Oranienburg is a *Salmonella* serotype normally seen infrequently in WA, with 6 – 12 cases notified each year. Twenty-seven cases were notified to the Department of Health in the last two weeks of November. An epidemiological investigation was

commenced and cases were interviewed with a hypothesis-generating *Salmonella* questionnaire that sought information on possible exposures in the week prior to illness, including foods, travel history and contact with infected people. Thirty-seven cases were interviewed but these interviews did not identify a likely source of the *Salmonella* infections. This early investigation did show that there were twice as many female cases as male cases, and that cases were spread throughout Western Australia.

A case-control study was subsequently commenced in January 2006 with a focus on foods more commonly consumed by females than males, particularly fruit and vegetables. In total, 35 cases and 60 controls were interviewed. Consumption of alfalfa sprouts was strongly associated with *Salmonella* infection (odds ratio=32.9). Further investigation showed that cases who reported eating alfalfa had all purchased a particular brand of alfalfa sprouts. Alfalfa sprouts collected from the fridge of one of the cases and from retail outlets in the Perth metropolitan area tested positive for *S. Oranienburg*. Pulsed-field-gel electrophoresis (PFGE) conducted on these isolates showed the PFGE pattern from clinical and food isolates to be indistinguishable.

On the basis of this evidence on 17 February 2006 the manufacturer instigated a voluntary recall of all products produced and distributed in Western Australia and ceased production. The alfalfa production facility was inspected by officers from the City of Cockburn and the Food Safety Branch and further extensive sampling undertaken. *S. Oranienburg* was detected in final product alfalfa samples from this production facility. Investigations to this point have not identified the source of the contamination at the facility. Lucerne seed samples and sprouted seed, swabs from the production facility and bore water samples were all negative for *S. Oranienburg*. Further enquiries are continuing, including enquiries about seed harvest, storage and transport.

In April 2006 the factory had a trial production run with lucerne seeds from a different supplier. *S. Oranienburg* with a PFGE pattern indistinguishable from that during the outbreak was detected in composite alfalfa samples from production drums. This suggests that the facility remained contaminated with *S. Oranienburg*, or that the new batch of seeds was contaminated with *S. Oranienburg* that was genetically indistinguishable from that found in the outbreak. The company recommenced alfalfa sprout production in late June 2006 under a produce, hold, test and release program.

In total, there were 126 notified *Salmonella* cases in Western Australia linked to this outbreak (Figure 25). After the recall in February case numbers declined to notification rates that were similar to those before the outbreak.

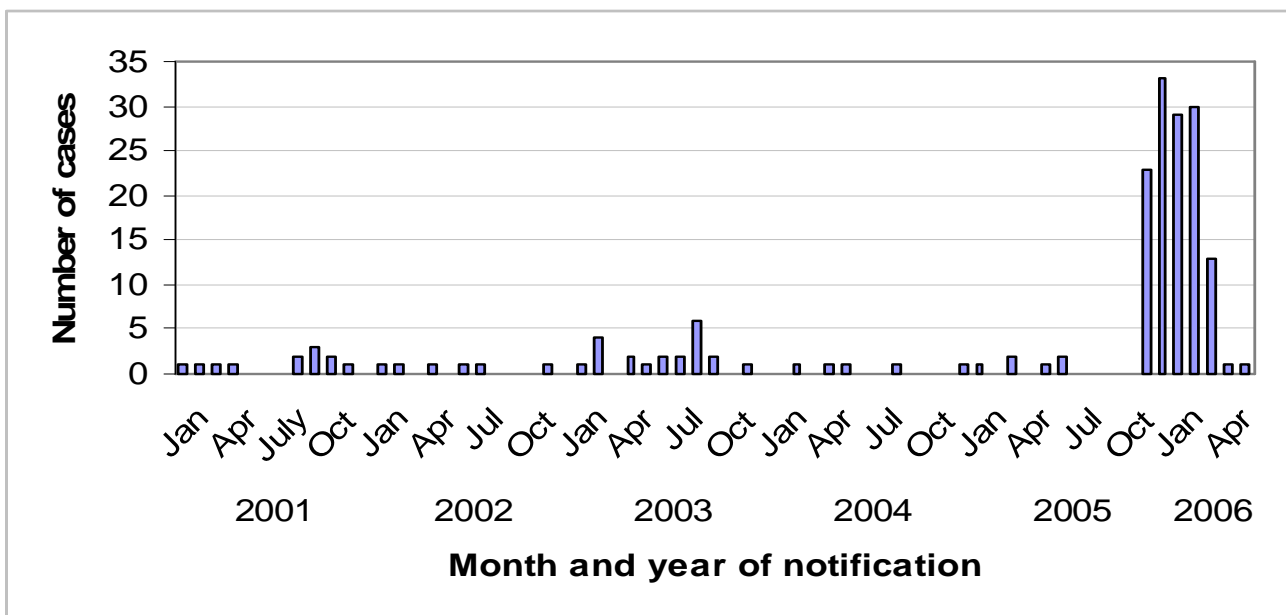


Figure 25: Epidemic curve of *Salmonella* Oranienburg infections, WA, January 2001-April 2006

4.2 Cluster investigations

Cluster investigations are carried out when there is an increase in infections that are epidemiologically related in time, place or person where investigators are unable to implicate a vehicle or determine a mode of transmission for the increase. An example is a temporal or geographic increase in the number of cases of a certain type of *Salmonella* serotype or phage type. Another example is a community-wide increase of an infection that extends over some weeks or months.

A cluster investigation was commenced in May and continued into the third quarter following an increase in the number of *Salmonella* Typhimurium phage type 12 notifications (one case in 2004 versus 26 cases in 2005). Information on food consumption was obtained from 16 of the 22 cases through detailed interviews using hypothesis-generating questionnaires and/or food safety follow-up questionnaires. Two of the cases reported consuming chicken nuggets bought from the same supermarket chain. Sampling of chicken nuggets of the same brand by officers of the Food Safety Branch did not identify any pathogens. Apart from commonly consumed products, no specific venues or brands were identified. In 2005 non-human isolates were found in bovines from the south west of WA and more recently in August in pig offal from an abattoir. One of the later cases was employed at a piggery. The Food Safety Branch investigated a link between pigs and *S. Typhimurium* 12 but did not establish a link.

Two other local clusters were investigated in November and December following increases in the number of notifications. Twelve cases of *Salmonella* Bovismorbificans were investigated with onset dates from 8 October to 27 December. There were five male and seven female cases ranging in age from 2 months – 80 years. Five cases were from regional areas and eight cases were from the metropolitan area. None of the cases reported recent travel. Phage typing revealed that isolates from eight cases were PT 24, one was PT

13, one was PT 11 and 2 were untypable. PFGE typing of isolates from nine cases revealed that six isolates were indistinguishable by PFGE (all PT 24) and three isolates had different PFGE patterns (all other phage types). Eight cases were interviewed and a further case was a recent refugee whose isolate was identified through clearance stools. Two of the cases were cousins who were ill following a church lunch. They reported that a number of other parishioners were also ill but were reluctant to provide contact details.

Twelve cases of *Salmonella* Virchow with specimen collection dates from 9 December to 6 January 2006 were also investigated. There were six male and six female cases ranging in age from 3 – 43 years. Eleven cases were from the metropolitan area and one case was from northern WA. Six cases were interviewed using *Salmonella* hypothesis-generating questionnaires. Within the cluster were mother/daughter and aunt/nephew linked cases. Eleven isolates were PT 8 with the PT pending on the remaining isolate. PFGE typing of isolates from four cases revealed all were indistinguishable. *S. Virchow* was isolated in chicken nuggets in October 2005, however PFGE analysis showed this was a different pattern to the clinical isolates and hence not the likely source of infection. The investigation failed to find a source of infection.

Six cases of *Salmonella* Typhimurium PT 44 with specimen collection dates from 8 – 28 December 2005 were investigated as part of a national investigation into an increase of this particular serotype across several States. These were the first cases seen in WA in 2005. All cases were from the metropolitan area. There were two male and four female cases ranging in age from 34-52 years. Three of the cases were of the same ethnic background but were not linked to each other. Cases were interviewed using the OzFoodNet National hypothesis-generating questionnaire, but no link was established between the cases. The results of a national case control study were equivocal with data suggesting illness was associated with chicken and eggs. No public health action was taken.

Table 3: Outbreaks of non-foodborne gastrointestinal illness in WA by month, setting and agent, 2005

Month of outbreak	Setting	Agent responsible	Number			Evidence*	Epidemiological study†
			Affected	Hospitalised	Deaths		
January	Aged care facility	Unknown	10	0	0	D	CS
January	Aged care facility	Norovirus	18	0	0	M	CS
January	Aged care facility	Unknown	4	0	0	D	CS
January	Aged care facility	Unknown	5	0	0	D	CS
January	Aged care facility	Unknown	3	0	0	D	CS
March	Aged care facility	Unknown	10	0	0	D	CS
March	Aged care facility	Norovirus	7	0	0	M	CS
April	Restaurant	Norovirus	27	0	0	M	N
April	School	Norovirus	25	0	0	M	C
April	Aged care facility	Unknown	30	0	0	D	CS
April	Aged care facility	Rotavirus	13	1	0	M	CS
May	Aged care facility	Norovirus	3	0	0	M	CS
July	Aged care facility	Rotavirus	4	0	0	M	CS
August	Aged care facility	Norovirus	21	0	0	M	CS
August	Aged care facility	Unknown	33	0	0	D	CS
September	Disability Centre	Norovirus	21	0	0	M	CS
September	Aged care facility	Norovirus, Rotavirus	12	1	0	M	CS
September	Aged care facility	Norovirus	9	0	0	M	CS
September	Child care	Unknown	25	2	0	D	CS
September	Aged care facility	Unknown	17	0	0	D	CS
September	Aged care facility	Norovirus	60	1	0	M	CS
October	Aged care facility	Norovirus	14	0	0	M	CS
October	Respite Centre for Disabled Children	Rotavirus, Norovirus, Adenovirus	15	1	0	M	CS
October	Aged care facility	Norovirus	43	3	0	M	CS
October	Aged care facility	Norovirus	44	0	0	M	CS
October	Aged care facility	unknown	16	0	0	D	CS
October	Aged care facility	unknown	15	0	0	D	CS
November	Disability Care Centre	Norovirus	25	0	0	M	CS
November	Aged care facility	Norovirus	59	1	0	M	CS
November	Restaurant	Norovirus	8	1	0	M	N
November	Camp	Unknown	79	0	0	D	C
December	Aged care facility	Unknown	6	0	0	D	CS
Total			681	21	0		

* A=analytical epidemiological evidence; D=descriptive evidence; M=microbiological evidence

† C=cohort study; CS=descriptive case series; CCS=case control study; N=no study.

Table 4: Outbreaks of foodborne disease in WA by month, setting and agent, 2005

Month of outbreak	Setting	Agent responsible	Number				Evidence*	Epidemiological study [†]	Suspected responsible vehicles
			Exposed	Affected	Hospitalised	Deaths			
April	Youth Camp	Unknown	55	14	0	0	A	D	Salad rolls
June	Staff lunch	Unknown	30	18	1	0	A	D	Chicken curry
October	Restaurant	Unknown	95	21	1	0	D	C	Unknown
October	Restaurant	Unknown	42	15	0	0	D	C	Unknown
November	Primary Produce	S. Oranienburg	Unknown	128	11	0	AM	CCS	Alfalfa sprouts
Total				201	13	0			

* A=analytical epidemiological evidence; D=descriptive evidence; M=microbiological evidence

† C=cohort study; CCS=case control study

Table 5: Cluster investigations of Salmonella by month and agent, 2005

Month of outbreak	Setting	Agent responsible	Number			Evidence*	Epidemiological study [†]
			Affected	Hospitalised	Deaths		
May	Unknown	S. Typhimurium PT 12	9	0	0	M	D
August	Unknown	S. Typhimurium PT 12	13	1	0	M	D
November	Unknown	S. Bovismorbificans	12	6	0	M	D
December	Unknown	S. Virchow	12	2	0	M	D
December	Unknown	S. Typhimurium PT44	6	1	0	M	D

5.0 OzFoodNet Activities

5.1 Case control studies

In 2005, the WA OzFoodNet site continued to participate in two national multi-centre case control studies on the pathogens *Salmonella* Enteritidis (locally acquired cases only) and STEC to identify risk factors for these infections. Data collection for the *Salmonella* Enteritidis study was completed in December 2005 and analysis has begun. Participating States included WA, SA, Victoria, NSW and Queensland. In total, 303 cases were notified from November 2001 to December 2005, of which 293 were enrolled into the study. Five of the notified cases were locally acquired infections (1.7%). Nationally, the majority of locally acquired cases recruited into the study were from Queensland (79%) and were predominantly phage type 26. Cases of *S. Enteritidis* with no obvious travel history continue to be followed up intensively by OzFoodNet staff and Food Safety Officers.

The case control study examining risk factors for STEC infection commenced in April 2005 and data collection is on-going. South Australia, Victoria, Queensland and the Hunter Valley region in NSW are the other participating States/regions.

5.2 National information sharing

In 2005, WA contributed to a fortnightly national cluster report to identify foodborne illness occurring in the State. The report is submitted by each State and Territory and collated centrally each fortnight into a national report. The report is useful for identifying common events affecting different parts of Australia as well as for tracking the investigation of multi-state clusters. The cluster report supplements information sharing on a closed list server, at teleconferences and at quarterly face-to-face meetings. In depth epidemiological analyses were also carried out in quarterly and annual reports.

In addition, key de-identified summary information on outbreaks was entered into a centralised national database, allowing the identification of risk factors that occurred repeatedly with similar food vehicles or settings of preparation. The WA OzFoodNet site also participated in a number of national investigations where the pathogen or food product was implicated in WA as well as other States. This national surveillance and investigation allowed the identification of potential lapses in food safety and the assessment of foodborne disease risks, an important step in the planning of future food safety policy interventions.

5.3 Enhanced screening of bloody diarrhoeas for STEC by non-culture based methods

In May 2005, OzFoodNet WA funded a non-culture based STEC screening project for a trial period in collaboration with PathWest. Direct screening of all bloody diarrhoeas using PCR on raw faeces (as opposed to conventional culture followed by confirmatory PCR on nucleic acid derived from the isolate) was employed.

The project was proposed to examine the differences in notification rates between States, and to determine if PCR presented a more sensitive method than culture. Notification rates of STEC infections vary considerably

from State to State, from 0.2 per 100,000 population in WA to 2.5 per 100,000 in South Australia (SA). The high notification rate in SA is likely to be due to screening practices of their reference laboratory, which screens all microscopic and macroscopic bloody diarrhoea samples for the STEC toxin genes *stx I* and *stx II* by PCR, rather than the culture-based screening used in other laboratories.

Validation of the PCR test at PathWest was carried out in collaboration with laboratory staff at the IMVS. A total of 360 samples were tested with collection dates ranging from 27 February 2005 to 1 October 2005 (a seven month period). Thirty-four of these specimens were tested retrospectively. Prospective testing was commenced in mid-May. Of these, pathogens other than *E. coli* were isolated from 106 (29%) specimens. *Campylobacter* species was the most commonly isolated pathogen (67, 18.6%), of which *Campylobacter jejuni* was the most common (34, 9.4%). Of the 360 specimens tested, ten were positive for STEC (2.8%).

In 2005, 1665 specimens were tested in SA, of which 35 were positive, giving a positivity rate of 2.1%, and an annual notification rate of 2.3 per 100,000 (B. Combs 2006, pers comm., 4 August). However, it is interesting to note that only 382 specimens met the criteria for testing in a seven month period. Thus, while the detection rate by PCR does in part explain the difference in the notification rate, more specimens are tested in SA than in WA. Several other key considerations also need to be explored to explain this difference in testing. These include screening criteria for STEC, sensitivity of the tests and referral practices of referring laboratories.

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7.0 References

Department of Health and Ageing 2005, *Foodborne Illness in Australia*, Australian Government Department of Health and Ageing, Canberra.

Eyles R, Niyogi D, Townsend C, Benwell G and Weinstein P 2003, Spatial and temporal patterns of *Campylobacter* contamination underlying public health risk in the Taieri River, New Zealand, *J. Environ. Qual.*, vol. 32, pp. 1820-1828.

Gillespie I A, O'Brien S J, Adak G K, Ward L R and Smith H R 2005, Foodborne general outbreaks of *Salmonella* Enteritidis phage type 4 infection, England and Wales, 1992-2002; where are the risks, *Epidemiol. Infect.*, vol. 133, no. 5, pp. 795-801.

Hlavsa M C, Watson J C and Beach M J 2005, Cryptosporidiosis surveillance-United States 1999-2002, *MMWR Surveill. Summ.*, vol. 54, no. 1, pp. 1-8.

Laupland K B and Church D L 2005, Population-based laboratory surveillance for *Giardia* sp. And *Cryptosporidium* sp. Infections in a large Canadian health region, *BMC Infect. Dis.*, vol. 5, pp. 72.

Lee M B and Middleton D 2003, Enteric illness in Ontario, Canada, from 1997 to 2001, *J. Food Prot.*, vol. 66, no. 6, pp. 953-61.

Parry S M, Salmon R L, Willshaw G A and Cheasty T 1998, Risk factors for and prevention of sporadic infections with vero cytotoxin (shiga toxin) producing *Escherichia coli* O157, *Lancet*, vol. 351, no. 9108, pp. 1019-22.

Sarna M, 2003, *Gastrointestinal illness in Australia: incidence, notifications and outbreaks. Annual report of the WA OzFoodNet site*, Western Australian Department of Health.

Appendix 1: Number of notifications, crude notification rate and ratio of current to historical mean by pathogen/condition, 2001-2005, WA

Pathogen/ Condition	Year										Mean rate 2001-2004 ⁴	Rate ratio 2005 to mean ⁵
	2001 (n=1,901,159)		2002 (n=1,927,322)		2003 (n=1,949,439)		2004 (n=1,973,671)		2005 (n=2,000,459)			
	No.	Rate	No.	Rate	No.	Rate	No.	Rate	No.	Rate		
Campylobacter	2579	135.6	2175	112.8	1947	99.9	1969	99.8	2422	121.1	112.0	1.1
Salmonella	862	45.3	735	38.1	612	31.4	615	31.2	791	39.5	36.5	1.1
Giardia	977	51.4	965	50.1	772	39.6	926	46.9	747	37.3	47.0	0.8
Cryptosporidiosis	167	8.8	220	11.4	444	22.8	121	6.1	189	9.4	12.3	0.8
Shigella	81	4.3	127	6.6	111	5.7	112	5.7	151	7.5	5.6	1.3
Hepatitis A	38	2.0	31	1.6	82	4.2	58	2.9	73	3.6	2.7	1.3
STEC ^{1,2}	3	0.2	4	0.2	2	0.1	1	0.05	12	0.6	0.1	6
Amoebiasis	14	0.7	11	0.6	7	0.4	8	0.4	8	0.4	0.5	0.8
Typhoid fever	14	0.7	5	0.3	10	0.5	5	0.3	7	0.3	0.4	0.8
Listeria	11	0.6	11	0.6	8	0.4	9	0.5	4	0.2	0.5	0.4
Paratyphoid fever	6	0.3	5	0.3	0	0	13	0.7	4	0.2	0.3	0.7
Hepatitis E	1	0.05	0	0	0	0	2	0.1	3	0.1	-	-
Cholera	0	0	0	0	0	0	1	0.05	1	0.05	-	-
HUS ¹	0	0	0	0	1	0.05	1	0.05	1	0.05	-	-
Vibrio parahaemolyticus	2	0.1	6	0.3	3	0.15	3	0.15	0	0	-	-
Yersinia	3	0.2	3	0.2	3	0.15	1	0.05	2	0.1	-	-
Total	4758		4298		4002		3845		4415			

¹Abbreviations: STEC: Shiga-toxin producing *E. coli*; HUS: Haemolytic Uraemic Syndrome ²Shiga-toxin producing *E. coli* (STEC) and Cryptosporidiosis were made notifiable in 2001 ³Rate per 100,000 ⁴Mean of rates between 2001 and 2004 where applicable ⁵Ratio has not been calculated for diseases with small number of case

