NSW HEALTH

Vol. 19(1–2) • January–February 2008
State Health Publication PH 070070
ISSN 1034 7674

NSW PUBLIC HEALTH BULLETIN

Special Issue – Enteric diseases

Improving foodborne disease surveillance in NSW

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The term 'enteric diseases' includes a multitude of conditions. In the public health context, it usually refers to infections (or intoxications) that are food- or waterborne, or otherwise transmitted by the faecal-oral route. Symptoms are usually non-specific – nausea, vomiting, diarrhoea, abdominal pain and fever in varying combinations – and unpleasant, rather than serious or life threatening. The importance of enteric infections lies mainly in their numbers. The results from *The National Gastroenteritis Survey 2001–2002* estimated that 17.2 million cases of gastroenteritis occur annually in Australia, leading to 7 million courses of medication (including antibiotics), 6 million days of paid work lost, 3.7 million doctor visits and 0.5 million stool tests.[1] A third of gastroenteritis cases (5.4 million each year) are due to foodborne infection. There are also approximately 6000 cases annually of other foodborne diseases in which gastrointestinal symptoms are not prominent, such as invasive listeriosis.[2] Overall, foodborne diseases are responsible for approximately 18000 hospital admissions and 120 deaths, and cost $1249 million, annually in Australia.[3]

The perception within the community that foodborne and institutional outbreaks of enteric disease may represent negligence or system failure can often provoke substantial and disproportionate attention and, occasionally, litigation.[4] Combined with the high disease burden, this provides a strong incentive for active surveillance to identify outbreaks. Periodic reviews of the value and effectiveness of disease prevention programs are important and this special issue of the *NSW Public Health Bulletin* contributes to this field.

Routine notifiable disease data capture only a tiny fraction of enteric disease burden, namely from laboratory-confirmed cases due to some specific pathogens and easily recognised disease outbreaks. The number of notifications depends on variables other than actual disease rates, including: whether a general practitioner orders a stool examination; laboratory diagnostic and strain typing methods; and the local capacity for outbreak recognition and investigation. For example, the article by Cretikos, Telfer and McAnulty on enteric disease outbreak reporting in New South Wales notes that the highest rate of outbreaks was reported by the only Public Health Unit with resources dedicated to enteric disease surveillance and control. Presumably this reflects better ascertainment of cases rather than higher disease rates. An evaluation of the enteric disease outbreak surveillance system by the same authors showed that many users found it cumbersome and labour intensive. The proposed changes should improve the system's efficiency and reduce inconsistencies of rates and delays in outbreak reporting.

The consistency and timeliness of disease notification depend on resources and competing priorities; and the value of the data obtained depends on data quality and how rapidly it is analysed and acted on. Increasingly, the time-consuming form filling and duplication of paper-based notification is being replaced by electronic systems. These have the potential for simultaneous, immediate data transfer from multiple sources to a central database where information can be combined and analysed rapidly. Although electronic systems, such as the *NetEpi Collection* discussed by Viney and McAnulty, have significant advantages, they may not be immediately acceptable to users without access to appropriate facilities, training and support.[5] The Public Health Real-time Emergency Department Surveillance System (PHREDSS) is an electronic syndromic surveillance system, recently introduced in NSW.[6] It automatically receives and analyses data from existing emergency department clinical information systems, in near real-time. The PHREDSS is designed to identify outbreaks rapidly that

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may not be easily recognised by individual public health units. As illustrated by the report of a gastroenteritis outbreak at a school music camp, Mannes et al. show that PHREDSS can also provide individual patient information and identify additional cases in recognised outbreaks.

When based on spatiotemporal clustering of specific pathogens, laboratory notification can also identify outbreaks that are not otherwise recognisable, as described in two following papers by Viney et al. and Wang et al. Although insensitive, this method is generally consistent, objective and specific and, in conjunction with epidemiological investigations, can identify risk factors and potential sources. However, diagnostic laboratories identify common enteric pathogens, like *Salmonella enterica* and *Campylobacter jejuni*, only to the level of species, and additional typing is needed to identify outbreaks against high background rates. None of the many possible *Campylobacter* strain-typing methods are generally accepted or used routinely.[7] Therefore outbreaks are rarely recognised and campylobacteriosis is not notifiable in NSW, although data from elsewhere in Australia indicate that it is significantly more common than salmonellosis. State reference laboratories throughout Australia perform *Salmonella* serotyping (see Wang et al. in this issue). Reference laboratories can identify suspected outbreaks due to uncommon serotypes, but further subtyping of the most common serotype, *S.* Typhimurium, is needed. Faster, more discriminatory methods for *S.* Typhimurium typing, as described by Gilbert, have the potential to identify more outbreaks more rapidly. While such methods may stretch public health resources, they should increase the success rate of investigations.

The mainstays of prevention for most enteric diseases are safe food production and handling, and good infection control practice, especially to prevent outbreaks of norovirus in institutions. A few enteric diseases are preventable by vaccines, including polio, which is close to being eradicated worldwide, and hepatitis A, which occurs at low rates in Australia (see Ward and McAnulty in this issue). Continued surveillance of these viral diseases is needed to monitor, control and identify appropriate target populations for immunisation. More generally, the consistent surveillance and investigation of foodborne disease and institutional outbreaks of enteric infections, are essential for understanding the changing epidemiology of these diseases and for evaluating the effectiveness of interventions, on which control and prevention depend.

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The Bulletin would like to acknowledge Jeremy McAnulty and Jennie Musto for their contribution to this issue.

Enteric disease outbreak reporting, New South Wales, Australia, 2000 to 2005

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Abstract: Objective: To review enteric disease outbreaks reported to the NSW Department of Health. **Methods:** Data from existing electronic enteric disease outbreak summary databases were used to describe the number and type of outbreaks reported, burden of illness and cause of the outbreaks. **Results:** Between 2000 and 2005, 998 enteric disease outbreaks were reported (148 foodborne and 850 non-foodborne), affecting 24 260 people and associated with 771 hospitalisations and 21 deaths. *Salmonella* was confirmed in 28 per cent of foodborne outbreaks, and norovirus in 18 per cent of non-foodborne outbreaks.
Conclusions: Enteric disease outbreaks cause a substantial burden of disease in NSW.

While many infectious agents can cause outbreaks, most of the outbreaks reported in NSW involve enteric diseases, which are caused by the ingestion of infectious agents or toxins, and result in nausea, vomiting, diarrhoea, abdominal cramps and fever, or other symptoms.[1,2]

Enteric diseases place a substantial burden on the community. It has been estimated that in Australia there are approximately 17.2 million cases of gastroenteritis each year.[3] A national survey of gastroenteritis in Australia during 2001–2002 found that one-third of working adults miss at least one day of work when they have gastroenteritis, and one-third of cases result in a caregiver missing work.[3]

Of these gastroenteritis cases, 5.4 million are estimated to be cases of foodborne-associated gastroenteritis, equivalent to 0.3 episodes of foodborne gastroenteritis per person per year, and resulting in approximately 18 000 hospitalisations and 120 deaths in Australia annually.[4] Many outbreaks of gastroenteritis occur in institutions, such as schools, childcare centres and residential care facilities. Institutional outbreaks of gastroenteritis are usually caused by highly infectious viruses such as norovirus and rotavirus, and spread predominantly through person-to-person contact.[1,5]

Under the *Public Health Act (NSW) 1991*, 12 enteric conditions are currently notifiable in NSW and must be reported to NSW Health by doctors, laboratories and hospitals, including: botulism, cholera, cryptosporidiosis, giardiasis, haemolytic uraemic syndrome (HUS), shigatoxigenic *Escherichia coli* (STEC) infection, hepatitis A, hepatitis E, listeriosis, salmonellosis, shigellosis and typhoid. Some of these diseases are primarily foodborne in origin, such as salmonellosis, typhoid and listeriosis.

In addition to individual disease notifications, outbreaks of suspected foodborne disease in two or more people related in time or place, and outbreaks of gastroenteritis among people of any age in an institution (eg an educational, residential, childcare or healthcare institution) are also notifiable by doctors and institutions. Where public health units identify clusters of disease or outbreaks, they will launch an outbreak investigation. Summary reports of enteric disease outbreaks are provided by public health units and entered into the relevant enteric disease outbreak databases held at the NSW Department of Health.

We analysed enteric disease outbreaks reported to the NSW Department of Health for the five-year period July 2000–June 2005. This analysis aimed to describe the number, epidemiology and cause of the enteric disease outbreaks reported during this period.

Methods

The two existing NSW Health enteric disease outbreak databases – the OzFoodNet Outbreak Summary Database and the Gastroenteritis in Institutions Database – were used. For the purposes of the present study, the data contained within the two enteric disease outbreak summary databases was separated into two categories:

- foodborne and suspected foodborne outbreaks, and
- non-foodborne outbreaks of enteric disease.

If the setting of the outbreak was an institution (ie aged care facility, hospital, child care centre, school, correctional facility or other institutional setting) and the method of transmission was unknown, the outbreak was assumed

Table 1. Number and type of enteric disease outbreaks, New South Wales, 2000–2005

Financial year	Foodborne or suspected foodborne		Non-foodborne	
	Institutional *n*	Non-institutional *n*	Institutional *n*	Non-institutional *n*
2000–01	0	25	43	7
2001–02	1	36	51	11
2002–03	2	28	121	21
2003–04	11	26	390	9
2004–05	0	19	181	16
Total	14	134	786	64

Source: Gastroenteritis in Institutions Database; OzFoodNet Outbreak Summary Database.

to be a non-foodborne outbreak unless *Salmonella*, which is commonly transmitted by the foodborne route, was identified. Only two *Salmonella* outbreaks in institutions were attributed to a foodborne route as a result of this decision. For the purposes of analysis, all foodborne and suspected foodborne outbreaks (including those occurring in an institutional setting) were considered to be foodborne outbreaks. All other outbreaks were considered to be non-foodborne outbreaks.

The number and type of enteric disease outbreaks, the reported number of people affected, the reported number of hospitalisations, the reported number of deaths and the annual rate of enteric disease outbreaks reported by NSW public health units for each 100 000 population were estimated. Mode of transmission, extent of spread and cause were also examined.

An enteric agent (organism or toxin) was considered to have caused the outbreak if it was identified through microbiological testing of a clinical specimen and/or the implicated food vehicle and the epidemiologic features were consistent with the features of disease caused by the agent.

Tests of proportions were performed using the chi-square test. Tests of duration of illness were performed using the Kruskal–Wallis test.

Results

Between 1 July 2000 and 30 June 2005, 998 enteric disease outbreaks were reported in NSW (Table 1). The number of foodborne outbreaks reported each year was reasonably stable, with an average of 30 (15 per cent of all outbreaks). The number of reported non-foodborne outbreaks increased substantially between 2000 and 2005, with an epidemic year of viral gastroenteritis in 2004 (Figure 1). The majority of non-foodborne outbreaks occurred in institutions.

These outbreaks affected 24 260 people. At least 2072 people (8.5 per cent) reported seeing a doctor as a result of

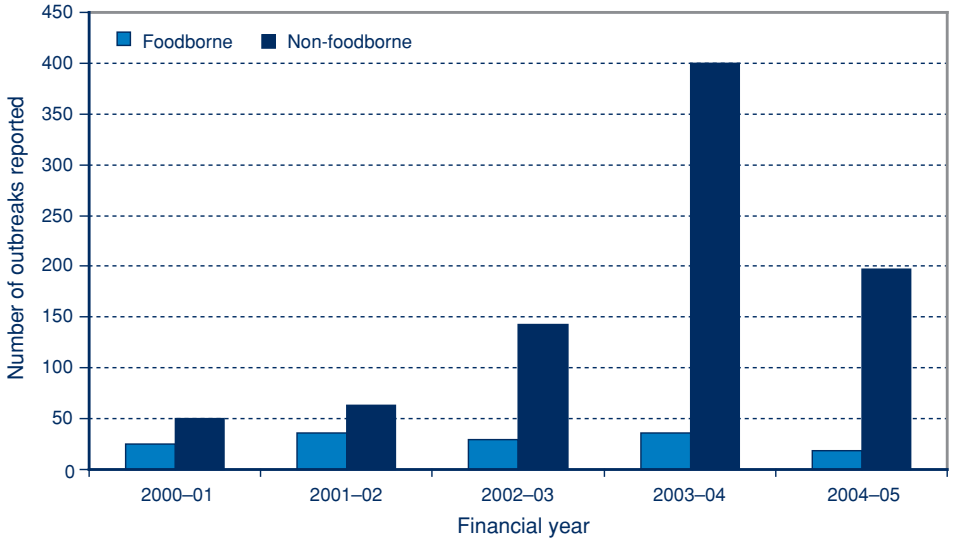

Figure 1. Total number of enteric disease outbreaks reported by financial year in New South Wales, 2000–2005.
Source: Gastroenteritis in Institutions Database; OzFoodNet Outbreak Summary Database.

Table 2. Number of people affected, hospitalised and dying in association with enteric disease outbreaks, New South Wales, 2000–2005
[a]Of those affected

Financial year	Foodborne or suspected foodborne				Non-foodborne				Total enteric disease outbreaks			
	Affected *n*	Hospitalised *n*	(%)[a]	Died[a] *n*	Affected *n*	Hospitalised *n*	(%)[a]	Died[a] *n*	Total affected *n*	Total hospitalised *n*	(%)[a]	Total died[a] *n*
2000–01	273	10	(3.7)	0	1126	57	(5.1)	1	1399	67	(4.8)	1
2001–02	693	52	(7.5)	1	1437	27	(1.9)	0	2130	79	(4.0)	1
2002–03	550	29	(5.3)	0	3462	157	(4.5)	0	4012	186	(4.5)	0
2003–04	616	81	(13.2)	1	11626	287	(2.5)	16	12242	368	(3.0)	17
2004–05	185	20	(10.8)	0	4292	51	(1.2)	2	4477	71	(1.6)	2
Total	2317	194	(8.4)	2	21943	579	(2.6)	19	24260	771	(3.2)	21

Source: Gastroenteritis in Institutions Database; OzFoodNet Outbreak Summary Database.

their illness, 771 people (3.2 per cent) required hospitalisation and 21 people (<0.1 per cent) died (Table 2).

The median duration of reported illness in affected people was 1.5 days (inter-quartile range (IQR) 1–2 days). The median duration was significantly longer for foodborne illness (2 days, IQR 1–4 days) than for non-foodborne illness (1.3 days, IQR 1–2 days, $p < 0.001$). In 17 of the outbreaks, several people were still reported to be ill at the time the summary report was provided, which indicates the true duration of illness would be longer than that reported.

Among the foodborne outbreaks, 14 of the 198 non-institutional outbreaks were quite widespread, with five multi-state outbreaks and nine outbreaks involving multiple Area Health Service regions within NSW. Nine of these 14 multi-region outbreaks were reported to be caused by various *Salmonella* species. In the remainder, the cause was unknown. The most commonly reported mode of transmission of non-foodborne outbreaks was person-to-person (320/850 person-to-person: 37.6 per cent; 452/850 suspected person-to-person: 53.2 per cent).

The total rates of reporting of enteric disease outbreaks by public health unit were highly variable, ranging from 0.5 to 7.4 per 100000 population (Figure 2).

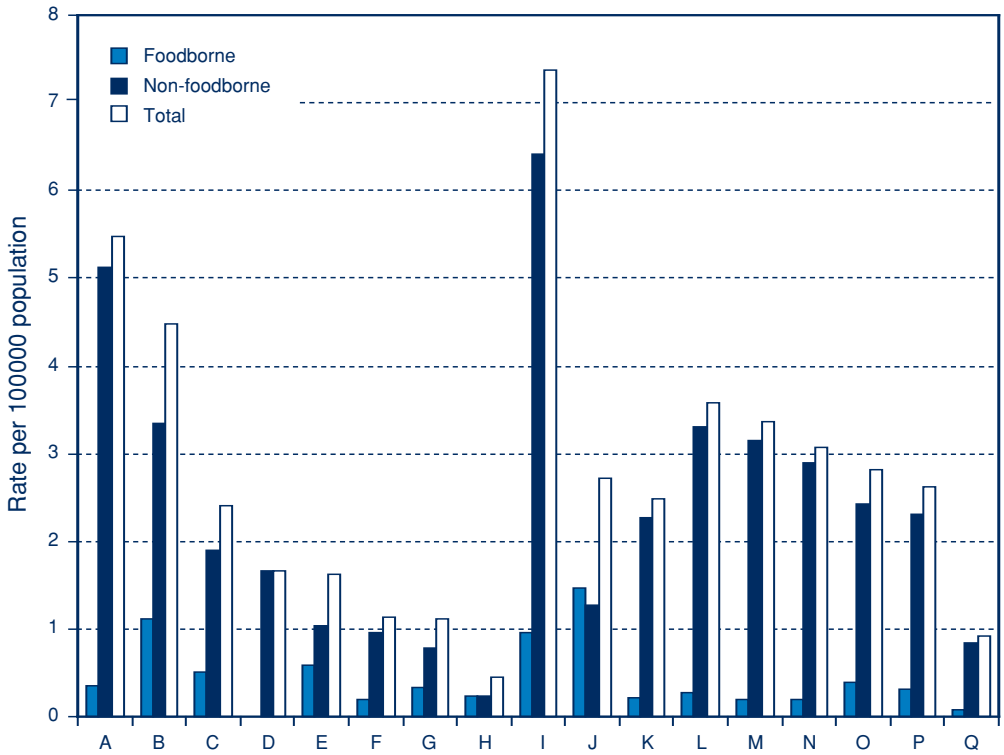

Figure 2. Rate of reporting of enteric disease outbreaks in New South Wales by public health unit, 2000–2005.
Source: Gastroenteritis in Institutions Database; OzFoodNet Outbreak Summary Database.

Table 3. The reported cause of enteric disease outbreaks, New South Wales, 2000–2005

Reported organism/toxin	Foodborne *n*	Non-foodborne *n*	Total *n*
Ciguatoxin	1	0	1
Hepatitis A	1	0	1
Vibrio parahaemolyticus	1	0	1
Blastocystis hominis	0	1	1
Cryptosporidium	0	1	1
Dientamoeba fragilis	0	1	1
Clostridium difficile	0	2	2
Giardia	0	2	2
Clostridium perfringens	3	0	3
Scombrotoxin	3	0	3
Toxin of unspecified type	4	0	4
Campylobacter jejuni	2	2	4
Both norovirus and rotavirus	0	4	4
Rotavirus	1	24	25
Salmonella species	42	2	44
Norovirus	2	150	152
Unknown	88	661	749
Total	148	850	998

Source: Gastroenteritis in Institutions Database; OzFoodNet Outbreak Summary Database.

At least one stool specimen was reported to have been collected in a significantly smaller proportion of foodborne (54/148: 36 per cent) than non-foodborne outbreaks (391/850: 46 per cent, $p = 0.02$). The causative agent for the outbreak was determined in a significantly larger proportion of the foodborne (60/148; 41 per cent) than non-foodborne outbreaks (189/850: 22 per cent, $p < 0.001$). By far the most common cause of the outbreaks was salmonellosis for foodborne outbreaks, and norovirus and rotavirus for non-foodborne outbreaks (Table 3).

Discussion

Enteric disease outbreaks create a substantial burden of illness in NSW, but these outbreaks rarely result in severe illness or death. The majority of the enteric disease outbreaks reported during 2000–2005 were non-foodborne institutional outbreaks of viral gastroenteritis, particularly during the epidemic year of 2004. These outbreaks affected many thousands of people and were associated with a small number of deaths. Rapid control of viral institutional outbreaks should be a priority in order to prevent such large institutional outbreaks in the future.

There are several limitations to the present study. First, given that there are approximately 17.2 million cases of gastroenteritis in Australia annually, it is likely that many enteric disease outbreaks that occur in NSW are not detected or reported.[3] This indicates that these results are likely to be an underestimate of the true burden of disease from enteric disease outbreaks in NSW. Second, the rate of reporting of enteric disease outbreaks is also inconsistent in public health units, which suggests that the enteric disease outbreaks reported do not represent the true distribution of disease in NSW. Finally, the mode of transmission for the majority of the outbreaks was unconfirmed, making the determination of a transmission mode uncertain.

It is important to note that the public health unit with the highest rate of reporting of enteric disease outbreaks (PHU I) is the only unit in NSW that has staff funded exclusively to perform enteric disease surveillance and control activities. This may account for the high rates of reporting of enteric disease outbreaks from this area. Other reasons for the variation in outbreak reporting are unclear, but may include: variations in public health unit capacity due to other competing public health activities; the number of staff trained to investigate enteric diseases; the number of institutions, such as aged care facilities, in each area; the level of notification from institutions and the community; and other local characteristics, including exposure to farm animals, water quality and other environmental factors.

Conclusion

Given the burden of illness caused by enteric disease outbreaks, the identification, investigation, control and prevention of enteric disease outbreaks in NSW are important public health activities.

Acknowledgements

Thanks to Jennie Musto for reviewing earlier versions of this document and providing valuable feedback. Thanks also to all the public health unit staff who provided outbreak summary reports during the period of the evaluation.

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Evaluation of the system of surveillance for enteric disease outbreaks, New South Wales, Australia, 2000 to 2005

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Abstract: Aim: To evaluate the NSW enteric disease outbreak surveillance system. **Evaluation methods:** We performed unstructured interviews with NSW Health Communicable Diseases Branch staff and analysed summary outbreak reports for July 2000 to June 2005. **Performance of the surveillance system:** The system provided a mechanism for meeting all of its objectives to some level. Limitations included difficulty in monitoring outbreaks, incomplete outbreak information, difficulty in linking and collating information, and the cumbersome and inflexible data management system. **Conclusions:** The NSW enteric disease outbreak surveillance system is serving a useful public health function, but could be improved through the use of more sophisticated electronic data management techniques.

Surveillance systems are designed to identify, investigate, control and report rapidly on outbreaks of disease, and to identify factors that can help prevent future outbreaks.[1] Enteric disease outbreaks have been notifiable in NSW since 1991, and surveillance has been progressively improved from 2000 onwards with the introduction of OzFoodNet, a collaborative national network for the surveillance of enteric disease.[2] This network aims to provide better understanding of the causes and incidence of foodborne disease in the community, and an evidence base for policy formulation.[2]

A description of the notification and reporting requirements for enteric disease outbreaks in NSW is provided in Table 1. In NSW, public health units (PHUs) are responsible for investigating notifications of enteric disease, and providing summary reports of enteric disease outbreaks to the NSW Department of Health using standard reporting forms. Outbreaks may also be detected through review of routinely collected notifiable diseases surveillance data, the NSW Food Authority complaints hotline, or reports from clinicians, institutions or laboratories, and members of the public (Figure 1).

The NSW enteric disease outbreak surveillance system collects data from all NSW public health units; it therefore covers the entire population of NSW. The specific objectives of the NSW enteric disease outbreak surveillance system as described in the NSW Health *Notifiable Diseases Manual* are to:

- identify the source of the outbreak
- prevent further cases
- monitor the epidemiology to inform the development of better prevention strategies
- monitor the development of unusual or emerging pathogens
- fulfil international reporting requirements.[3]

We undertook the first evaluation of the NSW enteric disease outbreak surveillance system, which aimed to:

- determine whether the objectives of the system are being met
- evaluate the specific attributes of the system
- identify areas for improvement.

Evaluation methods

The evaluation was based on the Centers for Disease Control and Prevention guidelines for evaluating surveillance systems (Table 2).[4,5] The public health importance was derived from the results of the accompanying enteric disease outbreak review presented in this issue.[6]

The simplicity, flexibility and acceptability of the system were examined through unstructured interviews with key Communicable Diseases Branch (CDB) staff, and by detailing the flow of information through the system (Figure 1). Other system attributes were examined by analysing the summary reports from the two NSW enteric disease outbreak databases (the Gastroenteritis in Institutions Database and the OzFoodNet Outbreak Summary Database), where symptom onset for the first case occurred between 1 July 2000 and 30 June 2005.

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Table 1. Notification and reporting requirements for enteric disease outbreaks in New South Wales

Type of outbreak	Definition	Public health unit notification and reporting requirements	Responsibility for investigation
Suspected or confirmed foodborne outbreak, or person-to-food-to-person outbreak	Two or more people who are linked in time or place with acute onset of enteric or other symptoms caused by ingestion of infectious agents or toxins that may have been acquired by consuming contaminated food or drink	Within 1 working day of identifying an outbreak, notify CDB and NSW Food Authority. On the day epidemiological results are determined, send completed 'Initial Epi Report Form' and any other relevant data forms to the CDB and NSW Food Authority. Within 1 month of finalisation of an investigation send a completed OzFoodNet Outbreak Summary Form to the CDB	NSW Health is responsible for investigating the epidemiology of the outbreak and the NSW Food Authority is responsible for providing an environmental investigation and conducting trace-back investigation of the source of food products, and dealing with food industry partners
Non-foodborne outbreaks including: • waterborne • animal-to-person • environment to person • person-to-person transmission not in an institutional setting • outbreaks of unknown origin	Two or more people who are linked in time or place report acute onset of enteric or other symptoms	Within 1 working day of identifying an outbreak notify CDB and NSW Food Authority. On the day epidemiological results are determined send completed 'Initial Epi Report Form' and any other relevant data forms to the CDB. Within 1 month of completion of an investigation send a completed OzFoodNet Outbreak Summary Form to the CDB	NSW Health
Gastroenteritis in an institutional setting (e.g. residential, educational, child care, or health care institutions)	A person within an institution with vomiting or diarrhoea thought to be infectious, at a time when at least one other person at the institution has vomiting or diarrhoea	Within 1 working day of identifying an outbreak notify CDB and NSW Food Authority. Within 1 month of completion of an investigation send a completed 'PHU Report Form for Investigation of a gastroenteritis outbreak in an institution' to the CDB	NSW Health as well as the institution and its infection control officer

CDB: Communicable Diseases Branch, NSW Department of Health.

To assess the completeness of enteric disease outbreak reporting by PHUs, we calculated the proportion of all outbreaks first reported in any form (including phone calls, emails and other initial reports) to the CDB from January to December 2005 that had a completed final outbreak summary report. Timeliness of reporting was evaluated by determining the period between the date of symptom onset for the first case and the date of the final summary outbreak report.

Completeness was assessed by examining the completeness of key data fields, including: onset date for the first case; summary report date; number of people at risk; number of people affected; number of hospitalisations; number of deaths; and number of clinical specimens collected.

The usefulness of the system was examined by reviewing the objectives of the surveillance system, interviewing key informants from the CDB enteric diseases team and reviewing the output of the system, including reports and policy interventions. Policy outputs for the period 1 July 2000 to 30 June 2005 were identified in past editions of the *NSW Public Health Bulletin* and through interviews with the CDB staff.

Performance of the surveillance system

Simplicity

Enteric disease outbreaks in NSW were identified and reported through several mechanisms (Figure 1). The maintenance and integration of the flow of outbreak information along these pathways was primarily a manual

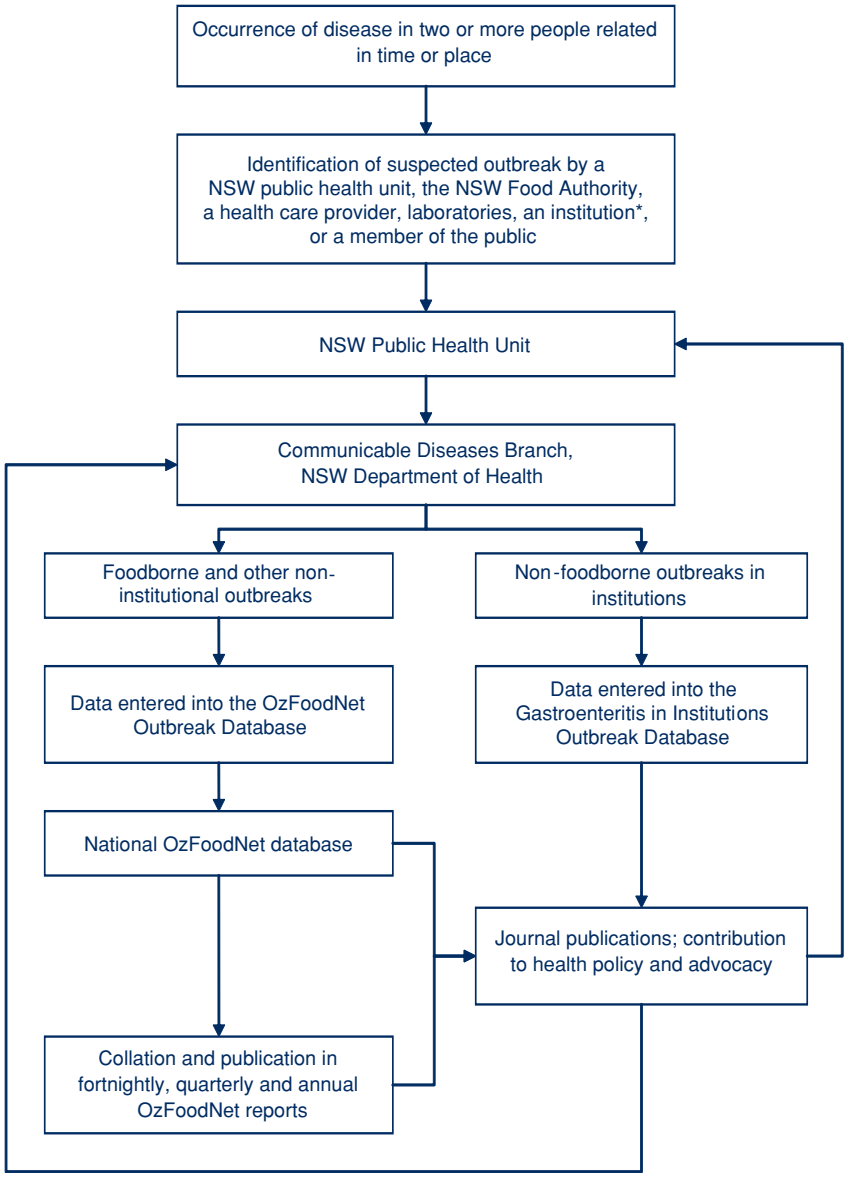

Figure 1. The flow of information relating to suspected or confirmed enteric disease outbreaks in New South Wales.
*An institution includes residential, educational, health care, childcare and correctional facilities.

Table 2. Tasks for the evaluation of a surveillance system

Task	Components
Assess the public health importance of the health event	Total number of cases, incidence and prevalence Indicators of severity, such as the mortality rate and the case-fatality ratio Preventability
Describe the components and operation of the surveillance system	What are the system objectives? What is the population under surveillance? What is the period of time of the data collection? What information is collected? Who provides the surveillance information? How is the information transferred? How is the information stored? Who analyses the data? How are the data analysed and how often? How often are reports disseminated and to whom? How are the reports distributed?
Evaluate the surveillance system attributes	Simplicity, flexibility, level of integration with other information systems, acceptability, data confidentiality and security, representativeness, completeness, sensitivity, positive predictive value, timeliness, stability
Assess the level of usefulness of the system	What actions are taken as a result of the data from the surveillance system? Who has used the data to make decisions and take action? Are there any other anticipated uses of the data?
Describe the resources used to operate the system	Direct cost of the surveillance system
Provide conclusions and recommendations	Are the system objectives met, and to what extent? What modifications or improvements could be made? Should the surveillance system continue?

Adapted from the Centers for Diseases Control and Prevention guidelines.[4,5]

process involving considerable work by staff from notifying laboratories, PHUs, CDB and the NSW Food Authority. There were few automated components to this process. The CDB enteric diseases team entered all data from outbreak summary forms received from PHUs into the relevant enteric disease outbreak database – either the OzFoodNet Outbreak Summary Database or the Gastroenteritis in Institutions Database – as there was no mechanism for PHUs to enter their own data.

Flexibility

Key informants reported that these databases only captured final summary outbreak information and provided no mechanism to track the course of outbreaks from initial identification through to completion. Some outbreaks were incompletely reported and others were not entered into the database. The system could not readily capture and organise the large amount of information generated throughout the course of an outbreak investigation in a timely fashion. The outbreaks reported ranged in size and scope. The surveillance system catered for this; however, the databases were not flexible enough to incorporate additional information where required, and data from other Australian states could not be readily accessed.

Level of integration with other surveillance and health information systems

It was not possible to determine the potential relationships between individual disease notifications in the NSW Notifiable Diseases Database and enteric disease outbreaks recorded in the enteric disease outbreak databases. In addition, cross-checking whether cases identified in outbreaks had also been entered into the NSW Notifiable Diseases Database was not possible, as information on individuals was not captured as part of the outbreak summary reporting process.

Acceptability

The CDB staff who managed and maintained the surveillance system for enteric disease outbreaks reported that it was cumbersome and labour intensive. The system for reporting enteric disease outbreaks created a large paper trail and involved many hours of data entry work, particularly during epidemic winter seasons of viral gastroenteritis. The outbreak summary forms provided by PHU staff often required interpretation by the CDB enteric diseases team, and required additional work to gather missing information where forms were incomplete.

Data confidentiality and security

Completed outbreak summary forms were mailed, sent by secure fax or emailed to the CDB. Individual case identifiers were not included in the data forms; information was reported in summary form only. Information on individual institutions and businesses with suspected or confirmed outbreaks was included, highlighting the need to ensure security of data at all times. With no system for tracking outbreaks, and the large paper, email and fax trails generated by the outbreak reporting system along the numerous reporting pathways, there was considerable potential for information to be misplaced or lost.

Representativeness

The true representativeness of the surveillance system could not be evaluated because this can only be measured through comparison to the true rate of enteric disease

Table 3. Completeness of enteric disease outbreak data field by reporting unit, New South Wales, 2000–2005

Reporting unit	Hospitalisation recorded		Deaths recorded		Onset date recorded		At least one stool sample collected	
	n	(%)	*n*	(%)	*n*	(%)	*n*	(%)
Communicable disease branch	7	(88)	7	(88)	8	(100)	5	(63)
Rural public health units								
A	26	(55)	24	(51)	45	(96)	26	(55)
B	34	(77)	34	(77)	42	(95)	29	(66)
C	19	(58)	14	(42)	33	(100)	14	(42)
D	4	(100)	4	(100)	4	(100)	3	(75)
E	12	(55)	10	(45)	22	(100)	6	(27)
F	5	(83)	6	(100)	6	(100)	4	(67)
G	8	(80)	7	(70)	10	(100)	4	(40)
H	4	(67)	4	(67)	6	(100)	3	(50)
Regional public health units								
I	101	(49)	100	(49)	201	(98)	102	(50)
J	33	(80)	32	(78)	41	(100)	5	(12)
K	13	(29)	12	(27)	45	(100)	22	(49)
Metropolitan public health units								
L	46	(52)	39	(44)	87	(98)	21	(24)
M	52	(39)	36	(27)	133	(100)	61	(46)
N	40	(82)	36	(73)	42	(86)	14	(29)
O	75	(74)	68	(67)	94	(92)	57	(56)
P	51	(50)	49	(48)	95	(92)	56	(54)
Q	25	(68)	24	(65)	34	(92)	11	(30)
Total	556	(56)	507	(51)	951	(95)	445	(45)

Pre 2005 Area Health Service boundaries.
Source: Gastroenteritis in Institutions Database; OzFoodNet Outbreak Summary Database.

outbreaks in NSW, which is not known. An approximate assessment of representativeness can be made by comparing the rates of enteric disease outbreaks across different areas of NSW, assuming that the occurrence of enteric disease outbreaks do not vary by location. The population-adjusted public health unit total rate of reporting of enteric disease outbreaks was highly variable, ranging from 0.5 to 7.4 per 100 000 population.[6]

Completeness

The completeness of the surveillance system was judged by assessing the proportion of critical data fields that were completely collected for all outbreaks during the period of the evaluation. The outbreak data were almost 100% complete for the date of onset of the first case and the number of people affected. Completeness for other important fields, such as the number at risk, the number hospitalised, the number of deaths, the number of stool specimens and the summary report date, was variable both over time and between public health units (Table 3). In addition, CDB staff reported that it was common for data on the environmental component of foodborne disease investigations to be incomplete, and that they were unable to monitor the completeness of outbreak reporting or be sure that they had received all essential outbreak data.

During 2005, final summary outbreak forms were received for 132 (63 per cent) of 209 provisionally reported outbreaks, including summary reports for 49/116 (42 per cent) of foodborne outbreaks and 83/93 (89 per cent) of institutional outbreaks. Some of the outbreaks provisionally reported may not have been considered to warrant further investigation or summary reporting by the PHU.

Sensitivity and positive predictive value

Sensitivity could not be evaluated, as data on the true total number of enteric disease outbreaks in NSW is not available. Positive predictive value could also not be formally evaluated, but all of the outbreaks reported using the final summary reporting form appeared to be true enteric disease outbreaks, as no non-enteric pathogens were identified.

Timeliness

There was inevitably a delay between the date of onset of symptoms in the first case and the final date of the outbreak summary report. The median time to summary reporting of all enteric outbreaks over the 5-year period was 20 days (inter-quartile range 6–52 days). For foodborne outbreaks, the median time to a summary report was 32 days (inter-quartile range 10–105 days), and for non-foodborne outbreaks 19 days (inter-quartile range 6–47 days). The time to

summary reporting of foodborne outbreaks was significantly longer than for non-foodborne outbreaks ($p < 0.001$, Kruskal–Wallis test), although the median time to summary reporting for all outbreaks was well within the required 30 days since the last outbreak case was identified.

Stability

As the period of evaluation covered the introduction of two outbreak databases, one during early 2000 and another in 2003, the methods for surveillance and reporting of enteric disease outbreaks changed over the five-year time period investigated, both in terms of the reporting forms used, data fields collected and databases used. This meant that some data were incomplete or not completely comparable, even over this relatively short time period.

Cost of the surveillance system

The cost of the surveillance system was not assessed due to time constraints.

Public health importance of the surveillance system

Between 2000 and 2005, 998 enteric disease outbreaks were reported (148 foodborne and 850 non-foodborne), affecting 24 260 people, and associated with 771 hospitalisations and 21 deaths. The outbreaks reported during the evaluation period are described in more detail in 'Enteric disease outbreak reporting, New South Wales, Australia, 2000 to 2005' in this issue.[6]

Usefulness of the surveillance system

Despite the limitations of the surveillance system, the key informants indicated that they were able to use the enteric disease outbreak surveillance system data to produce useful information, such as: the incidence of outbreaks in NSW; assessment of the success of outbreak control efforts; identification of the probable cause of an outbreak; and identification of measures that could contribute to more effective prevention of enteric disease outbreaks.[6]

The CDB users of the system, and the results of an analysis of the information available from the enteric disease outbreak databases, indicated that the surveillance system did provide a mechanism for meeting all the objectives of the surveillance system at some level.

System products

Policy outputs of the NSW enteric disease outbreak surveillance system were difficult to identify due to an absence of a system for identifying, organising and recording policy outcomes of outbreak investigations. Products of the surveillance system that were identified through document review and interviews with key informants included:

- fortnightly, quarterly and annual NSW Health and OzFoodNet reports (published as part of the national OzFoodNet reports in *Communicable Diseases Intelligence*)
- summary institutional outbreak data published in each issue of the *NSW Public Health Bulletin* and available from the NSW Health website (www.health.nsw.gov.au)
- *ad hoc* provision of outbreak data as requested by other jurisdictions, government agencies, research institutions and industry
- information that contributed to policy development within the NSW Department of Health and the NSW

Table 4. Examples of policy outputs from enteric disease outbreak investigations, New South Wales, 2000 to 2005

Outbreak type	Policy or practice intervention
Hepatitis A in a food handler	Guidelines for operating mass post-exposure prophylaxis clinic developed by Central Sydney Public Health Unit. Tool developed by Communicable Diseases Branch to assess risk of exposure to hepatitis A or other gastrointestinal infection arising from a sick food handler.
Salmonella Typhimurium 9 and *S.* Typhimurium 126 outbreaks linked to eggs	NSW outbreak investigations contributed to an OzFoodNet report that will inform the development of the Food Standards Australia and New Zealand Primary Production and Processing Standards for the egg industry.
Salmonella montevideo outbreak linked to Egyptian tahini	Product recall, increased sampling of imports, Australian Quarantine and Inspection Service placed tahini on their risk list, international alert.
Various *Salmonella* outbreaks linked to chicken consumption	Used to inform the Food Standards Australia New Zealand Primary Production and Processing Standards for the poultry industry and industry risk assessments; increased attention from regulators; used to inform NSW Food Authority work with the poultry industry.
S. Paratyphi B bv java linked to contact with tropical fish and fish tanks	OzFoodNet developed fact sheets for the pet industry and purchasers of fish about the health risks and how to prevent infection.
S. Typhimurium 197 linked to lambs liver	Consumer education material produced by the NSW Food Authority.
Viral gastroenteritis epidemics in institutions	Development of the GASTRO PACK, formation of Aged Care Facility Outbreak Response Working Group, development of a new Viral Gastroenteritis in Institutions Reporting Form, regular media releases throughout winter viral gastroenteritis seasons.

Food Authority, and also to national foodborne illness and health policies
- information that contributed to advocacy measures, including media releases (Table 4).

Discussion

This evaluation determined that the surveillance system was performing a useful function, and was able to meet all of its pre-defined objectives to some extent. A principal limitation of the system was the inability to track the course of outbreaks efficiently and comprehensively, making central monitoring of the extent of outbreaks and the impact of control efforts difficult. In addition, the fact that enteric disease data was not linked with the NSW Notifiable Diseases Database delayed the sharing and reporting of outbreak information. A final limitation was the complicated, cumbersome and time-consuming nature of the enteric disease outbreak surveillance databases and information collation techniques, and the lack of flexibility of the data management system.

Given the absence of a system for tracking outbreaks and resultant policy outcomes, the findings presented here are likely to be incomplete. Nevertheless the system did appear to perform reasonably well with respect to prevention efforts and policy outputs. This report provides an imprecise indication of the comprehensiveness and timeliness of reporting due to the absence of a method for systematically tracking the course of an outbreak, and the absence of critical data fields, such as onset date of last case.

The sensitivity and specificity of the system could not be evaluated. Due to enhanced surveillance in the one site with the highest rates of reporting, the surveillance system is unlikely to be representative of the true distribution of enteric disease outbreaks. Furthermore, approximately 17.2 million cases of gastroenteritis occur in Australia annually, but only a minority of people with gastroenteritis go to a doctor, and only a minority of these provide a stool sample.[7] Hence, the reported rates of enteric disease outbreaks are likely to be a substantial underrepresentation of the true rates of enteric disease outbreaks in the community. The rates of enteric outbreaks reported from this evaluation were comparable with previous estimates of reported rates of enteric disease outbreaks in NSW, indicating some system stability.[8]

The evaluation itself was limited: it was not independent of the health system, and only a limited number of key informants within the NSW Department of Health were interviewed. Local PHU staff were not interviewed, and may have been able to provide important insights into the performance and usefulness of the enteric disease surveillance system. This evaluation focussed mainly on the surveillance processes within the Department, and not on the notifications to PHUs, or the PHU investigations themselves. In addition, the cost-effectiveness of the system was not evaluated.

This evaluation produced some recommendations to improve the efficiency and effectiveness of the surveillance system. Recommendations from the review, together with the development of a tender for a new notifiable diseases surveillance database system, have resulted in agreement to the following improvements to the system:
- consolidation of all disease outbreak information into one database
- linkage of enteric disease outbreak data with enteric disease data in the redeveloped state and national notifiable disease databases
- development of a mechanism to monitor the course of outbreaks and assist in comprehensive outbreak data collection.

Once the new database has been constructed and implemented, simplification and consolidation of the data collection forms will be undertaken as a matter of priority.

Conclusion

The NSW enteric disease outbreak surveillance system is serving a useful public health function and should be continued. The system could be improved through the use of more sophisticated electronic data management techniques.

Acknowledgements

Thanks to Jennie Musto for reviewing earlier versions of this document and providing valuable feedback. Thanks also to all the public health unit staff who provided outbreak summary reports during the period of the evaluation.

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The evaluation of web-based data collection for enhanced surveillance of cryptosporidiosis

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Abstract: Following an increase in the number of people diagnosed with cryptosporidiosis in November 2005, the Communicable Diseases Branch initiated enhanced surveillance using a developmental version of *NetEpi Collection*, an open-source, web-based data collection tool. We evaluated the usefulness of *NetEpi Collection* for enhanced surveillance, using the Centers for Disease Control and Prevention's Updated Guidelines for Evaluating Public Health Surveillance Systems as a guide. Most staff (73 per cent) who used *NetEpi Collection* found it easy to use. Although ongoing support was thought to be adequate by 82 per cent of respondents who used *NetEpi Collection*, 36 per cent reported that training was limited and 27 per cent reported technical problems such as internet, server and password problems. In order to improve its usefulness in enhanced surveillance, training in *NetEpi Collection* should be enhanced and the stability of the system improved.

Cryptosporidiosis is a diarrhoeal disease caused by the intestinal parasite, *Cryptosporidium*.[1] Outbreaks are common and have been associated with swimming pools, drinking water supplies and, rarely, consumption of contaminated beverages.[1] Under the *NSW Public Health Act 1991*, cryptosporidiosis is a notifiable disease and must be reported to NSW Health by laboratories. In NSW, eight public health units (PHUs) located across 16 sites are responsible for following up notifications of communicable diseases including cryptosporidiosis. PHU staff enter cases of cryptosporidiosis onto the state-wide surveillance system, the Notifiable Diseases Database (NDD), and investigate these using a standardised cryptosporidiosis questionnaire.

Establishing enhanced surveillance

In November 2005, analysis of routinely collected surveillance data by the NSW Department of Health identified an increase in the number of people notified with cryptosporidiosis. Following initial anecdotal reports that many of these cases reported contact with farms or cattle, the NSW Department of Health initiated enhanced surveillance to explore and quantify risk factors among the cases.

From 1 November 2005 to 29 May 2006 (the outbreak period), PHU staff were asked to interview all cases of cryptosporidiosis using the standard NSW Health questionnaire. The questionnaire included potential risk factors associated with cryptosporidiosis, including farm visits, preschool attendance, swimming and contact with another case. Cryptosporidiosis cases were entered into NDD in the usual way. However, as NDD does not have the capacity to capture enhanced surveillance data, staff from the Communicable Diseases Branch at the NSW Department of Health designed a database using *NetEpi Collection* software for this purpose. *NetEpi Collection* is an open-source, web-based software that has been developed by the Centre for Epidemiology and Research, at the NSW Department of Health, to collect structured information about cases and contacts through web browsers on the internet.[2]

One PHU staff member from each PHU site was asked to enter additional risk factor data and the NDD case number into the developmental version (version 0.95) of *NetEpi Collection*. Training was provided to PHU staff via a teleconference, and ongoing individual support was provided using email or telephone. Data from NDD and *NetEpi Collection* were then merged and analyses performed using SAS (version 8.2) to determine what risk factors were important in ongoing transmission during the outbreak.[3]

The aim of the present study was to assess the usefulness of a web-based data collection system (such as *NetEpi Collection*) for enhanced surveillance during a communicable disease outbreak.

Methods

We used the Centers for Disease Control and Prevention's Updated Guidelines for Evaluating Public Health Surveillance Systems as a framework to:

- assess the public health importance of the surveillance system
- assess the usefulness of the surveillance system

- describe attributes of the surveillance system (i.e. simplicity, flexibility, data quality, acceptability, predictive value positive, representativeness, timeliness and stability).[4]

Public health importance of cryptosporidiosis

A review of the public health literature was undertaken to assess the outbreak potential and preventability of cryptosporidiosis. NSW cryptosporidiosis data were analysed to compare notifications for the outbreak period with the previous five years (2000–2005).

Description of surveillance system components

Managers of NDD and *NetEpi Collection* were asked to provide a description of each system component.

Usefulness of the surveillance system

At the end of the outbreak period, one person from each of the 16 PHU sites was asked to complete a questionnaire (Box 1). The questionnaire outlined their experience of using *NetEpi Collection*, and responses were collated in Excel (version 5.0.2). Risk factor data was analysed at a state level to determine if *NetEpi Collection* was useful in assessing statewide risk factors associated with cryptosporidiosis.

Attributes of the surveillance system

Acceptability

We asked PHU staff if they used *NetEpi Collection*, whether it was useful or burdensome, and whether there were barriers to using *NetEpi Collection* in this outbreak. We also asked about the adequacy of training and ongoing support provided by the Communicable Diseases Branch.

Stability

To determine the system's ability to manage data without failure, PHU staff were asked about any technical difficulties experienced while using *NetEpi Collection*.

Quality and representativeness of data

A comparison of the cryptosporidiosis data in *NetEpi Collection* and NDD was used to determine the completeness of *Net Epi* data and whether it was representative of all cases. This allowed us to determine which PHUs entered data into *NetEpi Collection* and what was different about the cases that were not entered. We also examined the completeness of data in the risk factor fields in *NetEpi Collection*.

Box 1. Items addressed in the questionnaire

* Did PHU staff use *NetEpi* Collection?
* Was *NetEpi* Collection easy to use?
* Could PHU staff extract and use data to assess local risk factors associated with cryptosporidiosis?

Results

Public health importance of cryptosporidiosis

In NSW and elsewhere, cryptosporidiosis has been linked to swimming in contaminated swimming pools.[5–7] Although outbreaks associated with drinking water supplies are rare in Australia, there is the potential for large outbreaks due to contaminated water sources, such as the Milwaukee outbreak of 1993.[8,9] Early detection of cryptosporidiosis outbreaks may help public health attempts to develop interventions aimed at preventing further disease.

Between 1997–2006, the number of cryptosporidiosis notifications in NSW has ranged from 1130 in 1998 to 121 in 1999, with an average of 423 cases per year. In the outbreak period, a total of 871 cases of cryptosporidiosis were reported to NSW Health. During the same time period in the previous five years, there was average of 235 cases of cryptosporidiosis reported each year.

Components of the surveillance system

Surveillance of cryptosporidiosis begins when a person with symptoms visits a doctor, and a faecal specimen is ordered and sent to a laboratory for testing. If the result is positive, the laboratory notifies the local PHU where the case is entered into the NDD and the person interviewed for risk factors. PHU staff review these data to identify clustering of cases (time and location) and, where necessary, initiate control measures following the *NSW Health Cryptosporidiosis Response Protocol for NSW Public Health Units*.[10] Data are then available for analysis by the Communicable Diseases Branch, and reports are compiled for the *NSW Health Public Health Bulletin* and NSW Health website.[11,12]

The NDD is a secure, decentralised database used for storing information on cases of notifiable diseases in NSW.[13] Cryptosporidiosis data entered into NDD includes demographic variables, disease characteristics, disease outcome, laboratory information, organism, specimen type, identification method and other variables. Risk factors are not entered into NDD, but NDD does contain a 'clinical notes' text field, where risk factors for disease may be entered.

During the outbreak period, specific risk factor information was entered into the *NetEpi Collection* database. This database contained a unique identifier common to NDD and *NetEpi Collection*, as well as fields on risk factors for cryptosporidiosis such as: swimming; contact with another case; preschool attendance; and farm visit. PHU staff entered the common identifier and risk factor data into the *NetEpi Collection* database after entering the case into NDD in the usual manner.

Usefulness of the surveillance system

Just over two-thirds of PHU sites reported using *NetEpi Collection* during the outbreak. Of those who used *NetEpi*

Table 1. Responses to questions on enhanced surveillance using *NetEpi Collection* from 11 Public Health Unit staff located across NSW, during a statewide cryptosporidiosis outbreak

Question	'yes' responses		Reasons given for a 'no' response
	n	%	
Was *NetEpi* easy to use?	8	73	Unable to attend training Internet/technical problems
Was training adequate?	7	64	Unable to attend training
Was ongoing support adequate?	9	82	Not sure how to access data (i.e. for export and analysis)
Any technical difficulties?	7	64	Internet problems. Server crashing. Unable to use return key. Problems recalling password
Did you export and analyse data?	3	27	Didn't try to export
Did you assess local risk factors using your data?	2	18	Not applicable
Barriers to using *NetEpi* for analysis?	Not applicable		Time. Small number of cases. Not sure how to export and analyse data. General difficulties in using *NetEpi*. Trouble accessing website/ internet connectivity
Would it be helpful if *NetEpi* was incorporated into NDD?	10	91	Depends on ease of use
Was enhanced surveillance useful?	9	82	Analysis not carried out so not sure of usefulness
Was enhanced surveillance burdensome?	5	45	Time

Collection, most found it easy to use (Table 1). Just under one-third of those who used *NetEpi Collection* exported data for analysis and the data were infrequently used to assess local risk factors associated with cryptosporidiosis.

At the statewide level, Communicable Diseases Branch staff thought that *NetEpi Collection* provided useful information on risk factors, in particular the geographic distribution and timing of exposures.[14] Analysis of statewide data entered into *NetEpi Collection* revealed that the outbreak may have started in rural areas of NSW with farm animal contact as a risk factor. The outbreak then appeared to involve spread by contact to other parts of NSW, with person-to-person contact and swimming pool contamination becoming more important as risk factors.[14]

Attributes of the surveillance system

Acceptability

Of the 11 PHU staff who used *NetEpi Collection*, 9 (82 per cent) found it useful because it provided: an improvement of surveillance; an analysis of disease trends; and assistance in determining clusters and risk factors. Time was a factor for those who found the system burdensome, and barriers to using *NetEpi Collection* for analysis ranged from technical difficulties to lack of training (Table 1).

While training was perceived to be adequate by more than half of the users, most reported that they did not analyse the data collected through *NetEpi Collection* (Table 1). Two people who thought that training was not adequate were unable to attend the initial training session.

Stability

PHU staff experienced technical difficulties using NetEpi Collection (Table 1). These included: difficulty with accessing the internet; occasional server/program crashes; slow internet connection; and an inability to use the return key to move between data entry fields (the tab key needs to be used instead). Some respondents mentioned that problems had been fixed after liaison with staff from the Communicable Diseases Branch.

Quality and representativeness of data

At June 2007, a total of 846 cases of cryptosporidiosis were reported for the outbreak period (Table 2). Of these, 458 (54 per cent) were entered into *NetEpi Collection* (Table 2). In the risk factor fields in *NetEpi Collection*, the data fields were, on average, 86 per cent complete.

Lack of time meant that it was not possible to enter data into *NetEpi Collection* at all PHU sites (5/16 or 31 per cent). Cases entered into *NetEpi Collection* were similar to the NDD cases with respect to gender and age ($p = 0.073$ and 0.900 respectively) (Table 2). A larger proportion of cases from metropolitan Sydney were entered into *NetEpi Collection* than from other parts of NSW ($p = 0.045$) and a higher proportion were entered at the beginning of the outbreak than later in the outbreak ($p = 0.000$).

Discussion

In an outbreak setting, enhanced surveillance of cryptosporidiosis using a web-based data collection system allowed identification of risk factors for disease that could not have been achieved using NDD alone. Information describing risk factors also allowed identification of area of residence of those potential exposures over time.

PHU staff infrequently exported and used *NetEpi Collection* data to assess local risk factors for transmission. No comparison can be made with using the NDD system in this way. Nevertheless, despite time and techno-

Table 2. Number and proportion of cryptosporidiosis cases entered and not entered into *NetEpi Collection*, 1 November 2005 to 29 May 2006

Cases	Entered into *NetEpi*		Not entered into *NetEpi*		*p* value
	n	%	*n*	%	
Gender					
Male	219	48	190	49	
Female	237	52	194	50	0.073
Unknown	2	<1	4	1	
Age					
0–4 years	188	41	161	41	0.900
>5 years	270	59	227	59	
Residence					
Metro Sydney	231	50	168	43	0.045
Remainder of NSW	227	50	220	57	
Report date					
1 Nov–31 Dec 2005	169	37	66	17	<0.001
1 Jan–29 May 2006	289	63	322	83	
Total	458	100	388	100	

logical limitations, most PHU staff thought that using enhanced surveillance would help in determining clusters and risk factors.

The information entered into *NetEpi Collection* was useful for analysing risk factors at a statewide level. Such web-based data collection systems may offer greater advantages for the co-ordinating site, as they enable analysis of risk factor information for the whole dataset. The use of web-based data collection tools may also be a more streamlined method of data collection than other methods (such as faxing or emailing single databases to a central point) and so could prevent time delays and errors in data transcription. However, where broadband access is unreliable or slow, the use of web-based data collection tools may be limited.

The observed decreasing compliance with entering data into *NetEpi Collection* suggests that enhanced surveillance may be more sustainable over short periods.

To improve the use of web-based data collection tools, both training and support with technical problems should be improved. This could include repeating introductory training sessions and providing training in data analysis. In addition, PHU sites require adequate and reliable access to the internet.

With regards to the system itself, the stability needs to be improved and the data entry interface should allow staff to use the return key to move between fields when entering data. The authors of *NetEpi Collection* have advised that version 1.0 of the software, which includes numerous enhancements, will be publicly available at http://www.netepi.org when this paper is published.

In previous outbreak investigations carried out by NSW Health, risk factor information has been collected by faxing forms to a central point, then entering into a database. Elsewhere in Australia, Canada and the United Kingdom, enhanced surveillance has been used to collect risk factor data for a range of diseases in outbreak situations. This approach has been recently applied to the analysis of risk factors for hepatitis B,[15,16] meningococcal disease,[17] haemolytic uraemic syndrome,[18] hepatitis C[19] and campylobacter,[20] among other diseases. OzFoodNet have used an even earlier developmental version of *NetEpi Collection* during an outbreak of *Salmonella* Hvittingfoss in order to identify potentially implicated foods and the investigators concluded that using a web-based data collection system such as *NetEpi Collection* was a dramatic improvement in the collection of data in a geographically dispersed outbreak.[21]

Conclusion

Enhanced surveillance of cryptosporidiosis provided useful information on potential exposures during an outbreak with widespread geographic distribution. Using the web-based data collection tool, *NetEpi Collection*, made analysis of data easier for the co-ordinating site, but was difficult and time-consuming for some PHU staff.

Where systems data are complete and representative, enhanced surveillance during outbreaks using web-based data collection tools can provide useful information about exposures for disease.

Acknowledgements

Many thanks to Tim Churches (Population Health Information Branch, NSW Health) for advice on the manuscript and to the Public Health Unit network for their co-operation during the outbreak and in returning questionnaires.

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Investigation of an outbreak of acute illness in a school group visiting Sydney, September 2006

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Abstract: Objective: We describe the investigation into an outbreak of acute illness in approximately 40 people attending Darling Harbour in Sydney during a school music camp. **Methods:** We used three methods, including the Public Health Realtime Emergency Department Surveillance System, to obtain information on the food and travel history of the group and symptoms of the cases rapidly. **Results:** Forty-five cases of gastroenteritis were identified in people on the bus trip. Most dates of onset of illness were obtained from triage text fields in the NSW Public Health Real Time Emergency Department Surveillance System, and were verified through medical record review and interviews. No causative agent was identified. **Conclusion:** The investigation suggested person-to-person transmission rather than a point source, and demonstrates how the NSW Public Health Real Time Emergency Department Surveillance System can assist with case finding in public health investigations.

In September 2006, NSW Health was notified that up to 40 children on a school bus trip from Queensland were acutely unwell with gastroenteritis. The group was visiting Darling Harbour (a tourist precinct adjacent to the central business district of Sydney) and group members affected were being transported by ambulance to three hospitals in inner Sydney. Preliminary reports suggested that the students might be suffering from a large scale acute poisoning incident. Public health authorities needed to obtain information on the cases rapidly in order to determine a possible cause.

This report describes the features of the public health investigation and the methods used to assess the incident quickly.

Methods

Three methods were used to obtain information rapidly on the food and travel history of the group, and on the symptoms of affected people in a case series.

Case definition for this investigation was: *any person travelling on the bus trip who experienced one or more of the following symptoms between 16 and 29 September 2006*: *vomiting*, *nausea*, *diarrhoea*, *stomach cramps or abdominal pain*.

We interviewed a senior teacher about the travel and food history of the group. Data collected included information on where the group stayed, the meals consumed and the symptoms reported by students, teachers and drivers. Cases occurring before 16 September were identified, including several cases in students whose illness had resolved and who therefore did not present to any of the three Sydney hospitals. The recreation camp was inspected to investigate the possibility of food or water-borne transmission.

We reviewed medical records and interviewed clinicians regarding cases that presented to Hospital 1 to obtain onset dates and symptoms. At Hospital 2, we conducted a medical record review and brief interviews with each affected person. At Hospital 3, the NSW Public Health Real Time Emergency Department Surveillance System (PHREDSS) was used to obtain information recorded in nursing triage notes swiftly.[1] The triage text field of the PHREDSS included details on disease onset and symptom descriptions, allowing verification of the history of known cases presenting to Hospitals 1 and 2. Further information on cases presenting to Hospital 3 was then obtained by reviewing medical records.

Stool samples were taken from two people who were treated at Hospital 2. These samples were examined by faecal microscopy, bacterial culture and viral studies, including testing for rotavirus and norovirus using both enzyme immunoassay and reverse transcriptase polymerase chain reaction testing.[2]

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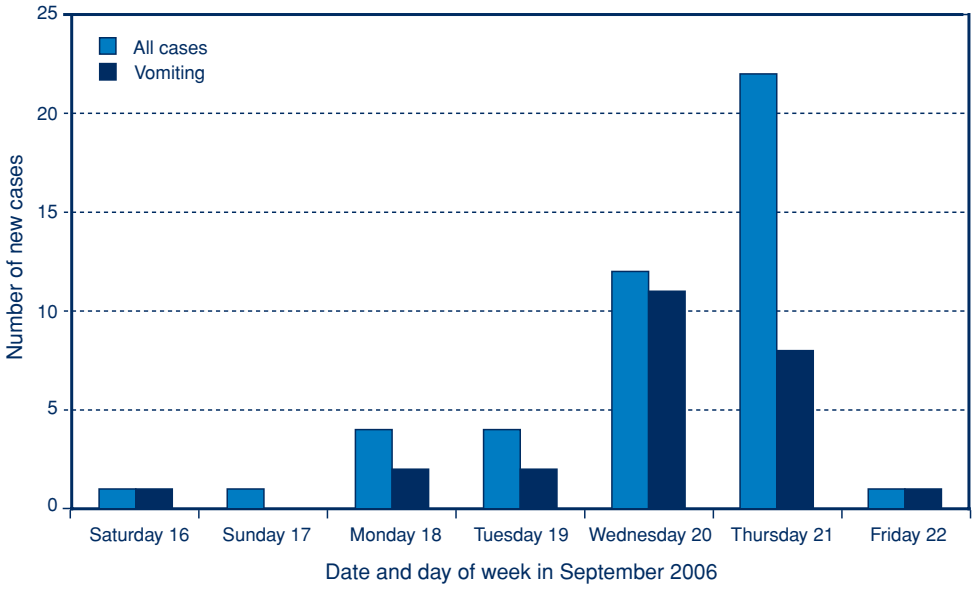

Figure 1. Onset of symptoms among all cases of illness and only those cases reporting vomiting in an outbreak of gastroenteritis in a school music camp, September 2006.
Source: PHREDSS, interview and medical record review.

Active surveillance for additional cases through contacting teachers continued up until 29 September. Data were analysed in MS Excel.

Results

History of the group obtained by interview

The students were on a two-week band camp organised through schools in southern Queensland. One hundred school students aged 11 to 18 years were travelling on two buses accompanied by six teachers and four bus drivers. The tour departed from Queensland on 15 September, arriving the next day at a recreation camp in rural NSW. The students stayed at the camp from 16–20 September 2006 and visited Sydney for one day en route to Queensland.

Case identification

Forty-five cases of gastroenteritis were identified in people on the bus trip, including two teachers, two bus drivers and 41 students. Most onset dates were obtained from triage text fields in PHREDSS and verified through medical record review and interviews. Seven additional cases were identified through interviews with the teacher. The first case occurred on 16 September after the group arrived at the camp and the last case occurred on 22 September (Figure 1). The overall attack rate was 41 per cent (45 /110): 41 per cent (41/100) in students; 33 per cent (2/6) in teachers; and 50 per cent (2/4) in drivers. Thirty-nine people fitting the case definition presented to one of three hospitals.

The most commonly reported symptom was vomiting and/or nausea ($n = 34$, 76 per cent) (Table 1). Among the 25 people reporting vomiting, the peak of the epidemic curve occurred on 20 September (11/25 cases, 44 per cent) (Figure 1).

Food and exposure history

The teacher reported that a variety of takeaway food was consumed at meals en route, except for a barbeque breakfast on 16 September. During the stay all meals were supplied by the camp, with the exception of a barbeque dinner on 19 September. During this meal volunteers cooked meats and students served their own salads from a self-serve area.

Students stayed in shared cabins of between four and eight occupants. Each cabin had its own bathroom.

It was reported that at least one student who became ill early on vomited in shared accommodation, where other students were present. A subsequent case of gastroenteritis occurred in a person who had cleaned vomitus without gloves or other protection.

Table 1. Symptoms experiences by 45 people travelling on a school music camp between 16 and 22 September affected by gastroenteritis

Symptom	*n*	%
Nausea	34	76
Abdominal pain or cramp	29	64
Vomiting	25	56
Diarrhoea	17	38
Headache	11	24
Fever	3	7

Source: teacher interview, case interviews, case notes and public health real-time emergency department surveillance system (PHREDSS) triage text.

The recreation camp was inspected on 27 September. The camp is supplied with bore water supplemented with rainwater. Drinking water is filtered, softened and chlorinated. Regular tests recorded by camp staff and an independent laboratory indicate that the drinking water complies with drinking water guidelines.[3] Samples collected at the site visit also complied with these guidelines.

Laboratory investigation

All stool samples were negative for any pathogen.

Public health action

We counselled teachers and students on appropriate infection control measures, including thorough hand washing and cohorting sick people. Teachers were advised against travelling back to Queensland as planned on the evening of 21 September. Emergency accommodation was arranged for the school group in Sydney and a registered nurse was provided to assist with infection control. The group departed by bus on 22 September.

We alerted public health staff in Queensland and the local hospital emergency department of the outbreak before the return of the bus. Active surveillance (to 29 September) identified one additional case with an onset of 22 September.

Discussion

Despite early reports suggesting an aetiology of a more sinister nature, the epidemiological and symptom profile of this outbreak is consistent with an outbreak of viral gastroenteritis, probably norovirus, with person-to-person transmission.

Initially person-to-person transmission was suspected. Due to the large number of people presenting to emergency departments across Sydney, significant media attention and the fact that the children were 'stranded', an urgent public health response was necessary. A retrospective case series was used to obtain information rapidly, describe the outbreak and inform public health action in a timely manner. This strategy was limited by incomplete case ascertainment at the time – a problem overcome by using real-time emergency department surveillance and medical record review.

Emergency department surveillance systems are usually designed as outbreak detection tools. In addition, PHREDSS was used to provide and verify case details after the outbreak of gastroenteritis had been notified.

No causative agent was identified in this investigation. This may have occurred because insufficient samples were collected or the samples were inadequate.[4]

Norovirus was considered a pathogen likely to be responsible for this outbreak because:

- vomiting and/or nausea were the most commonly reported symptoms in this outbreak
- most cases resolved quickly after onset (characteristic of norovirus)[5,6]
- norovirus is a common cause of gastroenteritis and gastroenteritis outbreaks in older children and adults[6–11]
- the attack rate in this outbreak is typical of outbreaks of norovirus in institutional settings.[12–14]

The epidemic curve for this outbreak is consistent with person-to-person transmission, with a small number of early cases followed by a rapid increase in cases.[13,14] Probable settings of viral transmission, including shared accommodation, transport and self-service meals, were identified during the interviews. Person-to-food-to-person transmission may have also been involved in this outbreak.

At the peak of the epidemic (on 21 September), reported symptoms may have been exaggerated due to the stress of the incident. Several people presenting to emergency departments experienced only one non-specific symptom: nausea, headache or abdominal pain. The peak of the epidemic curve for those who experienced vomiting actually occurred one day earlier, on 20 September. The outbreak may have been subsiding by the time the mass transfer of cases to hospitals occurred on 21 September.

Conclusion

This outbreak required significant mobilisation of emergency service, hospital and public health resources. The public health response required communication between two public health units and the NSW Department of Health, as well as with other agencies involved in the provision of emergency assistance to the travelling party. The coordination of the rapid response was aided by the additional data gained from PHREDSS. Such emergency department surveillance systems provide valuable information in public health investigations. This incident could be viewed as a test of the health response to large incidents, such as pandemic influenza and bioterrorism.

Acknowledgements

We wish to acknowledge the assistance of the following people: public health unit staff in Greater Southern Area Health Service and Chris Oxenford, OzFoodNet epidemiologist, Australian Capital Territory for assistance with the investigation in Sutton and Canberra; and public health unit staff in Sydney South West and South Eastern Sydney Illawarra Area Health Services for assistance with the investigation in Sydney.

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Salmonella typing in New South Wales: current methods and application of improved epidemiological tools

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Abstract: Salmonellosis caused by enteropathogens of the genus *Salmonella* is a major public health concern in Australia. Serotyping is usually performed in enteric reference laboratories for the initial characterisation and differentiation of *Salmonella* species. Further strain identification within serovars may be achieved by phage typing and this is used as an epidemiological tool for outbreak investigations. Phage typing has limited discriminatory ability and the necessity of sending specimens interstate from NSW for this test causes delays in recognising outbreaks and reduces the likelihood of identifying the source. Multilocus variable-number tandem-repeat analysis has a high discriminatory power and faster turnaround time, and is the method of choice for outbreak investigation. Additionally, a newly developed multiplex PCR-based reverse line blot hybridisation system is able to identify most of the phage types prevalent in NSW. Combining these last two molecular methods will significantly enhance outbreak investigations and surveillance of *Salmonella* infections in NSW.

Salmonella is a genus of Gram-negative bacteria in the family *Enterobacteriaceae*. The classification of salmonellae is confusing and controversial, but most belong to a single species, *Salmonella enterica*, which is divided into more than 2500 serovars.[1] Many of these cause human salmonellosis, a gastrointestinal illness of significant public health importance. Salmonellosis commonly presents with diarrhoea, headache, abdominal cramps, fever, nausea and vomiting. A small percentage of infections are more invasive and salmonellae can be isolated from blood, urine or other extra-intestinal sites. Salmonellae also infect or colonise many animal species and most cases of human salmonellosis are acquired from raw, undercooked or contaminated animal products.[2–4]

The most virulent serovars are *S. enterica* serovar, Typhi and *S.* Paratyphi A and B, the aetiological agents of enteric fever (typhoid and paratyphoid fever respectively).[5] Salmonellae constitute the largest group of notifiable enteric pathogens reported in NSW, despite the likelihood of cases being grossly underreported.[6] Between 1991–2006, an average of 1800 cases were notified in NSW each year (or 12 to 48 cases per 100 000) and these probably represent less than 10 per cent of cases.[6,7]

Laboratory diagnosis and culture identification

Laboratory diagnosis of salmonellosis is usually made through the culture of stools. Specimens are inoculated onto selective media (xylose-lysine-decarboxylase agar, [CHROMagar, Paris] and selenite-F [Difco, Le Pont du Claix, France]) and the salmonella-like isolates are identified.[8,9] Blood cultures are also performed for suspected enteric fever and other invasive salmonellosis, and in septic cases where enteric fever or invasive salmonellosis can be the cause but usually are not suspected at presentation. Biochemical tests to identify isolates are often performed by commercial bacterial identification systems. These systems provide automated species identification and antibiotic susceptibility testing, but are less reliable for enteric pathogens than the conventional methods of individual tube biochemical tests and disc diffusion susceptibility testing. In a recent internal review of results generated by the Phoenix 100 (Becton Dickinson, Franklin Lakes, NJ), 15 per cent of 84 salmonella isolates were initially incorrectly identified (unpublished data, Centre for Infectious Diseases and Microbiology).

In NSW, up to 100 public and private diagnostic laboratories submit *Salmonella* cultures for serotyping to the NSW Enteric Reference Laboratory at the Centre for Infectious Diseases and Microbiology Laboratory Service. All isolates are first tested to confirm their identity. The traditional tube methods of biochemical testing are the gold standard for reference laboratories.[9]

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Salmonella typing in NSW

Although serotype identification is not required for patient management, it is an important epidemiological tool for outbreak investigations and surveillance. For common serovars, further subtyping is needed and phage typing is currently most commonly used. Serotyping and phage typing are the basis of *Salmonella* reporting to the National Enteric Pathogens Surveillance Scheme. In NSW, results are reported electronically to the NSW Department of Health.

Serotyping

Serotyping involves the use of specific diagnostic sera and slide agglutination, the results of which are read in a light box. The Kauffman and White *Salmonella* classification scheme is based on cell wall (O) and flagellar (H) antigens and, for a few serovars (notably *S.* Typhi and *S.* Dublin), a capsular (Vi) antigen.[10] Common O antigens are the basis of serogroups and subgroups. Flagellar antigens create greater serotype diversity, and are expressed as phase I, phase II or phase I and II. Various combinations of the numerous O and H antigens contribute to over 2500 serovars, each of which can be described by a 'formula' based on these antigens – for example: *S.* Typhimurium is (O)1,4,5,12:(H)i:1,2; *S.* Enteritidis is 1,9,12: g,m:-; and *S.* Typhi is 9,12,Vi:d:-.[1,10]

There were 1500–2000 human isolates typed by the NSW Enteric Reference Laboratory for each year between 2001 and 2005, representing 86–104 serovars. The majority of cases of human salmonellosis are caused by a small number of serovars. The 10 most common serovars identified by the NSW Enteric Reference Laboratory are listed in Table 1, and account for 75–80 per cent of isolates. *S.* Typhimurium accounts for 50–55 per cent of all cases in NSW. Other common serovars include *S.* Enteritidis, *S.* Virchow and *S.* Infantis (Table 1).

Phage typing

Bacteriophages are viruses that may be present in certain bacteria without causing damage, but that under certain circumstances, or when applied to a susceptible bacterial culture, can kill (or lyse) the bacteria. Bacteriophage (phage) typing refers to a standard method of characterising selected *Salmonella* serovars. It is based on patterns of lysis and uses an international set of phages. The phage typing scheme for *S.* Typhimurium was developed 30 years ago by Anderson et al. and is still widely used.[11] Two laboratories perform phage typing on a small number of serovars for NSW: the Microbiological Diagnostic Unit, Melbourne (*S.* Typhimurium, *S.* Typhi, *S.* Paratyphi A and B including bioser java, *S.* Enteritidis, *S.* Virchow, *S.* Hadar); and the Australian Salmonella Reference Centre, Adelaide (*S.* Heidelberg, *S.* Bovismorbificans).

Phage typing is based on the fact that most *Salmonella* strains are infected with one or more bacterial viruses

Table 1. Ten most common *Salmonella* serovars isolated from humans and identified in the NSW between 2001 and 2005

n = denotes number of records for that year. The percentage of each *Salmonella* serovar for the year is given in brackets.

Ranking	2001 (*n* = 1590)	2002 (*n* = 1802)	2003 (*n* = 1774)	2004 (*n* = 2029)	2005 (*n* = 2047)
1	*S.* Typhimurium (51.2%)	*S.* Typhimurium (51.9%)	*S.* Typhimurium (52.1%)	*S.* Typhimurium (56.1%)	*S.* Typhimurium (53.1%)
2	*S.* Enteritidis (4.4%)	*S.* Bovismorbificans (4.6%)	*S.* Infantis (5.8%)	*S.* Enteritidis (4.1%)	*S.* Enteritidis (5.2%)
3	*S.* Birkenhead (4.1%)	*S.* Virchow (4.5%)	*S.* Virchow (3.7%)	*S.* Virchow (4.0%)	*S.* Virchow (3.7%)
4	*S.* Virchow (3.5%)	*S.* Montevideo (3.6%)	*S.* Bovismorbificans (2.6%)	*S.* Infantis (2.5%)	*S.* Infantis (2.6%)
5	*S.* Bovismorbificans (3.5%)	*S.* Enteritidis (3.1%)	*S.* Chester (2.6%)	*S.* Bovismorbificans (2.2%)	*S.* subsp. 1 ser 16: lv:- (2.1%)
6	*S.* Stanley (3.4%)	*S.* Infantis (2.2%)	*S.* Saintpaul (2.2%)	*S.* Typhi (1.9%)	*S.* Bovismorbificans (1.8%)
7	*S.* Infantis (2.4%)	*S.* Potsdam (2.1%)	*S.* Enteritidis (2.1%)	*S.* Saintpaul (1.8%)	*S.* Saintpaul (1.6%)
8	*S.* Saintpaul (2.1%)	*S.* Saintpaul (1.9%)	*S.* Singapore (1.3%)	*S.* subsp. 1 ser 16: lv:- (1.7%)	*S.* Typhi (1.5%)
9	*S.* Singapore (1.4%)	*S.* Birkenhead (1.8%)	*S.* subsp. 1 ser 16: lv:- (1.9%)	*S.* Chester (1.4%)	*S.* Stanley (1.4%)
10	*S.* Typhi (1.4%)	*S.* Agona (1.6%)	*S.* Montevideo (1.7%)	*S.* Singapore (1.3%)	*S.* Hvittingfoss (1.3%)
No. of Serovars	86	98	95	102	104

Source: Enteric Reference Laboratory's *Salmonella*/EPS database, Centre for Infectious Diseases and Microbiology Laboratory Services.

known as bacteriophages (or phages), which either remain, silently, in the bacterial cell or lyse it and are released. The susceptibility of a particular *Salmonella* strain to infection and lysis by different phages varies according to which phages it already contains. For example, for *S.* Typhimurium a set of 34 phages is used to identify 207 phage types. Phages are 'spotted' on to a lawn culture of bacteria and incubated overnight. 'Punched out' areas without growth, in the otherwise even lawn culture, indicate lysis of the salmonella strain by the corresponding phage. The combination of phages to which a particular *Salmonella* isolate is susceptible determines its phage type.

Standard phage typing sets are maintained at the Central Public Health Laboratory of the Health Protection Agency in the United Kingdom. Historically, the sets have been made available to a limited number of reference laboratories, usually only one per country. The need to refer isolates interstate for phage typing after serotyping has been performed causes potential delays of 2–4 weeks before results are available. In addition, phage typing has limited discriminatory power for some serovars, including *S.* Typhimurium, and some isolates are non-typeable or the pattern they produce does not fit any recognised phage type. When this occurs, it is reported as RDNC: reacts does not conform.

There are numerous potential delays in the process of identification and typing of salmonellae, and 4–5 weeks may elapse from the time of consuming contaminated food, until results required for public health action are available (see Box 1, for *S.* Typhimurium). As a result, the chances of obtaining a reliable food history and identifying food sources are very low. This is a particular problem for common serovars like *S.* Typhimurium, for which it is difficult to identify outbreaks against a background of high endemicity.

Molecular typing: the future of Salmonella *typing?*

Many molecular typing methods have been used for further discrimination of *Salmonella* serovars and phage types, but these methods are generally slow and expensive. Molecular typing has variable reproducibility and discriminatory power but an advantage is that it can be performed by larger laboratories, so there is no need for interstate referral.

Pulsed-field gel electrophoresis has been widely used and is regarded as the 'gold standard' for *Salmonella* genotyping.[12] It is the basis for the US Centers for Disease Control and Prevention's 'PulseNet', an international surveillance system for *Salmonella* and other foodborne pathogens. However, pulsed-field gel electrophoresis is time-consuming, its ability to distinguish subtypes within *S.* Typhimurium is limited and comparison of results between laboratories and over long time periods requires painstaking standardisation of methods and expensive image-recognition software. Amplified-fragment length polymorphism is more discriminatory but, like pulsed-field gel electrophoresis, is technically difficult, slow, expensive and requires specialised equipment.[13,14] Multilocus sequence typing is expensive and has limited discriminatory ability because it uses highly conserved housekeeping genes.[15]

Recently multilocus variable-number tandem-repeats analysis (MLVA) has been developed for various *Salmonella* serovars, including *S.* Typhimurium, and has the potential to become the method of choice in many laboratories (for more detail on MLVA see Gilbert, 'Using MLVA to type strains of *Salmonella* Typhimurium in New South Wales' in this issue).[16–20] This technique has a high discriminatory power to differentiate *Salmonella* strains within phage types. Even genetically homogenous phage types such as *S.* Typhimurium definitive type (DT) 104 can be differentiated.[21] NSW introduced this method for routine typing of *S.* Typhimurium in May 2006 and, by November 2007, over 1500 isolates, comprising more than 60 phage types, had been typed using this method. More than 400 MLVA types and dozens of outbreaks have been identified. The largest outbreaks include *S.* Typhimurium outbreaks in a catering college in the Blue Mountains area and another in a hot bread shop in Homebush, in which more than 300 patients were involved (unpublished data; Centre for Infectious Diseases and Microbiology-Public Health, *Salmonella* outbreak molecular typing report, November 2006–May 2007). In addition, MLVA was used to investigate a nationwide *S.* Saintpaul outbreak in 2006.

The challenge posed by *S.* Typhimurium and the need to develop a practical, cost-effective, rapid strain typing system is the rationale for a post-doctoral research project at Centre for Infectious Diseases and Microbiology (CIDM) Public Health. For simultaneous detection, identification and typing of *S.* Typhimurium isolates, we have developed a multiplex PCR-based reverse line blot hybridisation system. The system is based on known phage sequences and phage type-specific amplified-fragment length polymorphism fragments.[13,22–24] Most common *S.* Typhimurium phage types can be identified by their reverse line blot patterns. Preliminary testing of the system with 168 selected *S.* Typhimurium isolates (representing 46 phage types), produced 102 reverse line blot patterns.

This method has a discriminatory power similar to that of MLVA and is suitable for epidemiological investigation of outbreaks. The reverse line blot data are stored as digital profiles and data libraries can be set up so that reverse line blot patterns can be compared historically and geographically. Once sample DNA is extracted, results are available within 24 hours. The multiplex PCR-based reverse line blot hybridisation system has been applied in several out-

break investigations in NSW. It rapidly identified human outbreak isolates and isolates from suspect food sources, and successfully predicted the phage type involved more than a week before the phage typing results were available.

In addition, the multiplex PCR-based reverse line blot hybridisation system allows a total of 43 samples to be tested in a single run and, with current available resources, up to two runs can be performed each week. In future, this typing method can be further expanded by adding more gene markers to the system. This will improve its discriminatory power and provide a genetic marker base for further development of a microarray system that is capable of simultaneously identifying serovar and phage type, and distinguishing different *Salmonella* strains.

Conclusion

A major focus in the investigation of food poisoning is timeliness in obtaining a laboratory result and the usefulness of the result for identifying or pinpointing the likelihood of an outbreak. Serotyping still remains a useful initial tool for rapid differentiation of broad groups of *Salmonella* into serovars. The results of our outbreak-based molecular studies strongly suggest that due to its limited discriminatory ability, phage typing will eventually be replaced by reliable molecular typing methods. This will also overcome the delays and cost of sending cultures to interstate reference laboratories. The combination of current MLVA typing and the new multiplex PCR-based reverse line blot hybridisation system will become the method of choice for improving outbreak investigation and surveillance, and will lead to better foodborne disease control in NSW.

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Using MLVA to type strains of *Salmonella* Typhimurium in New South Wales

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Summary: Phage typing has been the traditional strain typing (or 'fingerprinting') method used in Australia for surveillance of common salmonella serovars (such as *Salmonella* Typhimurium) and outbreak investigations. The need for more accessible, discriminatory and objective methods has been recognised but, until now, none has been widely accepted. Recently, the molecular typing method, known as MLVA (multilocus variable number tandem repeat analysis), has been applied to several *Salmonella* serovars and promises to provide faster strain typing and cluster identification than phage typing, with comparable or better sensitivity. The present article is intended as a short primer on MLVA typing, which has recently been introduced into routine use at the New South Wales Enteric Reference Laboratory at the Centre for Infectious Diseases and Microbiology, Institute of Clincial Pathology and Medical Research, Westmead.

The New South Wales Enteric Reference Laboratory receives 1800–2000 *Salmonella* isolates for serotyping each year, of which more than 50 per cent are *S.* Typhimurium. Such large numbers of a single serotype make it very difficult to identify potential outbreaks without additional strain typing (or 'fingerprinting'). Phage typing has provided valuable epidemiological data and assisted in outbreak investigations for nearly 60 years but has major limitations and is increasingly inadequate for 21st century disease surveillance.

Current methods for subtyping *Salmonella*

Phage typing has been, until recently, the subtyping method of choice for *S.* Typhimurium and several molecular typing methods are also used when further discrimination is needed. These methods are described briefly in the article by Wang et al. in this issue.

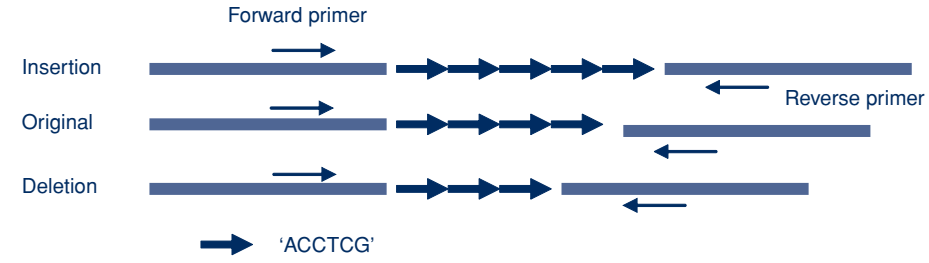
Introducing multi-locus variable number tandem repeat analysis

Recently, multi-locus variable number tandem repeat analysis has been successfully applied to many bacterial species, including several *Salmonella* serotypes, and has the potential to largely replace both phage typing and pulsed field gel electrophoresis as the primary subtyping method for salmonellae.[1–4]

Most bacterial genomes contain several sites or loci (genes or intergenic sequences), which contain variable numbers of repeated sequences that may be duplicated or deleted as part of the natural genetic variation of the species. This means that the total length of the locus varies between different strains. Development of a multi-locus variable number tandem repeat analysis (MLVA) scheme for a particular organism involves identifying up to 10 suitable loci within the genome. Suitability depends on the length of each sequence, by how much and how frequently the numbers of sequences vary, and whether there are conserved flanking sequences at each end that can be targeted by polymerase chain reaction primers. Strain-specific profiles derived from examination of these loci, allow objective strain comparison.

MLVA involves first amplifying the target loci by polymerase chain reaction and then measuring (either by gel or capillary electrophoresis) the lengths of the amplified DNA segments (amplicons). The number of repeats for each locus is inferred by subtracting the known length of the flanking sequence from the total amplicon length and dividing the result by the known length of each repeat sequence (as illustrated in Figure 1). The MLVA result or strain-specific profile is a series of numbers, each of which represents the number of repeats at one of the loci in a standard order.

For *S.* Typhimurium, loci are designated as STTR – *Salmonella* Typhimurium tandem repeat – and an arbitrary number. The scheme devised by Lindstedt et al., involves five loci – STTR9, STTR5, STTR6, STTR10pl ('pl' refers to the fact that this locus – STTR10pl – is on a plasmid, whereas the other loci are on the chromosome) and STTR3.[2] The lengths of repeat sequences at these loci, in base pairs, are: 9 for STTR9, 6 for STTR5, 6 for STTR6, 6 for STTR10pl and a combination of 27 and 33 base pair repeats for STTR3. There are various possible formats in which the MLVA profile could be expressed but, so far, none has been generally adopted.[1,5] Recently, representa-



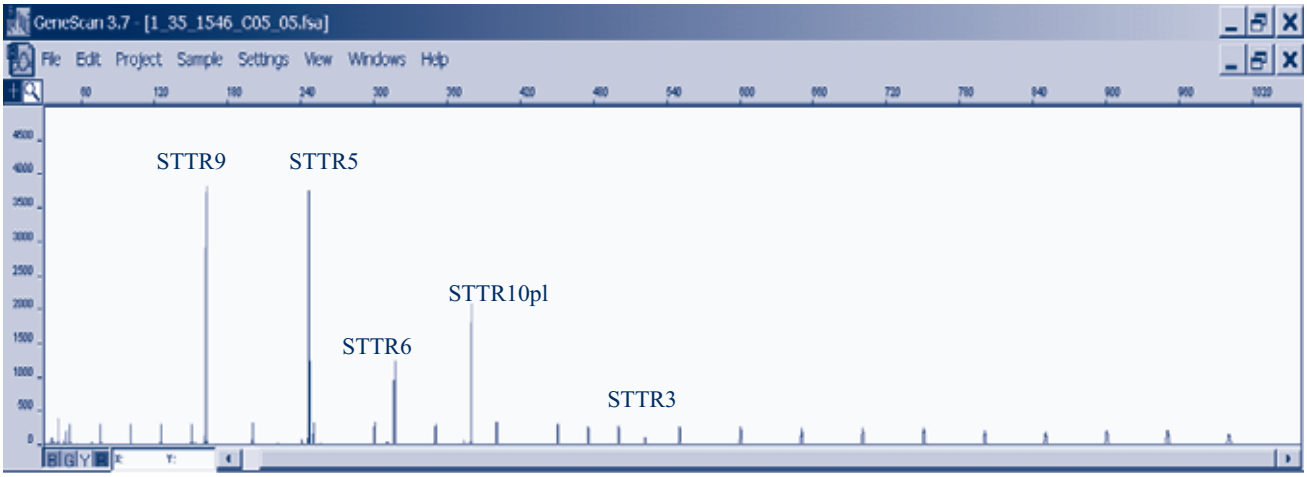
Tandem repeat sequences are represented by arrows, each with the hypothetical nucleotide sequence ACCTCG. Series of tandem repeat sequences vary in number, depending on whether a sequence is inserted or deleted. Forward and reverse polymerase chain reaction primers are complementary to conserved flanking sequences and used to amplify the locus. The number of repeats present in a particular strain is inferred from the total length of the amplified sequence.

Figure 1. Schematic representation of multilocus variable number tandem repeat analysis.

tives from several Australian reference laboratories agreed on the following convention for *S.* Typhimurium. For all loci except STTR3, the result will be expressed as 0 if there is no amplicon (i.e. the locus is absent); 1 if the size of the amplicon corresponds with that of the flanking region (i.e. the locus is present but no repeat sequences are present); 2 if the amplicon length corresponds with the sum of the flanking region and one repeat, and so on.

For STTR3, which is complicated by the potential presence of variable numbers of repeats of two different lengths, it was agreed that the actual amplicon length would be given (although it is possible to calculate the actual number of repeats of each length). This agreed convention is illustrated in Figure 2. This coding system may need to be modified in future but, in the meantime, it provides a method by which Australian laboratories can compare results.

A quality assurance program has recently been introduced; the first panel of isolates has been distributed and tested to ensure that the methods followed in participating labora-



Each locus is amplified using primers labelled with coloured dyes, for easy recognition. The size of each amplicon, in base pairs, is read automatically by the software. The number of repeats is calculated by subtracting the flanking region length from amplicon length, dividing by the repeat sequence length, and then adding 1.

Locus	Flanking length*	Amplicon length	Repeat length	Repeat No./ code
STTR9	144	162	9	2/ 3
STTR5	175	247	6	12/ 13
STTR6	264	318	6	9/ 10
STTR10pl	311	377	6	11/ 12
STTR3	106	523	27/33	2+11/ 523

*All lengths are in base pairs.
multilocus variable number tandem repeat profile: 03-13-10-12-523

Figure 2. Capillary electrophoresis 'read-out' for multilocus variable number tandem repeat analysis typing of *Salmonella* Typhimurium.
Source: New South Wales Enteric Reference Laboratory at the Centre for Infectious Diseases and Microbiology, ICPMR, Westmead.

tories generate consistent results. Consistency, is essential to enable the identification of disease outbreaks that cross state borders. A similar successful quality assurance exercise was recently reported from Scandinavia.[5]

In a recent study of 168 *S.* Typhimurium isolates, representing 46 phage types, STTR3, STTR5 and STTR9 were present in all isolates tested, STTR6 was present in 96 per cent and STTR10pl in 85 per cent of isolates. The numbers of repeats varied at different loci from as few as one or two for STTR9 to as many as 30 for STTR5 (Wang Q, Kong F, Jelfs P, Gilbert GL, unpublished data).

Using MLVA to identify clusters of disease

An important issue that is yet to be decided is the definition of a cluster. This requires further investigation. Preliminary data show that there is a high rate of clustering of isolates (when a cluster is defined as two or more isolates with the same MLVA profile). For example, during a 4-month period, 85 per cent of 185 *S.* Typhimurium isolates received consecutively by the NSW Enteric Reference Laboratory and tested by MLVA, were clustered, with 2–20 isolates per cluster. Over a longer period, it is likely that nearly all isolates would be clustered – that is, few, if any, individual MLVA profiles will be unique.

It is impractical to investigate every cluster, irrespective of the frequency or distribution of individual cases. The number that can be investigated will depend on available resources. One proposed cluster definition, suitable for a relatively low incidence country like Australia, is five or more cases of the same MLVA type occurring in a defined geographic area in a 4-week period.[6] Using this definition, 59 per cent of the 185 NSW isolates were clustered into 6 clusters over 4 months – a more feasible number for follow-up. Because of the relatively short time period in which a cluster is defined, the chance of identifying a source is relatively high.

Finally we need to determine the level of variation between isolates that can occur before isolates are no longer regarded as belonging to the same outbreak or cluster. The loss or gain of repeats occurs quite frequently at loci 2–4 but rarely at loci 1 and 5. Thus, profiles that vary by one or two digits at one of loci 2–4 can be regarded as probably related and investigated accordingly. Isolates are less likely to be related if there are differences at two of the inner loci, and are very unlikely to be related if there are differences at all three inner loci or at either locus 1 or locus 5. Further experience is required to develop more precise cluster definitions.

Next steps

During the next 12 months, the NSW Enteric Reference Laboratory, in collaboration with the Communicable Diseases Branch of the NSW Department of Health and the NSW Food Authority, will be evaluating MLVA prospectively, by comparing the results available within approximately 2 weeks of the receipt of isolates with those of epidemiological investigations of suspected clusters. We will also evaluate a novel molecular phage type identification system developed in our laboratory, which provides complementary information. In addition, we aim to develop a web-based reporting system. This will describe the geographic distribution of cases and clusters based on postcodes over defined time periods (spatiotemporal distribution) and will assess the risk that an individual case is part of a cluster based on detailed analysis of MLVA data.

Acknowledgements

I would like to thank Qinning Wang, Vitali Sintchenko, Peter Howard, Peter Jelfs and Robert Chiew for their contributions to the work described in the present study. Qinning Wang kindly provided images and data for the figures.

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Hepatitis A: who in NSW is most at risk of infection?

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Abstract: The incidence of hepatitis A in NSW has declined in recent years, but the relative importance of risk factors remains unclear. **Methods:** We analysed case data from the NSW Notifiable Diseases Database from 1991 to 2006. **Results:** Hepatitis A notification rates fell from 18.9 to 1.4 cases per 100000 between 1991 and 2006. International travel to endemic areas was the likely exposure for 50 per cent of cases between 2002 and 2006. Rates were five times higher in travellers born in countries where hepatitis A is endemic compared with those born in Australia. **Conclusion:** Travellers born in endemic countries should be carefully assessed for vaccination before departure.

Hepatitis A causes considerable morbidity worldwide with an estimated annual total of 1.5 million clinical cases.[1] Hepatitis A infection is primarily spread by the faecal oral route. In children, infection is usually asymptomatic but in adults it can cause fever, malaise, anorexia, nausea and/or abdominal discomfort followed by jaundice. Lifelong immunity develops following infection.[2] Recent seroprevalance studies report declining rates of naturally acquired immunity in some countries where the disease is endemic, presumably due to significant improvements in sanitation.[3–5]

In NSW in the 1990s, several outbreaks of hepatitis A were reportedly associated with male-to-male sexual contact and illicit drug use, and in 1997 a large outbreak was associated with the consumption of raw oysters.[6,7]

A highly effective vaccine was introduced in the 1990s and is recommended for: all people travelling to moderate to high endemic areas; those with intellectual disabilities or chronic liver disease; and those whose occupation or lifestyle may increase their risk of acquiring the infection.[8] It is likely that the introduction of a commercially available vaccine has contributed to the overall reduction of hepatitis A in NSW; however, other factors contributing to the decline remain unclear.

Uptake of immunisation among Australian travellers to areas where hepatitis A is endemic is reportedly low.[9] Other research has reported travel to endemic areas from low risk countries as an important risk factor for acquiring hepatitis A infection.[10–13] Recent data from England and Wales indicates that a higher rate of hepatitis A is observed in South-Asian-born residents, with the majority of these cases acquiring their infection while travelling to areas where hepatitis A is endemic.[14]

The aim of the present study was to investigate the changing incidence of hepatitis A in NSW from 1991 to 2006, and the relative importance of several risk factors from 2002 to 2006.

Methods

Under the *NSW Public Health Act 1991*, doctors, hospitals and laboratories must notify NSW Health of cases of viral hepatitis. Detection of anti-hepatitis A IgM, in the absence of recent vaccination, or the detection of hepatitis A virus by nucleic acid testing is required to confirm a diagnosis of hepatitis A. Since 1991, public health unit staff have investigated notified cases and recorded basic demographic and disease details onto the NSW Notifiable Diseases Database (NDD). In 2002, routine surveillance data collection was enhanced to include information on exposure to risk factors for acquiring the disease between two and seven weeks before the onset of symptoms (incubation period). These risk factors included: recreational drug use; male-to-male sexual contact; travel to endemic areas (by the case or a household member); contact with another possible or notified case; attendance at child care; contact with raw sewerage; and consumption of shellfish. The country where most time was spent was identified for those cases that reported a travel history during their incubation period. It was assumed for cases reporting travel to an endemic country for any length of time during their exposure period that hepatitis A was most likely acquired overseas. Countries were considered endemic on the basis of the World Health Organization classification as shown in Figure 1.[15] Estimates for the NSW population born in Australia and in endemic countries were obtained using 2006 census data.

Results

In NSW, hepatitis A notification rates fell from 18.9 cases per 100000 in 1991 to 1.4 cases per 100000 in 2006

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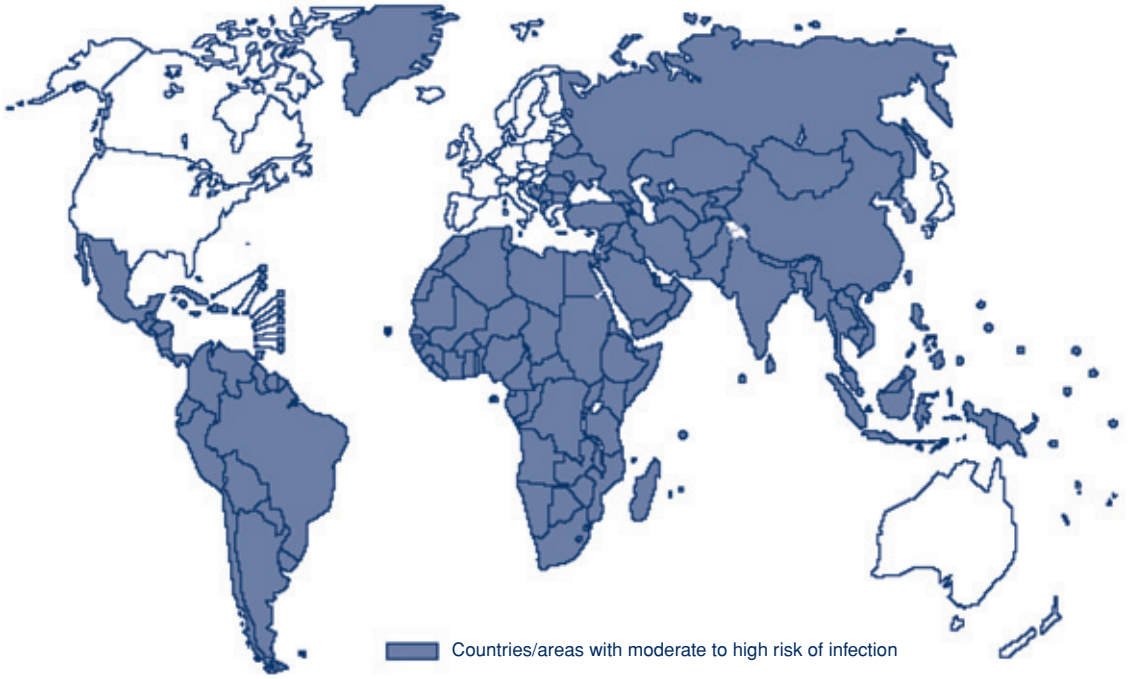

Figure 1. Worldwide distribution of hepatitis A endemicity, 2003
Source: World Health Organization. Hepatitis A vaccine. http://www.who.int/vaccines/en/hepatitisa.shtml

(Figure 2). The age of cases was consistent over time with a mean of 29.9 years in 1991 and 30.9 years in 2006. The proportion of cases that were male decreased from 81 per cent in 1991 to 48 per cent in 2006.

From 2002 to 2006, 586 cases were notified to NSW Health. For only 8 per cent of these cases was the field identifying whether the person was an Aboriginal or Torres Strait Islander person completed; of these 2 per cent were Aboriginal or Torres Strait Islander people. Information on vaccination status was complete for 49 per cent of cases. Of the cases where vaccination status was documented, 94 per cent were unvaccinated.

Information on risk factors was available for 78 per cent (n = 458) of cases. Of these, the most commonly reported risk factor was international travel to endemic areas (Table 1) and all other risk factors were less commonly reported.

Travel to endemic areas was associated with the highest number of notifications each year from 2002 to 2006 with

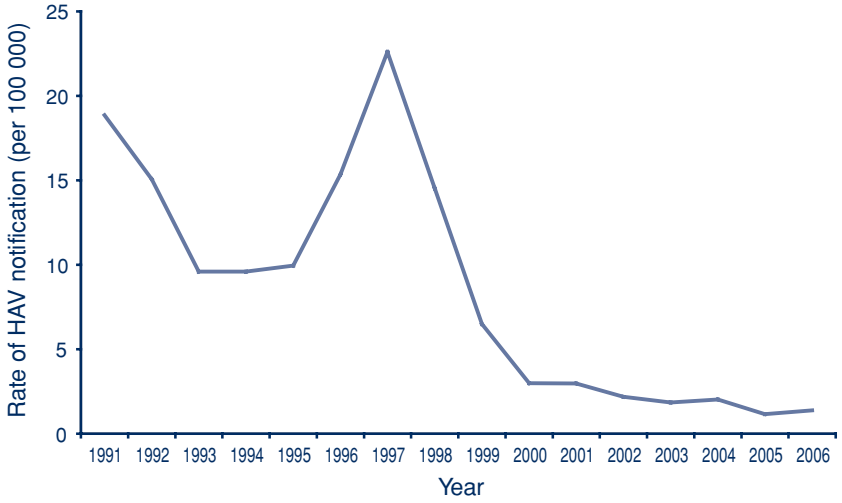

Figure 2. Hepatitis A notification rates from 1991 to 2006 in NSW.
Source: NSW Notifiable Diseases Database, NSW Health.

Table 1. Frequency of risk factors reported by hepatitis A cases in NSW, 2002 to 2006

Risk factor*	*n*	%
Travel to endemic areas	228	50
Household member travel to endemic area	121	26
Contact with another notified case	44	10
Contact with another possible case	42	9
Male-to-male sexual contact	31	7
Ate raw shellfish	30	7
Contact with raw sewerage	17	4
Recreational drug use	16	3
Attends child care centre	5	1

*Categories are not mutually exclusive and in many instances data was incomplete.
Source: NSW Notifiable Diseases Database.

an average of 46 notifications per year (range: 37–56). This increased as a proportion of all reported cases with risk factor information from 35 per cent (37/107) in 2002 to 65 per cent (53/81) in 2006. For the 96 per cent of cases for whom the country of travel was documented, South and South-East Asia were the most commonly reported travel destinations (Figure 3).

Information on place of birth was available for 89 per cent of cases reporting travel to endemic areas in their exposure period. By place of birth, the rate of hepatitis A in travellers born in endemic countries was significantly greater ($p < 0.0001$) than the rate in Australian-born travellers (12.5 cases per 100 000 and 2.3 cases per 100 000, respectively) (Figure 4). Cases born where hepatitis A is endemic primarily originated from South and South-East Asian countries (Table 2) and 90 per cent of cases acquired the infection while returning to their country of birth.

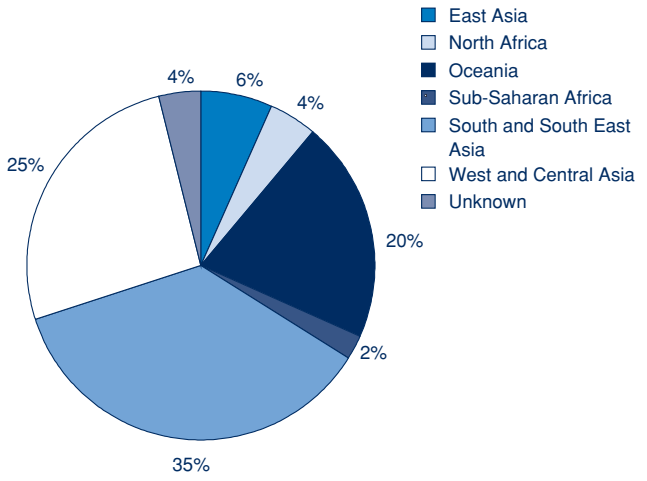

Figure 3. Likely geographical region where hepatitis A was acquired as reported by cases in NSW, 2002 to 2006.
Source: NSW Notifiable Diseases Database.

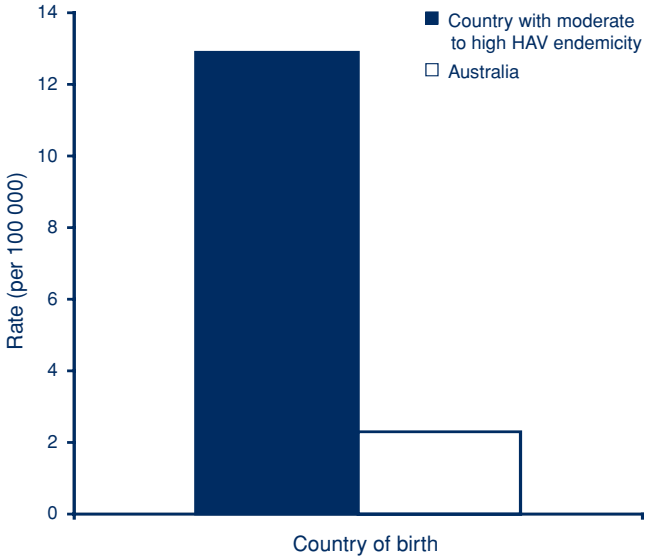

Figure 4. Rate of hepatitis A infection associated with international travel to endemic areas for persons born in Australia compared with those born in countries with moderate to high endemicity.
Source: NSW Notifiable Diseases Database.

Discussion

In recent years, the rate of hepatitis A has declined dramatically in NSW. While analysis of outbreak data indicates that in the 1990s male-to-male sexual contact and recreational drug use were important risk factors, these are now associated with only a small number of cases.[6] The dramatic reduction in the proportion of male cases from 81 per cent in 1991 to 48 per cent in 2006 is most likely a result of a decline in transmission between men who have sex with men in this period. Although there is no evidence that the absolute number of cases acquired during travel to endemic areas has increased, it is now the most common risk factor for hepatitis A cases in NSW.

Given the growing proportion of cases with hepatitis A reporting travel to endemic countries and the popularity of South Asian travel destinations for Australian travellers, efforts to promote pre-travel vaccination are increasingly important to reduce the burden of disease in Australia.[9] In

Table 2. Global region of birth for NSW hepatitis A cases associated with travel, 2002 to 2006 (*n* = 203)

Region of birth	Proportion (%)
Australia	52
South and South East Asia	21
West and Central Asia	10
Europe	5
East Asia	5
Oceania	5
Sub-Saharan Africa	2

Source: NSW Notifiable Diseases Database.

addition, given the infectiousness of hepatitis A before the onset of symptoms and diagnosis, vaccination of travellers is the most efficient means of preventing secondary cases in household members.

The present study is limited by the data available on the vaccination status of reported cases being incomplete. However, given the high vaccine efficacy reported in other studies, and low vaccine uptake reported in Australian travellers, it is reasonable to assume that the majority of notified cases were unvaccinated. Information on other risk factors was also incomplete. Self-reporting of behaviours such as male-to-male sexual contact and recreational drug use may underestimate the true prevalence of these risk factors but there is no evidence to suggest that underreporting varied over the period. More complete case information, including risk factors and vaccination status, would enhance understanding of the epidemiology of hepatitis A.

Compared with Australian-born travellers, travellers born in endemic countries returning to their country of origin are at increased risk of acquiring hepatitis A infection.[14] Factors that may influence the likelihood of overseas-born travellers acquiring natural immunity to hepatitis A in childhood include the country and region of origin, their socioeconomic status and the age they left the endemic area.

In highly endemic countries, most people experience asymptomatic infection within the first few years of life.[2] However, the declining risk of hepatitis A transmission in some endemic countries, primarily due to improved sanitation and standards of living, has resulted in a decreased risk of infection in early childhood.[1] Subsequently, travellers returning to their country of birth to visit family and friends living in areas with poorer sanitation may be at greater risk of hepatitis A than other tourists staying in hotels and dining in restaurants. Furthermore, travellers born in endemic areas who are returning 'home' may be less likely to seek travel advice before departure and more likely to have repeated and/or longer visits, increasing their overall exposure to risk of disease. A recent seroprevalence study involving Indian- and Chinese-born immigrants to the United States recommends assessing immunity before travel for younger immigrants given their greater susceptibility to infection.[16]

The NHMRC immunisation handbook currently recommends screening for pre-existing immunity in those individuals who spent their early childhood in endemic areas.[8] Economic analyses are required to compare the value of pre-vaccination screening to vaccination alone for endemic born travellers to endemic areas.

Based on NSW data, travellers born in countries where hepatitis A is endemic who are now residing in Australia may not have immunity to hepatitis A infection and should be carefully assessed for vaccination before departure to endemic areas.

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Polio

What is polio?

Poliomyelitis (or 'polio') is a viral infection that can cause paralysis and death. In the past, polio was common, especially in children. Now, due to immunisation, polio is rare in most parts of the world, although it persists in some areas. The last reported case of wild (disease-causing) polio in Australia occurred in 1978.

What are the symptoms?

- The majority of people infected with polio do not have symptoms.
- A minor illness causing fever, headache, lethargy, nausea and vomiting occurs in approximately 10 per cent of people who are infected with the virus. Most of these people completely recover. However, approximately 2 per cent go on to experience severe muscle pain with back or neck stiffness, called non-paralytic aseptic meningitis (inflammation of the lining of the brain without weakness).
- Less than 1 per cent of people who are infected develop severe weakness called acute flaccid paralysis. This usually affects the limbs but it can also affect the muscles of the head, neck and diaphragm muscle, which is used for breathing. Most people with acute flaccid paralysis recover, although the recovery is not complete in all people and some die.

How is it spread?

- Polio is a highly infectious disease spread by close contact with an infected person, via contact with minute amounts of faeces (eg, on unwashed hands) or from droplets from the throat of an infected person.
- Untreated sewerage that comes into contact with foods or drinking water can spread polio in parts of the world where there is poor sanitation.
- The polio virus enters the body through the nose or mouth and infection starts in the gut. It then enters the blood stream and is carried to other parts of the body, including the nervous system.
- The time from being exposed to the polio virus and getting sick can range from 3 to 35 days, but is commonly 7 to 14 days.
- Cases are most infectious from 10 days before onset of symptoms to 10 days after the onset of symptoms.
- People can continue to shed the virus in their faeces for up to six weeks.

Who is at risk?

- Owing to immunisation, Australia is currently free from polio.
- The World Health Organization is currently working towards eradicating polio worldwide but in 2007 it still exists in parts of Africa and South Asia.
- People who are not immune may become infected in countries where polio still exists. They may then bring the infection with them when they travel to another country.

How is it prevented?

Immunisation protects people against polio. While transmission does not currently occur in Australia, the polio virus could be imported and spread among unimmunised populations.

- For all children, a course of three injections at two, four and six months of age, with boosters at 4 years of age, is recommended.
- For some adults (travellers to countries with polio and health care workers who may look after patients with polio), a booster is recommended every 10 years.
- Adequate treatment of sewerage and provision of safe drinking water and foods is also important to prevent the disease from spreading.

How is it diagnosed?

- The doctor may suspect polio based on the person's symptoms and signs. However, some other infections can cause similar symptoms. Acute flaccid paralysis can also have other causes and these people need blood tests and stool tests to test if their symptoms are caused by polio virus. A national polio expert committee decides if the symptoms and tests could be polio.
- Isolation of the virus from stools, throat or spinal cord fluid is required to confirm the diagnosis of polio.
- Special studies are needed to distinguish the vaccine strain of the virus from the wild (disease-causing) virus.

How is it treated?

There is no cure for polio and treatment is mainly to support cases with acute flaccid paralysis while their weakness is severe. Cases with acute flaccid paralysis may require intensive care to assist breathing.

What is the public health response?

- Hospitals, laboratories, school principals and childcare centres must notify suspected cases of polio including acute flaccid paralysis to the local public health unit.
- Public health units will investigate suspect cases and review possible sources of infection to prevent further spread.
- If a case is detected in Australia, people who are at risk may need to be immunised again against polio.

For more information, please contact your doctor, local public health unit or community health centre.

NSW HEALTH

10.1071/NB07106

Communicable Diseases Report, NSW, November–December 2007

Communicable Diseases Branch, NSW Department of Health

For updated information, including data and facts on specific diseases, visit www.health.nsw.gov.au and click on **Infectious Diseases.**

Tables 1 and 2 and Figure 1 show reports of communicable diseases received through to the end of November and December 2007 in NSW.

Meningococcal disease

In NSW, meningococcal disease was notified in eight people in November and 12 people in December. In total, 108 cases were notified in the 12 months to December 2007, including five deaths. Of the 2007 cases, nine were due to serogroup C meningococcal bacteria and 73 were due to serogroup B. In 2006, 102 cases were notified including six deaths.

Enteric Diseases

In November, NSW public health units investigated 58 outbreaks of gastroenteritis, including two suspected to be foodborne and 56 suspected to be caused by person-to-person spread. The two suspected foodborne outbreaks involved groups of 11 and three people, respectively, at different restaurants; no specimens were available for testing from either outbreak. Among the 56 suspected person-to-person outbreaks, 39 were in age care facilities and affected 657 people, 11 were in hospitals that affected 85 people, five were in child-care centres and affected 45 children, and one was in a school and affected 16 children. For comparison, 70 outbreaks were reported in October 2007 and 29 were reported in November 2006.

In December, NSW public health units investigated 10 outbreaks of gastroenteritis, including three suspected to be foodborne outbreaks and seven suspected to be caused by person-to-person spread. The three foodborne outbreaks were caused by salmonellosis and consumption of undercooked or raw eggs was suspected to be a possible source of infection. No links between the outbreaks were identified. Of the seven outbreaks of gastroenteritis, four were in aged care facilities and affected 31 people, two were in hospitals and affected eight people, and one was in a child-care centre and affected six children.

Increase in reported cases of shiga toxin-producing Escherichia coli

Shiga toxin-producing *E. coli* (STEC) are bacteria that can cause serious gastrointestinal disease characterised by diarrhoea, which in some cases can be bloody. In a small proportion of cases STEC can progress to haemolytic uraemic syndrome (HUS), which results in kidney failure, bleeding and anaemia. Infections tend to increase in the warmer months.[1]

In November, NSW public health units were notified of seven cases of STEC (3 serotype O157, 1 serotype O111, and 1 serotype O26) and three cases of HUS. The ages of the cases ranged from 2 to 71 years. Seven cases were male and three female. Seven cases resided in the Hunter New England Area, two in South East Sydney Illawarra Area and one in the Greater Southern Area. The HUS cases were all children aged 2 to 5 years; STEC (untyped) was also identified in one of the HUS cases. Although 10 cases within a month appears unusually high, the total number of cases in 2007 (16 STEC and 11 HUS cases) is similar to previous years. Interviews with the cases or their carers did not identify a likely common source of infection.

In December, NSW public health units were notified of seven STEC (1 serotype O157, 1 serotype O111, 1 serotype O55 and 4 of unknown serotype) and two HUS cases. The age of cases ranged from 11 months to 75 years. Six were female and three male. All HUS cases were adults aged over 40 years. This number of STEC and HUS cases reported in December 2007 is slightly higher than the number seen in December 2006.

STEC infection can be transmitted through:

- eating contaminated food (undercooked hamburgers, unwashed salad, fruit, vegetables and unpasteurised milk or milk products)
- drinking or swimming in contaminated water
- person-to-person contact; for example, contact with faeces of an infected child when changing a nappy
- contact with infected animals.[2,3]

The most important ways to prevent infection with STEC and other foodborne diseases are to:

- cook hamburgers and sausages thoroughly to at least 71°C. Although colour alone is not necessarily a good

indicator, do not eat hamburgers or sausages if there is any pink meat inside
- wash hands well after handling raw meat
- use different knives and cutting boards for raw meat preparation and other food preparation
- wash raw vegetables and fruits thoroughly
- refrigerate perishable food until ready to eat
- wash hands well after touching animals or their faeces.

For more information see: http://www.health.nsw.gov.au/infect/pdf/stec_cdfs.pdf.

Listeriosis

In December, four cases of listeriosis were reported in NSW, two male and two female. The age of cases ranged from 28 to 75 years. Cases reported eating a range of high-risk foods; however, no common source of infection was identified. One case was a pregnant woman; she and her babies recovered.

Listeriosis is usually caused by ingestion of contaminated food and has been associated with consumption of undercooked or raw meat, runny eggs, soft cheeses, unpasteurised milk and pre-prepared and unwashed vegetables. Those at highest risk are unborn babies, the elderly, immune compromised people and pregnant women. Listeriosis is particularly important for pregnant women as the infection can cause foetal death.[4]

Increase in reported cases of cryptosporidiosis

There were 153 cases of cryptosporidiosis reported as having their onset date in November and 84 in December in NSW. This compares with 34 cases in October. The highest rate of infection was in children under five years of age (see: http://www.health.nsw.gov.au/data/diseases/cryptosporidiosis.html) and in rural areas.

Cryptosporidiosis is a diarrhoeal disease caused by a parasitic infection of the intestine. The most common symptoms include diarrhoea, abdominal cramps and sometimes fever, nausea and vomiting. Symptoms may last a few weeks in some people.[5]

Public health officers interviewed cases who report a range of possible risk factors, including contact with farm animals, drinking untreated water and swimming.

In the past, large outbreaks in NSW have been caused by people swimming in contaminated pools.[6] Pools can easily be contaminated by infectious swimmers. To keep pools free from contamination, people should not swim in a pool or spa until at least two weeks after they have completely recovered from a diarrhoeal illness.

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Figure 1. Reports of selected communicable diseases, NSW, January 2002 to December 2007, by month of onset.

Preliminary data: case counts in recent months may increase because of reporting delays.
Laboratory-confirmed cases only, except for measles, meningococcal disease and pertussis.
BFV, Barmah Forest virus infections; RRV, Ross River virus infections; Lab Conf, laboratory confirmed;
Men Gp C and Gp B, meningococcal disease due to serogroup C and serogroup B infection;
other/unk, other or unknown serogroups.
NB: multiple series in graphs are stacked, except gastroenteritis outbreaks.
NB: Outbreaks are more likely to be reported by nursing homes and hospitals than by other institutions.

NSW Population	
Male	50%
<5 y	7%
5–24y	27%
25–64y	53%
65+ y	13%
Rural	46%

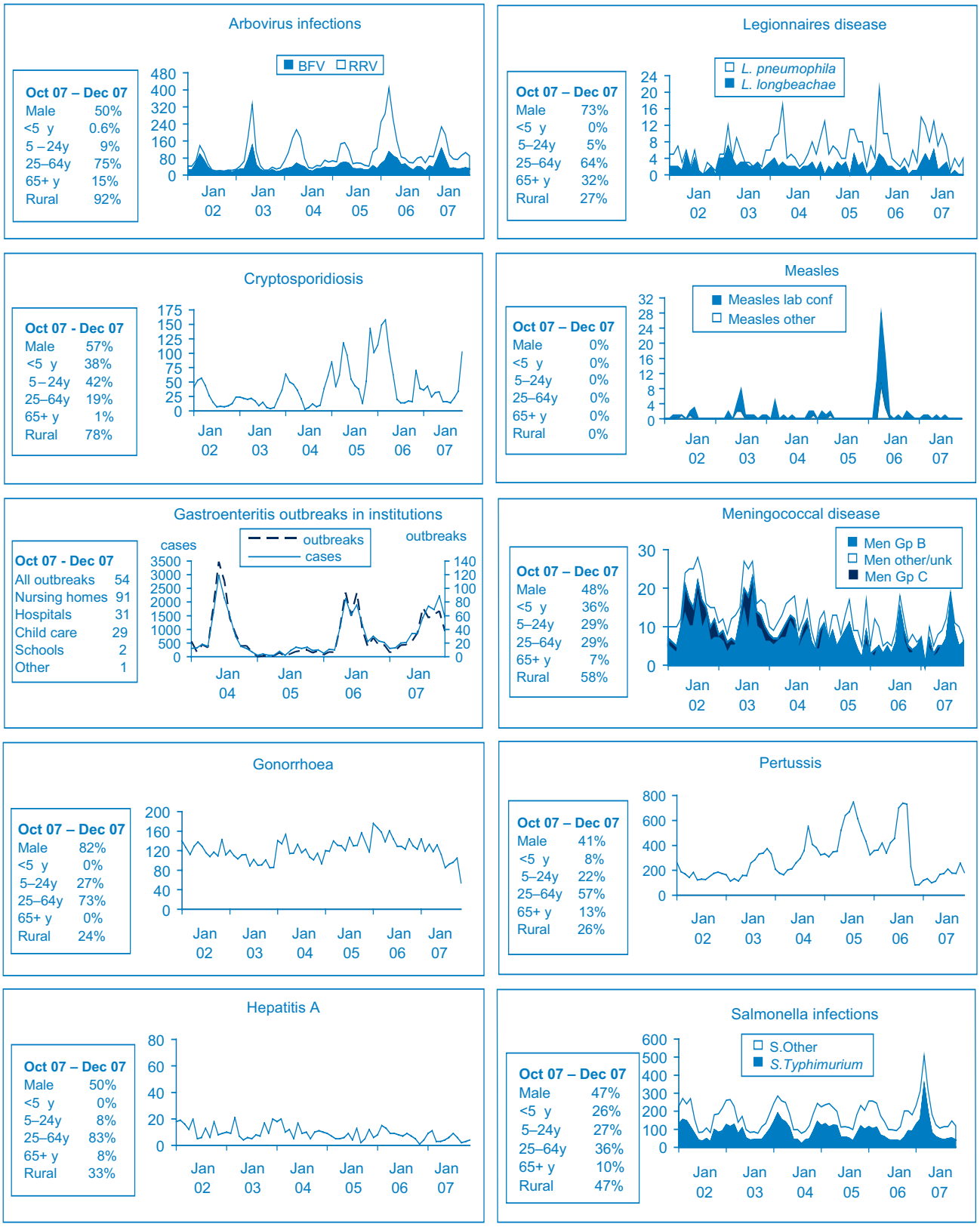

Table 1. Reports of notifiable conditions received in November 2007 by Area Health Services

Condition	Area Health Service (2007)																		Total	
	Greater Southern		Greater Western			Hunter/New England		North Coast		Northern Syd/ Central Coast		South Eastern Syd/Illawarra		Sydney South West		Sydney West		JHS		
	GMA	SA	FWA	MAC	MWA	HUN	NEA	MNC	NRA	CCA	NSA	ILL	SES	CSA	SWS	WEN	WSA		For Nov.[c]	To date[c]
Blood-borne and sexually transmitted																				
Chancroid[a]	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–
Chlamydia (genital)[a]	39	25	10	14	40	132	43	47	55	61	77	33	194	–	1	38	96	3	934	11548
Gonorrhoea[a]	2	2	–	–	1	15	–	1	2	2	8	5	25	–	1	6	10	–	81	1291
Hepatitis B – acute viral[a]	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	48
Hepatitis B – other[a]	–	3	–	1	1	4	1	4	6	5	31	4	38	1	6	5	46	–	159	2945
Hepatitis C – acute viral[a]	–	–	–	1	–	–	–	–	–	–	–	–	–	–	–	–	–	–	1	33
Hepatitis C – other[a]	17	10	5	4	7	46	10	33	25	30	27	30	38	1	1	18	34	22	366	4825
Hepatitis D – unspecified[a]	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	11
Lymphogranuloma vanereum	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–
Syphilis	–	–	1	2	–	4	1	1	4	–	9	1	43	5	1	1	12	–	87	1096
Vector-borne																				
Barmah Forest virus[a]	–	2	–	2	1	10	–	11	8	1	–	1	–	–	–	–	–	–	36	545
Ross River virus[a]	6	1	1	8	1	22	6	14	6	3	2	4	4	2	1	1	2	–	84	762
Arboviral infection (other)[a]	–	–	–	–	–	–	–	–	–	–	–	–	1	–	–	1	–	–	2	70
Malaria[a]	–	1	–	–	–	–	1	1	–	–	–	–	2	–	–	1	2	–	9	99
Zoonoses																				
Anthrax[a]	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–
Brucellosis[a]	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	4
Leptospirosis[a]	–	–	–	–	–	–	1	–	1	–	–	–	–	–	–	–	–	–	2	11
Lyssavirus[a]	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–
Psittacosis[a]	–	–	–	2	–	–	–	–	–	–	–	–	–	–	–	–	–	–	2	31
Q fever[a]	1	1	–	5	–	2	11	1	2	–	–	3	–	–	–	–	–	–	26	201
Respiratory and other																				
Blood lead level[a]	–	–	–	5	–	5	–	–	–	–	–	–	–	–	–	–	–	–	10	239
Influenza[a]	–	1	1	1	3	1	–	2	5	–	1	–	4	–	–	5	5	–	29	1731
Invasive pneumococcal infection[a]	3	–	–	–	1	8	–	8	2	–	3	–	5	4	6	2	3	–	45	502
Legionella longbeachae infection[a]	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	26
Legionella pneumophila infection[a]	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	1	3	–	4	60
Legionnaires' disease (other)[a]	–	–	–	–	–	–	1	–	–	–	–	–	–	–	–	–	–	–	1	2
Leprosy	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	3
Meningococcal infection (invasive)[a]	–	1	–	–	–	2	–	1	–	2	–	–	1	–	–	–	1	–	8	99
Tuberculosis	–	–	–	–	–	1	–	–	–	–	3	2	5	–	–	1	8	–	20	349
Vaccine-preventable																				
Adverse event after immunisation[b]	3	–	–	–	3	–	–	–	–	1	–	2	–	–	2	–	2	–	13	204
H. influenzae b infection (invasive)[a]	–	–	–	–	–	–	–	–	–	–	1	–	–	–	–	–	–	–	1	7
Measles	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	4
Mumps[a]	1	–	–	–	–	1	–	–	–	–	6	–	31	–	1	2	5	–	47	245
Pertussis	9	6	1	6	–	12	5	2	4	6	40	12	45	42	11	6	46	–	253	1865
Rubella[a]	–	–	–	–	–	–	–	–	–	–	1	–	–	–	–	–	–	–	1	13
Tetanus	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	4
Enteric																				
Botulism	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–
Cholera[a]	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	2
Cryptosporidiosis[a]	19	1	1	7	10	9	23	6	17	–	7	3	2	2	4	6	6	–	123	392
Giardiasis[a]	3	1	–	1	2	10	5	4	2	11	26	6	31	–	–	3	21	–	126	1819
Haemolytic uraemic syndrome	–	–	–	–	–	1	1	–	–	–	–	–	1	–	–	–	–	–	3	11
Hepatitis A[a]	–	–	–	–	–	–	–	–	1	–	–	–	–	1	1	1	–	–	4	61
Hepatitis E[a]	–	–	–	–	–	–	–	–	–	–	1	–	–	–	–	–	–	–	1	9
Listeriosis[a]	–	–	–	–	–	1	–	–	–	–	–	–	–	–	–	–	1	–	2	20
Salmonellosis[a]	6	13	1	5	5	18	10	4	14	6	17	3	26	12	14	5	13	–	173	2368
Shigellosis[a]	–	–	–	–	–	–	–	–	–	–	2	–	1	–	2	–	–	–	5	65
Typhoid[a]	–	–	–	–	–	–	–	–	–	–	–	–	1	–	–	–	–	–	1	37
Verotoxin-producing *E. coli*[a]	–	–	–	–	–	4	1	–	–	–	–	–	–	–	–	1	–	–	6	14
Miscellaneous																				
Creutzfeldt–Jakob disease	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	9
Meningococcal conjunctivitis	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	3

[a]Laboratory-confirmed cases only. [b]HIV and AIDS data are reported separately in the Public Health Bulletin quarterly. [c]Includes cases with unknown postcode.
NB: From 1 January 2005, Hunter/New England AHS also comprises Great Lakes, Gloucester and Greater Taree LGAs, Sydney West also comprises Greater Lithgow LGA.
NB: Data is current and accurate as at the preparation date. The number of cases reported is, however, subject to change, as cases may be entered at a later date or retracted upon further investigation.

GMA, Greater Murray Area; MAC, Macquarie Area; NEA, New England Area; CCA, Central Coast Area; SES, South Eastern Sydney Area; WEN, Wentworth Area; SA, Southern Area; MWA, Mid Western Area; MNC, North Coast Area
NSA, Northern Sydney Area; CSA, Central Sydney Area; WSA, Western Sydney Area; FWA, Far West Area; HUN, Hunter Area; NRA, Northern Rivers Area; ILL, Illawarra Area; SWS, South Western Sydney Area; JHS, Justice Health Service

Table 2. Reports of notifiable conditions received in December 2007 by Area Health Services

Condition	Area Health Service (2007)																			Total	
	Greater Southern		Greater Western			Hunter/New England		North Coast		Northern Syd/ Central Coast		South Eastern Syd/Illawarra		Sydney South West		Sydney West		JHS		
	GMA	SA	FWA	MAC	MWA	HUN	NEA	MNC	NRA	CCA	NSA	ILL	SES	CSA	SWS	WEN	WSA		For Dec.[c]	To date[c]
Blood-borne and sexually transmitted																				
Chancroid[a]	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–
Chlamydia (genital)[a]	27	19	5	9	23	82	32	19	32	46	67	28	146	12	6	30	77	1	681	12416
Gonorrhoea[a]	1	–	–	–	2	13	–	–	2	2	8	–	38	6	1	–	6	–	83	1398
Hepatitis B – acute viral[a]	–	–	–	–	–	–	–	–	1	–	–	–	–	–	1	–	–	–	2	62
Hepatitis B – other[a]	5	–	2	1	2	5	3	–	5	2	24	5	29	6	6	4	30	1	132	3178
Hepatitis C – acute viral[a]	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	34
Hepatitis C – other[a]	7	5	2	3	6	35	8	12	24	24	14	20	19	10	3	16	15	16	240	5236
Hepatitis D – unspecified[a]	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	11
Lymphogranuloma vanereum	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–
Syphilis	1	1	–	1	2	2	1	3	–	1	5	1	33	1	5	–	4	–	63	1167
Vector-borne																				
Barmah Forest virus[a]	–	–	–	1	1	8	1	2	8	–	–	1	–	–	–	–	–	–	22	572
Ross River virus[a]	11	–	3	9	–	20	3	10	5	5	–	3	–	–	1	–	4	–	73	838
Arboviral infection (other)[a]	–	–	–	–	–	1	–	1	1	1	–	–	–	1	–	–	–	–	5	75
Malaria[a]	–	–	–	–	–	1	–	–	–	–	–	1	–	–	–	–	2	–	4	103
Zoonoses																				
Anthrax[a]	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–
Brucellosis[a]	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	4
Leptospirosis[a]	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	10
Lyssavirus[a]	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–
Psittacosis[a]	–	–	–	–	1	–	–	–	–	–	–	1	–	–	–	–	–	–	2	34
Q fever[a]	–	–	–	3	1	–	2	–	–	–	–	–	–	–	–	–	–	–	6	207
Respiratory and other																				
Blood lead level[a]	3	–	–	2	–	1	–	–	–	–	1	–	1	–	–	–	–	–	8	248
Influenza[a]	2	2	–	–	1	1	–	1	1	–	1	1	2	–	–	3	6	–	21	1763
Invasive pneumococcal infection[a]	–	1	1	–	1	5	–	–	1	–	1	1	1	1	1	2	3	–	19	522
Legionella longbeachae infection[a]	1	–	–	–	–	–	–	–	–	–	2	–	–	–	–	–	–	–	3	30
Legionella pneumophila infection[a]	–	–	–	–	–	–	1	–	–	1	1	–	2	3	1	–	–	–	9	70
Legionnaires' disease (other)[a]	1	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	1	3
Leprosy	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	1	–	1	4
Meningococcal infection (invasive)[a]	–	1	–	–	–	2	–	–	1	2	4	1	1	1	1	–	–	–	14	112
Tuberculosis	–	–	–	–	–	–	–	–	–	1	–	–	8	–	–	–	2	–	11	371
Vaccine-preventable																				
Adverse event after immunisation[b]	1	–	–	1	–	1	–	–	–	–	–	–	1	–	2	–	2	–	8	214
H. influenzae b infection (invasive)[a]	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	8
Measles	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	4
Mumps[a]	1	–	–	–	–	–	–	–	–	1	5	5	23	2	2	1	10	–	50	304
Pertussis	1	1	–	–	2	16	1	3	8	3	40	4	26	31	12	10	29	–	187	2059
Rubella[a]	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	13
Tetanus	–	–	–	–	–	–	–	–	1	–	–	–	–	–	–	–	–	–	1	5
Enteric																				
Botulism	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–
Cholera[a]	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	2
Cryptosporidiosis[a]	17	4	–	3	12	9	20	3	11	6	5	2	3	4	9	1	3	–	112	510
Giardiasis[a]	4	2	–	2	4	15	2	1	1	4	20	8	13	3	2	6	14	–	101	1946
Haemolytic uraemic syndrome	–	–	–	–	–	1	–	–	–	–	–	1	–	–	–	–	–	–	2	13
Hepatitis A[a]	–	–	–	–	–	1	–	–	1	–	1	–	1	–	1	–	–	–	5	66
Hepatitis E[a]	–	–	–	–	–	–	–	–	–	–	–	–	1	–	–	–	–	–	1	9
Listeriosis[a]	–	–	–	–	–	1	–	–	–	–	1	–	1	–	–	1	–	–	4	24
Salmonellosis[a]	6	6	–	5	3	17	3	6	14	8	19	3	12	5	8	6	21	–	142	2543
Shigellosis[a]	–	–	–	–	–	–	–	–	1	–	–	–	–	–	1	–	–	–	2	65
Typhoid[a]	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	1	–	1	37
Verotoxin-producing *E. coli*[a]	2	–	–	–	–	2	2	–	–	–	–	–	–	–	–	1	–	–	7	21
Miscellaneous																				
Creutzfeldt–Jakob disease	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	9
Meningococcal conjunctivitis	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	3

[a]Laboratory-confirmed cases only. [b]HIV and AIDS data are reported separately in the Public Health Bulletin quarterly. [c]Includes cases with unknown postcode.
NB: From 1 January 2005, Hunter/New England AHS also comprises Great Lakes, Gloucester & Greater Taree LGAs, Sydney West also comprises Greater Lithgow LGA.
NB: Data is current and accurate as at the preparation date. The number of cases reported is, however, subject to change, as cases may be entered at a later date or retracted upon further investigation.

GMA, Greater Murray Area; MAC, Macquarie Area; NEA, New England Area; CCA, Central Coast Area; SES, South Eastern Sydney Area; WEN, Wentworth Area; SA, Southern Area; MWA, Mid Western Area; MNC, North Coast Area
NSA, Northern Sydney Area; CSA, Central Sydney Area; WSA, Western Sydney Area; FWA, Far West Area; HUN, Hunter Area; NRA, Northern Rivers Area; ILL, Illawarra Area; SWS, South Western Sydney Area; JHS, Justice Health Service

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The *NSW Public Health Bulletin* is a peer-reviewed journal produced by the NSW Department of Health and indexed in Medline. It has a NSW focus, however, it aims to support the practice of public health more broadly.

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