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FOODBORNE DISEASE SURVEILLANCE IN NEW SOUTH WALES

GUEST EDITORIAL

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The major objectives of the surveillance of foodborne disease are to identify emerging threats to human health and to monitor prevention interventions. This issue of the *NSW Public Health Bulletin* examines current surveillance activities and identifies areas of development.

The article by Dalton highlights the critical role and responsibility that NSW has as a partner in national foodborne disease surveillance, and the likely benefits of recent investments in surveillance infrastructure. The article borrows from clinical audit practices, by citing variations in outbreak reporting rates by area health service as a potential indicator of quality assurance.

The article by Lee et al. uses a standard case-control study to associate undercooked shrimp dumplings in a yum cha meal with an outbreak of hepatitis A. In previous outbreaks, the cooling of cooked shrimp in contaminated river water has been suggested as a source of contamination. This investigation warns of two emerging threats that will require the vigilance of local, state, and international food safety agencies. At the international level, the investigation provides further evidence of the need to develop Hazard Analysis and Critical Control Points,[1] to ensure that seafood cooked during its processing is not contaminated prior to its distribution. At the local level, it will be important to monitor the effectiveness of restaurant cooking in stacked steaming baskets to ensure even cooking of foods.

The current notifiable diseases surveillance system has been in place for over 10 years. The article by Persson and Bartlett describes a review of the this system, to determine its effectiveness and guide further improvements. This article outlines the nature of the review, its recommendations, and the progress to date in addressing those recommendations.

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The article by Kirk describes some of the shortfalls in foodborne disease surveillance in Australia, and argues for greater integration of surveillance information on the microbial contamination of food, animal carriage, human illness, and other hazards. Kirk cites the benefits that have been realised, in some Scandinavian countries, through the integration of surveillance information.

The EpiReview by Neville and McAnulty analyses the surveillance of notified enteric diseases and reports of foodborne disease outbreaks in NSW, and identifies the need to further enhance outbreak reporting.

Together, these articles provide an overview of the epidemiological and surveillance framework for the promotion of food safety in NSW. A future issue of the *NSW Public Health Bulletin* will explore the evolution to a single agency responsible for ensuring safe food production, the NSW Food Authority.

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FOODBORNE DISEASE SURVEILLANCE IN NSW: MOVING TOWARDS PERFORMANCE STANDARDS

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NSW Health has sole responsibility for the surveillance of foodborne disease in humans, through the receipt of notifications for a range of conditions that are predominantly or potentially foodborne in transmission. These conditions include: salmonellosis, listeriosis, shigellosis, typhoid, Verotoxin producing *E. coli* infection, cholera, hepatitis A, giardiasis, and cryptosporidiosis. In addition, outbreaks of foodborne disease affecting two or more people are notifiable. Surveillance methods used in NSW are described in detail in this issue of the *NSW Public Health Bulletin* by Neville and McAnulty. This article describes the evolution of, and recent investments in, foodborne disease surveillance and control in NSW, and discusses the opportunities to produce measurable enhancements to food safety from these investments.

THE EVOLUTION OF FOODBORNE DISEASE SURVEILLANCE IN NSW

In 1990, the Chief Food Inspector established a position of Foodborne Outbreak Investigation Coordinator in the Food Branch of the NSW Department of Health. The Foodborne Outbreak Investigation Coordinator was given authority to investigate outbreaks of foodborne disease, utilising the resources of the Food Branch. From this time, all foodborne outbreaks were reported by food inspectors employed by the NSW Department of Health, who were functionally located throughout the state. All reports were documented centrally by the Foodborne Outbreak Investigation Coordinator. These food inspectors, and the Department's Food Branch, took the lead in the surveillance of foodborne disease.

In 1992, with the administrative transfer of food inspectors to public health units (PHUs), it became the responsibility of the PHUs to report outbreaks to the Foodborne Outbreak Investigation Coordinator in the Food Branch. An outbreak report summary form was developed to assist PHU staff to complete this requirement.

Initially, food inspectors conducted both the environmental and epidemiological investigation of outbreaks and followed up sporadic cases of salmonellosis. However, over the last 10 years, the role of food inspectors has focussed more on the environmental aspects of outbreak investigations, as epidemiologists—both in the PHUs and the NSW Department of Health—began taking the lead on the epidemiological aspects of those investigations. This evolution continues, with the transfer, in 2004, of all NSW Health food inspectors to a new single agency responsible for ensuring safe food production in NSW,[1] which will be an enhancement and an expansion of the current SafeFood Production NSW (SafeFood). However, NSW Health will retain primary responsibility for the surveillance and investigation of illness due to foodborne disease.

The infrastructure for processing data describing foodborne disease has also evolved significantly over the last 10 years. Initially, each case involved in a foodborne disease outbreak was entered into the Infectious Disease Surveillance System (IDSS) database, which later became the Notifiable Diseases Database (NDD). However, each case was entered into the database without a standard set of summary outbreak information, such as the aetiological agent, food vehicle, or setting; therefore, these data could not be analysed to identify prevention opportunities. In 2002, PHUs adopted the OzFoodNet outbreak reporting form, which captured summary outbreak information. From October 2002, PHUs

were no longer required to enter single cases into NDD when cases were notified as members of a group affected by a foodborne outbreak. Instead, summary information on the group was captured solely with the OzFoodNet outbreak reporting form. The information is then summarised at the state level and the data is forwarded to the Commonwealth Department of Health and Aged Care for entry into the national OzFoodNet database of foodborne outbreaks. The summary information for the State has allowed the compilation and publication of useful information, as exemplified in the article by Neville and McAnulty in this issue in the Bulletin.

INVESTMENTS IN SURVEILLANCE CAPACITY

Foodborne disease surveillance is evolving in NSW, with enhancements to both the epidemiological and laboratory capacity and coordination. The appointment of a full-time enteric disease epidemiologist in the Communicable Diseases Branch in July 2002; the appointment of a full-time epidemiologist in OzFoodNet in September 2002; and ongoing collaboration with the Hunter Sentinel OzFoodNet site, are all signals of a significant commitment to foodborne disease surveillance and investigation in NSW. In addition, in 2003, a senior microbiologist was appointed to the position of Public Health Laboratory Liaison Officer (NSW Health), and the NSW Enteric Diseases Advisory Committee was initiated to advise NSW Health on epidemiological and laboratory issues for the control of enteric disease. The outlook for foodborne disease surveillance in NSW is excellent.

Variation in outbreak reporting

Variations in clinical practice among the area health services have become a focus of quality improvement. In the early 1970s, when marked differences in clinical practice were first identified in the United States, there was a simplistic preoccupation with identifying 'the bad apple' based on the variation documented.[2] Clinical quality improvement processes have matured; variations in clinical practice are now seen as opportunities for quality improvement.[3] Consequently, in 2004, variations in surveillance data should be examined, to improve public health systems.

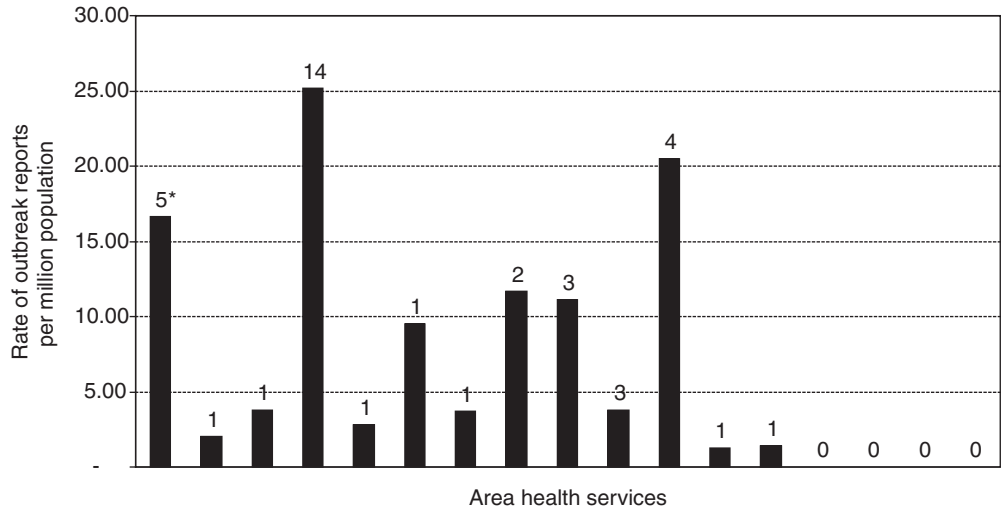
Based on the variation in outbreak reporting rates by area health service (AHS), there are still opportunities to improve the completeness of reporting outbreaks of foodborne disease in NSW. Rates of reported foodborne outbreaks will depend on: the willingness of cases to report their illness; the history taking, testing, and notification practices of doctors; the laboratory methods employed; and the investigation and reporting practices of the PHU.[4] In 2002, the number of outbreaks reported in each AHS varied from 0 to 14, a rate of 0 to 25 outbreak reports per million population. Four AHSs (24 per cent), including those with large and small populations, reported no outbreaks (Figure1). Investigation of this variation will very likely enhance the performance of our outbreak surveillance system.

Timeliness of *Salmonella* reporting

At the public health unit level, an analysis of the time between the collection of specimens and the receipt of

FIGURE 1

RATE OF FOODBORNE OUTBREAKS PER MILLION POPULATION AND NUMBER OF FOODBORNE OUTBREAKS REPORTED FOR 17 AREA HEALTH SERVICES, NSW, 2002



Source: Hunter OzFoodNet Sentinel Site, unpublished data.

results of *Salmonella* serotype or phage type reveals significant delays—particularly in phage typing—which hamper both identification and response to outbreaks (Hunter Public Health Unit, unpublished data). Phage typing is critical for epidemiologically-meaningful clusters or outbreaks to be identified from among the more common serotypes of *Salmonella*, such as *Salmonella* Typhimurium. However, because of a range of issues including quality assurance, specialisation, economies of scale, and even convention, only two laboratories in Australia are able to phage type *Salmonella* and neither are in NSW. This leads to inevitable delays, as NSW laboratories package and despatch interstate approximately half of all *Salmonella* isolates. The Institute of Medical and Veterinary Science in Adelaide, and the Microbiological Diagnostic Unit in Melbourne, provide phage typing services, with rapid turnaround from the time the isolate arrives at their laboratories.

Questions arise as to whether another subtyping method, other than phage typing, should be applied in NSW to speed up the process; however, there are national implications that need to be considered. While a molecular subtyping method could be used in NSW to reduce the delay in recognition of outbreaks by many days, this could create the problem of splitting Australia's *Salmonella* subtyping systems into two that have incompatible methods. If NSW is to trial a new subtyping system, it will be important to ensure compatibility between existing phage typing and the new system, by demonstrating that it is able to map to the current system. This is required to ensure that no connection is lost with either historical surveillance data from NSW or future data from other states and overseas.

The importance of Norovirus reporting

Norovirus, previously referred to as Norwalk-like viruses, may be the most common aetiology of foodborne disease in Australia, as is hypothesised to be the case in the United States.[5,6,7] While rarely fatal, the highly infectious nature of this organism can result in highly visible events. Closure of health care facilities and the cancellation of cruises are examples of the measures required to allow effective decontamination to extinguish an outbreak. Oyster-associated outbreaks of Norovirus have occurred in NSW, as have outbreaks thought to be associated with either symptomatic or convalescing foodhandlers.[8] Two NSW laboratories, and other laboratories around Australia, currently offer polymerase chain reaction (PCR) testing for Norovirus, and the University of NSW has an active research program in this area.[9] Because of the variability in sensitivity, and particularly sensitivity by genogroup, of this PCR test, it would be useful to have a national collaboration to identify the optimal primers, protocols, and reference reagents to better define the epidemiology of these diseases in NSW and throughout Australia.[10] Newer diagnostics, such as Enzyme Immunoassay (EIA), which allow more rapid turnaround times, albeit with lower sensitivity than PCR, are becoming available and their roles need to be considered.

Notification of campylobacteriosis

As we begin to benchmark our performance against other states, it will be important to consider the issue of campylobacteriosis, as NSW is the only state in which campylobacteriosis is not notifiable. Campylobacteriosis is the most common bacterial foodborne infection in Australia, which results in significant morbidity and occasional mortality. Notification of this disease has increased dramatically in all other states and territories from 1991 to 2002 (8,813 cases to 14,619 cases, or 165 per cent),[11] and it is reasonable to presume a similar increase in incidence in NSW over this time. If the national rate for 2002 of 112 cases per 100,000 is applied to the population of NSW, approximately 7,280 cases of campylobacteriosis would have occurred in NSW in 2002 compared to 2,094 cases of salmonellosis for the same period.

Many benefits could accrue to NSW if campylobacteriosis was made notifiable under the *Public Health Act 1991*. We could monitor trends, identify outbreaks, and study risk factors for infection through, for example, the recent national case-control study of campylobacteriosis. Additionally, national campylobacteriosis surveillance data would no longer underestimate the national burden of this disease by approximately 30 per cent.

There is concern that significant public health and laboratory resources could be consumed through notification and follow up of campylobacteriosis cases. There is also a reluctance to make these cases notifiable until direct electronic notification by laboratories to PHUs can be achieved for all notifiable conditions. However, a minimalist approach could be taken. If campylobacteriosis was notifiable, NSW Health could require quarterly or annual electronic downloads from laboratory computers to document trends in incidence and distribution by age, gender, season, and region. This would allow the monitoring of the effect of food safety programs designed to control campylobacteriosis. Promising initiatives to improve subtyping of *Campylobacter* isolates will enhance cluster investigations and make notification of greater public health benefit.

Enhanced laboratory infrastructure

Laboratory infrastructure is being enhanced to allow a broader range of testing for foodborne pathogens at the Institute of Clinical Pathology and Medical Research (ICPMR) in Sydney including testing for *Bacillus cereus* toxins, *Staphylococcus aureus* toxin, and *Clostridium perfringens* enterotoxin. In 2003, pulsed field gel electophoresis (PFGE) performed at the ICPMR has been useful in monitoring cases of listeriosis in a timely manner, to rule out any foodborne disease clusters, and PFGE will be an important public health resource in the future. The

development of a PFGE for *C. perfringens* would help to confirm clusters, where stool collection occurs too late to allow confirmation by conventional quantification of spore counts.

The role of public health units

NSW public health units perform frontline surveillance and investigation of foodborne disease infections and outbreaks. Their location in each area health service of NSW means that they can be more reactive to local conditions. However, this demands greater coordination and cooperation to achieve statewide policy outcomes. Because this network of PHUs, with their links with local and reference laboratories, covers approximately one-third of the national population, it provides information critical to national foodborne disease surveillance and control. PHUs are moving to a performance-based quality improvement process for foodborne disease surveillance. At the August 2003 meeting of the PHU Directors Forum, a range of quality initiatives was adopted. These measures included an annual review of the timeliness of *Salmonella* notifications, including feedback on the delay at each laboratory, to encourage slower laboratories to improve their performance. Local protocols for triggering the initiation of outbreak investigations will be developed, and PHUs will enhance their relationships with general practitioners to promote reporting of foodborne outbreaks. To improve the timeliness and completeness of outbreak information, PHUs will fast-track the completion of OzFoodNet outbreak report forms.

CONCLUSION

The new investments in the epidemiological and laboratory infrastructure supporting foodborne disease surveillance in NSW, described in this article, should provide insights into the causes of foodborne disease in the state. The challenge will be to translate these insights into food safety policy. The launch in 2004 of a dedicated agency in NSW responsible for safe food production should provide an excellent framework for epidemiologically-driven food safety policy. A future issue of the *NSW Public Health Bulletin* will focus on the role of this new agency, the NSW Food Authority.

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HEPATITIS A OUTBREAK ASSOCIATED WITH A MOTHERS' DAY 'YUM CHA' MEAL, SYDNEY, 1997

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This article describes the investigation of an outbreak of hepatitis A that occurred among 'yum cha' patrons at an eastern Sydney restaurant on Mother's Day, 1997.

BACKGROUND

In New South Wales, hepatitis A is notifiable by medical practitioners on clinical suspicion of acute viral hepatitis, and by laboratories on detection of IgM specific for hepatitis A virus (HAV). Hepatitis A, which is generally acquired by ingestion,[1] has been linked to the consumption of raw, or improperly cooked, contaminated foods (such as oysters and salad),[2,3] contaminated water,[4] and food contaminated by infected food handlers.[5]

THE OUTBREAK

On 11 June 1997, staff of the Sydney Children's Hospital and the Prince of Wales Hospital notified the South Eastern Sydney Public Health Unit of three cases of suspected hepatitis A. All three had eaten a 'yum cha' meal at the same restaurant on Mothers' Day, 11 May 1997. Over the following week, laboratories and general practitioners notified further cases, who had eaten at the same restaurant on the same day.

On 12 June, the manager provided the details of 52 parties representing at least 372 patrons who had made dining reservations for 11 May. The aim of the ensuing epidemiological and environmental investigation was to determine the source of hepatitis A infection and prevent further cases.

METHODS

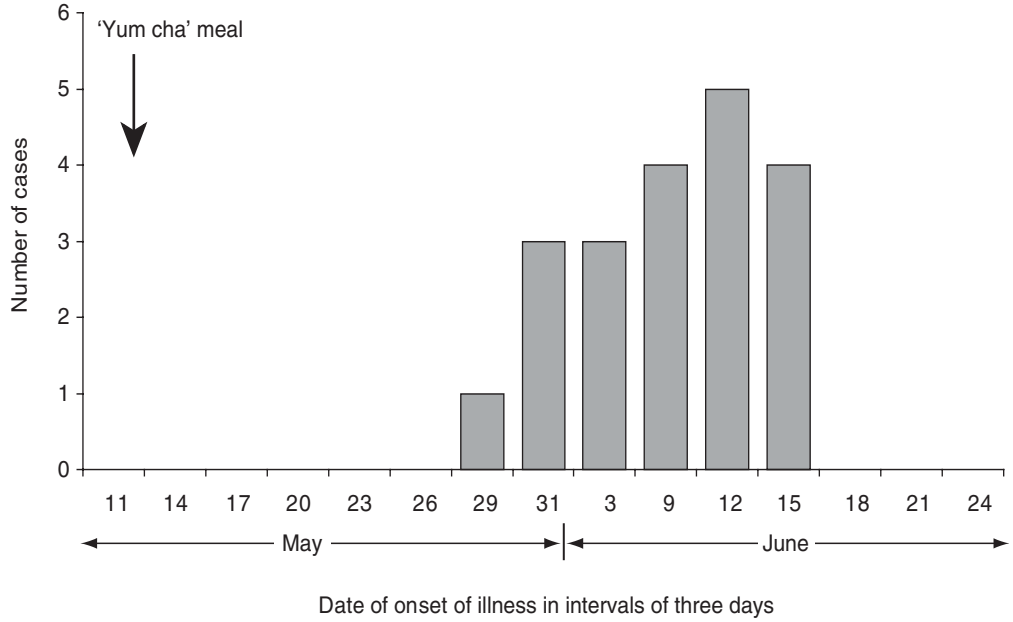
Epidemiological investigation

A case-control study was conducted using a standardised questionnaire that was administered by telephone. The questionnaire covered: demographics; number in party; time of meal; types of meat, seafood, noodles, vegetables, and desserts consumed; types and onset date of symptoms compatible with hepatitis A or gastroenteritis experienced after the meal; use of eating utensils, and use of restaurant toilets.

Cases were defined as: people who had eaten at the restaurant on Mothers' Day, developed serologically confirmed hepatitis A with onset between 26 May and 30 June 1997, and were notified by 9 July. Two cases were excluded because information on foods eaten was not available by the time of analysis.

FIGURE 1

DATE OF ONSET OF HEPATITIS A CASES ASSOCIATED WITH 'YUM CHA' MEAL, SYDNEY, NSW, MAY TO JUNE 1997



Source: South Eastern Sydney Public Health Unit

Controls were: people selected from the reservation list who ate at the restaurant on Mothers' Day, who were contactable, and who had not developed symptoms of hepatitis A by the time of interview, between 17 and 19 June. Controls were not tested for hepatitis A.

Data were entered into and analysed using EpiInfo version 5. Statistical methods used were odds ratio (OR) with 95 per cent confidence intervals for comparison of exposures; two-tailed Fisher exact test where an expected value was less than 5; and the Kruskal-Wallis test for comparison of means.

Environmental investigation

On 12 June, food inspectors reviewed food-handling procedures at the restaurant and took samples of frozen shrimp, the only food that was served on Mothers' Day that remained. The manager provided a list of the food items served for 'yum cha' that day.

All of the 20 food handlers identified as having worked on Mothers' Day were tested for HAV-specific IgM and total antibodies.

Microbiological analysis of shrimp

Six samples of frozen shrimp were collected, comprising two leftover samples from the restaurant, and four other samples from the same shipment of shrimp meat still in storage with two suppliers. Samples were tested for bacteria using standard microbiological methods, and by polymerase chain reaction (PCR) for hepatitis A virus and enterovirus nucleic acid.[6]

RESULTS

Epidemiological investigation

Nineteen cases satisfied the case definition, 10 males and nine females, who were 7–52 years of age (mean 30 years). The first case became symptomatic on 29 May and the last became symptomatic on 9 June (Figure 1). The mean incubation period was 25 days, with a range of 18–29 days. Among the cases, symptoms were typical of acute hepatitis and included fever (90 per cent), nausea (84 per cent), dark urine (84 per cent), anorexia (74 per cent), jaundice (74 per cent), vomiting (63 per cent), abdominal pain (63 per cent), pale faeces (47 per cent), joint pain (42 per cent), and diarrhoea (37 per cent).

Of the 71 controls**,** there were 30 males and 41 females. Their ages ranged from three to 82 years of age (for two adults their age was unknown) with a mean age of 44 years. Controls were significantly older than cases (Kruskal-Wallis test, $\chi^2 = 8.6$, $df = 1$, $P<0.01$). There was no difference in sex distribution between cases and controls (OR=0.7, 95 per cent CI 0.2–2.1).

Controls began to eat between 9.30 a.m. and 2.00 p.m. and cases began to eat between 10.00 a.m. and 1.00 p.m., but there was no difference in the proportion who began to eat before 12.00 p.m. or later (OR=1.3, 95 per cent CI 0.4–4.1).

The case-control study indicated an association between eating shrimp and developing hepatitis A (OR undefined, two-tailed Fisher exact test $P = 0.01$) (Table 1). All five who had eaten pork ribs had also eaten shrimp. There was no association between developing hepatitis A and having eaten steamed or fried noodles, various vegetables, or desserts. No case had eaten oysters.

Although for males, there was an association between using the restaurant toilet (used by both males and females) and developing hepatitis A (two-tailed Fisher exact test $P < 0.005$), there was no association for females ($P = 0.24$).

Environmental investigation

The restaurant had a seating capacity of around 200 patrons. Yum cha was served from 9.30 a.m. till about 2.00 p.m., with more than one sitting. On 11 May, 20 food handlers were reported to have worked as kitchen hands, waiters, or chefs. Their ages ranged from 21–55 years (mean 38 years). Six were females and 14 were males. All tested HAV-specific IgM negative and 18 (90 per cent) were total HAV antibody positive.

Each week, the restaurant received an average of six 16 kg cartons of frozen, raw, fresh-water shrimp. The shrimp used by the restaurant on 11 May were part of a shipment of approximately 10 tonnes imported from Myanmar (Burma) in late February. At the time of the investigation, frozen shrimp from the same shipment was still being distributed to various suppliers and restaurants. Neither the importer nor supplier kept records of batch numbers of shrimp that were sold to this restaurant.

Microbiological analysis of shrimp

The microbiological testing indicated low-level contamination with faecal coliforms and *E. coli.* In addition, two unusual *Salmonella* serotypes were isolated from two of the samples (Table 2). All samples were negative for hepatitis A virus, enterovirus, and *Vibrio* species.

Control measures

The South Eastern Sydney Public Health Unit contacted restaurant patrons, whose phone numbers were recorded in the reservation book, to advise them and their party members of the possible risk of hepatitis A. We also informed local hospitals and general practitioners of the outbreak, giving recommendations for contract tracing and prophylaxis. The NSW Department of Health issued a media release, in order to inform other restaurant patrons of the potential health risk and action to take. The manager voluntarily closed the restaurant.

TABLE 1

FOODS CONSUMED BY HEPATITIS A CASES AND BY CONTROLS AT 'YUM CHA' MEAL, SYDNEY, NSW, 11 MAY 1997

	Cases (*n*=19)		Controls (*n*=71)		Odds ratio	(95% CI)
	n	%	*n*	%		
Seafood						
Shrimp	19	100.0	52	73.2	∞	*
Mussels	1	5.2	5	7.0	0.8	(0.0–7.9)
Calamari	4	21.1	21	29.6	0.7	(0.2–2.6)
Crab	1	5.2	13	18.3	0.3	(0.0–2.2)
Meat Dishes						
Pork ribs	5	26.3	3	4.2	8.7	(1.5–54.8)
Chicken feet	4	21.1	7	9.9	2.6	(0.5–12.2)
Pork	11	57.9	46	64.8	1.0	(0.3–3.5)
Chicken	8	42.1	40	56.3	0.7	(0.2–2.3)
Beef	3	15.8	21	29.6	0.5	(0.1–2.2)
Other items						
Oyster sauce	9	47.3	23	32.4	2.4	(0.7–8.0)
Lettuce	2	10.5	4	5.6	2.1	(0.2–15.5)
Egg custard	4	21.1	16	22.5	1.0	(0.2–3.9)

* Fisher exact two-tailed *p*-value <0.01

Source: South Eastern Sydney Public Health Unit

TABLE 2

MICROBIOLOGICAL ANALYSIS OF SAMPLES OF SHRIMP MEAT ASSOCIATED WITH 'YUM CHA' MEAL, SYDNEY, NSW, 11 MAY 1997

Sample Source	SPC/g	Faecal coliforms/g	*E. coli*/g	*Salmonella*/25g
Restaurant 1	$1.2X10^8$	<3	<3	Not Detected
Restaurant 2	$4.1X10^6$	9	9	*S.* hvittingfoss
Supplier A1	$6.1X10^5$	<3	<3	Not Detected
Supplier A2	$6.4X10^5$	4	<3	Not Detected
Supplier A3	$3.4X10^6$	<3	<3	*S.* paratyphi B var Java
Supplier B1	$1.2X10^6$	23	9	Not Detected

SPC = standard plate count

Source: South Eastern Sydney Public Health Unit

DISCUSSION

This investigation found that the most likely cause of the outbreak was consumption of contaminated shrimp that were inadequately cooked. Shellfish implicated as a possible source of hepatitis A in the past have been raw oysters,[2,7] and steamed or raw clams,[7,8] but not shrimp.

HAV is viable for at least 10 days when stored at -20°C, [1] and for up to one month when stored dry at room temperature,[9] and hence would survive in frozen shrimp. When experimentally suspended in phosphate buffered saline, HAV is inactivated by heating at 70°C for four minutes, at 75°C for 30 seconds, at 80° C for five seconds, or virtually instantaneously at 85°C. However, HAV is inactivated in contaminated oyster and clam cockles once they have been cooked in water at 95°C for one-and-a-half minutes after their shells open.[10]

During cooking, the steamer baskets holding the delicately-parcelled dumplings of chopped shrimp are stacked, up to seven baskets high, over the boiler. As long as there is sufficient heat and water to maintain the flow of steam within the system, the cooking temperature should be relatively constant throughout the stack. However, environmental factors may hinder the core temperature from reaching the temperature required to inactivate the virus. Frozen food would require more time to reach the inactivation temperature than food held at room temperature. Other ingredients used in the preparation of the shrimp dumplings could insulate the virus from external heat. Also, pressure to serve more people than usual, as may have occurred on Mother's Day, may reduce the cooking time and so lower the core temperatures reached.[2]

It is unknown how these fresh-water shrimp were contaminated. The importer stated that they were harvested from a river in Myanmar, transported to a processor to be peeled, packed, and frozen for export. Shrimp from the same shipment were supplied to more than one restaurant in Sydney yet only one other patient who developed hepatitis A at the time of this outbreak reported having eaten a shrimp dish from a restaurant that bought from this shipment. It is possible that other recipient restaurants heated the shrimp sufficiently to inactivate HAV, or that only some shrimp in the shipment were contaminated with HAV.

In this outbreak, contamination by food handlers was unlikely, as those food handlers tested were either already immune or were negative for HAV IgM one month after the meal. However, it is possible that the manager may have overlooked casual staff. Finally, we considered the possibility that patrons infectious with hepatitis A may have contaminated the toilet door handle that other patrons then touched (the restaurant toilet was used by both male and female patrons). The analysis did not support this possibility, as there was no association between hepatitis A among female cases and use of the toilet. Possible explanations for why only 27 per cent of shrimp eating patrons interviewed developed hepatitis A are that: only some shrimp were contaminated; only some baskets were insufficiently cooked; or some patrons were already immune.

Shrimp may be a potential source of hepatitis A and should always be cooked properly. Only proper food handling and cooking will prevent foodborne hepatitis A.

ACKNOWLEDGMENTS

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NOTIFIABLE DISEASES DATABASE SYSTEM: REVIEW AND DEVELOPMENT STRATEGY

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BACKGROUND

The Communicable Diseases Branch of the NSW Department of Health recently undertook a review of the existing notification system, to determine its effectiveness and guide future development of the system. This report briefly outlines the nature of the review, its recommendations, and the progress to date in addressing the recommendations. A more complete description of the nature and outcome of this process will be presented in a future edition of the *NSW Public Health Bulletin*.

THE CURRENT SYSTEM

Under the *NSW Public Health Act 1991*, certain Scheduled Medical Conditions (SMCs) are required to be notified. The Notifiable Diseases Database (NDD) system is a tool for maintaining data related to notifiable communicable conditions. The primary objective of the NDD system is to provide timely and accurate data on notifiable communicable diseases in a flexible and secure manner.

The NDD system has been operational for over 10 years in a number of forms. The current system has been operational since 1997, with several updates.

The system covers all 17 area health services in NSW, and since early 2003 has also included Corrections Health as a separate area health service. Information on over 40 notifiable SMCs is maintained by the system, with over 30,000 cases notified in 2003. On average, 600 new records are added to the system every week.

To guide public health action, the NDD system encompasses the collection, analysis, interpretation, and dissemination of data regarding certain SMCs. The information generated by this system is used to:

- record the public health interventions and outcomes associated with the management of each case of a notified condition;
- assess the current status of individual conditions or disease groups;
- identify specific groups at risk;
- provide quantitative evidence related to control strategies;
- understand the epidemiology of specific conditions;
- inform the development of prevention strategies and policies.
- prioritise the allocation of resources.

STRUCTURE OF THE NOTIFIABLE DISEASES DATABASE SYSTEM

The goal of any information system is the manufacture of appropriate information products for end users. Data must be captured and prepared for processing by the *input* activity. Data are typically subjected to *transforming* activities such as calculating, comparing, sorting, classifying, and summarising. Information in various forms is transmitted to users and made available in the *output* activity. Data *storage* ensures that information is retained in an organised manner for later use. An information system should produce feedback regarding its input, processing, output, and storage activities in order to *control* performance.[1]

The NDD system can therefore be considered as having the following components:

- system control;
- data input;
- data transformation;
- information output;
- system architecture.

REVIEW METHODOLOGY AND FINDINGS

In October 2002 the Communicable Diseases Branch of the NSW Department of Health engaged the services of a consultant to evaluate the effectiveness of the NDD and make recommendations on the future development of the system.

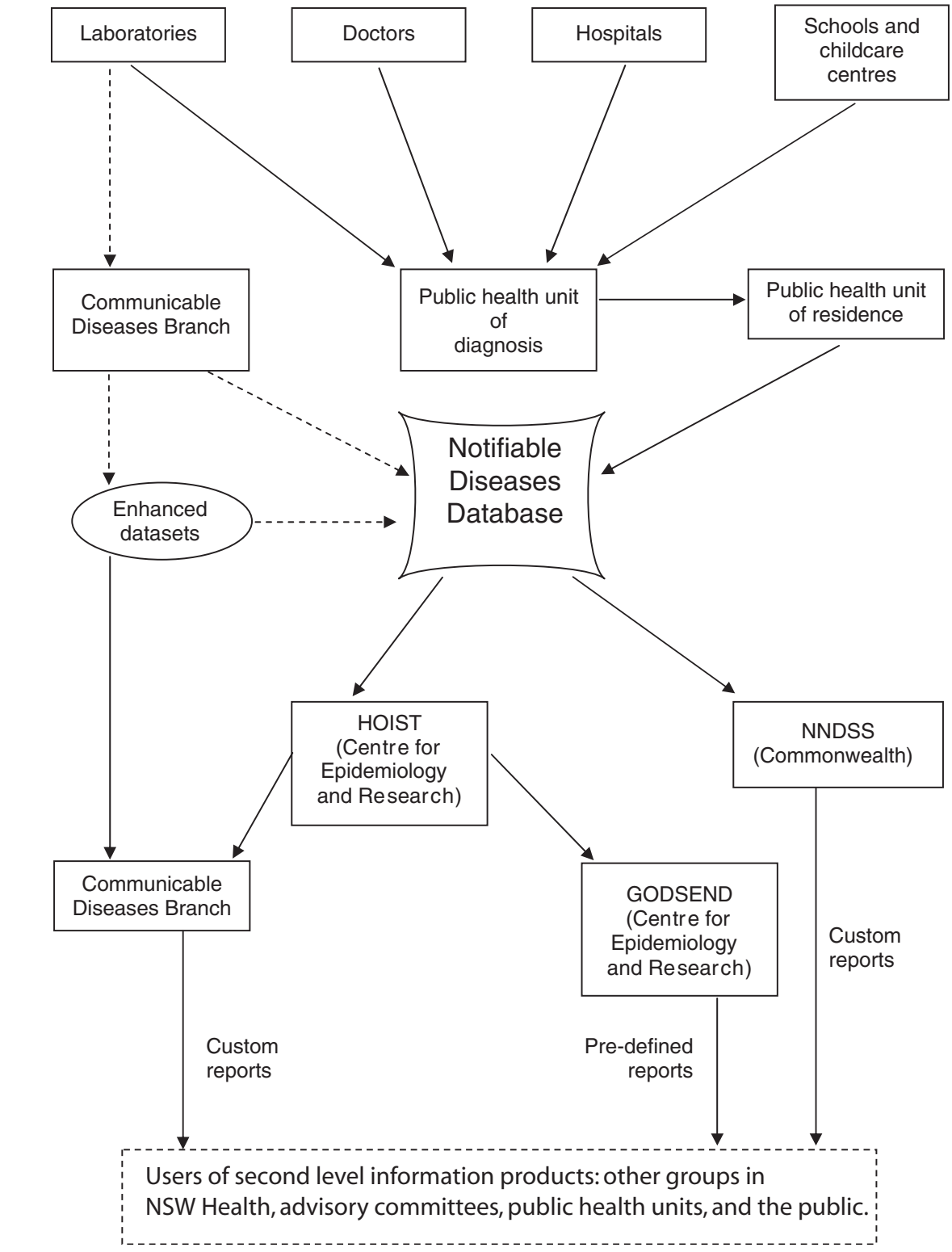
All information systems use people, hardware, software, network and knowledge resources to perform input, transformation, output, storage and control activities that convert data resources into information products.[1] Multiple sources of information from all these inter-related elements were needed to construct a balanced and reliable description of the system and provide a frame of reference for evaluating the system.

Representatives of stakeholder groups were identified to provide input into the review. Stakeholders included:

- *system control and system architecture*—staff from the Communicable Diseases Branch and the Enterprise Information Technology Branch;
- *data input*—public health unit surveillance staff and directors, pathology laboratories, medical practitioners, schools, and hospitals.
- *data processing and information output*—staff from the Commonwealth Department of Health and Ageing, the Centre for Epidemiology and Research, the AIDS and Infectious Diseases Branch, advisory committees, and the public.

FIGURE 1

THE RELATIONSHIP BETWEEN STAKEHOLDERS INVOLVED WITH THE NOTIFIABLE DISEASES DATABASE SYSTEM AND THE GENERIC FLOW OF DATA WITHIN THE SYSTEM



Note: HOIST = Health Outcomes Information and Statistical Toolkit
GODSEND = Graphical Online Data Surveillance and Evaluation for Notifiable Diseases
NNDSS = National Notifiable Diseases Surveillance System

Source: Communicable Disease Branch, NSW Department of Health.

An evaluation form was developed and stakeholder representatives were invited to use this to provide their perspective on the NDD system. A number of key stakeholders were also interviewed to collect information on operational aspects of the system. Existing documentation and current work practices were also reviewed.

ANALYSIS

The information collected from these sources was used to assess the effectiveness of the system by comparing current operations with established objectives and specific stakeholder requirements. In accordance with the *Guidelines for Evaluating Public Health Surveillance Systems* from the United States Centre for Disease Control and Prevention,[2] the following performance characteristics were considered: simplicity, flexibility, data quality, acceptability, sensitivity, predictive value positive (PVP), representativeness, timeliness and stability.

FINDINGS

The review found that the NDD system is currently limited in its ability to meet its primary objective of providing timely and accurate data on notifiable conditions in a flexible and secure environment.

The following barriers to further development were identified:

- the roles and responsibilities of different stakeholder groups involved with the system are unclear;
- the nature and type of data collected in the system has grown, and will continue to grow, in an informal way. The existing system cannot readily facilitate change, so alterations are resource intensive and untimely;
- data do not flow through the system consistently and is subject to different transformation rules depending on the way information is accessed.

The recommendations from the review were categorised into those relating to improving the existing system, and those guiding the development of a new system.

Recommendations to improve the existing database cover all aspects of the system from system control, data input, data transformation, and information output through to system architecture. The proposed improvements to the existing system will also clarify the requirements of a new system.

Recommendations guiding the development of a new system included the preliminary phase of a comprehensive systems analysis, which should encompass the principles of problem definition, system specification, system design, system development, and ongoing review and maintenance.

PROGRESS ON IMPLEMENTING THE REVIEW RECOMMENDATIONS

To date, several of the review recommendations have been implemented or are in the process of being implemented. These include:

- rationalising and strengthening the documentation surrounding the existing system;
- reviewing the existing network infrastructure in relation to capacity to support a web based information system;
- evaluating the feasibility of electronic transfer of laboratory-based notifications;
- reviewing privacy and security requirements of a notifiable conditions system;
- developing data conventions to ensure consistent data flow and processing for all notifiable conditions.

In the coming months, projects addressing these recommendations will be finalised and the remaining recommendations will be implemented.

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FOODBORNE DISEASE SURVEILLANCE NEEDS IN AUSTRALIA: HARMONISATION OF MOLECULAR LABORATORY TESTING AND SHARING DATA FROM HUMAN, ANIMAL, AND FOOD SOURCES

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Foodborne diseases cause significant morbidity and mortality in Australia and throughout the world. Outbreaks of foodborne disease often require investigators to collaborate across jurisdictional boundaries—even at times internationally. Notified cases of foodborne disease are only a small proportion of the total burden of foodborne disease affecting the community. Many pathogens that contaminate food are zoonotic in origin (that is, are transmitted to humans from lower vertebrates), although the pathway from animal to human via foods is complex and difficult to understand. This article describes how our ability to understand and control foodborne diseases in Australia can be enhanced through improving our surveillance datasets by harmonising methods for advanced microbiological testing of foodborne organisms, and sharing data obtained from human, food, and animal sources.

BACKGROUND

The World Health Organization and many countries around the world have recognised the importance of foodborne disease and the necessity of improving surveillance for this.[1,2] Routine surveillance of foodborne disease relies on reports from doctors or clinical laboratories regarding people diagnosed with gastrointestinal or foodborne infections. In Australia, there are several infections that are notifiable to health agencies, including: *Salmonella*, *Campylobacter*, toxigenic *E. coli*, and listeriosis.[3] These agencies maintain well-organised collections of data describing human infections, which have proven useful for determining trends and identifying outbreaks, particularly at the state and territory level and more recently at the national level.[4,5]

The nature of foodborne disease investigations has changed significantly, with more complex and wide-ranging investigations becoming the norm.[2] Contemporary outbreaks are more geographically widespread than they were in the past, and may be solved with smaller numbers of cases.[6] Increasingly, investigators rely on advanced microbiological evidence to supplement field epidemiology.[6,7] The overriding goal of investigations is to prevent further cases, either by removing contaminated food from the marketplace, or by changing policy or practice to avoid future events of contamination. These changes represent a new paradigm in outbreak investigation (Table 1).[8]

Despite these developments in surveillance and investigation methods, there are still many areas for improvement. Each year health agencies investigate many notified infections and apparent outbreaks. In the investigation of some foodborne outbreaks, they may be able to identify the vehicle of infection, which is the specific food people ate before becoming ill. However, it is far more difficult to identify the original source of contamination, which may be an infected human or animal or a flaw in handling the food.

Of the 7,917 cases of laboratory-confirmed *Salmonella* infection notified to health departments in 2002, 543 cases were linked to foodborne or waterborne outbreaks (OzFoodNet, unpublished data). For certain infections, the 'success rate' of linking notifications with outbreaks may be considerably lower. For example, while *Campylobacter* is the most common enteric infection notified to health departments throughout Australia—despite it not being notifiable in New South Wales—and

TABLE 1

THE CHANGING PARADIGM OF FOODBORNE DISEASE OUTBREAK INVESTIGATIONS

Old Strategy	New Strategy
Culture all leftover foods	Develop and test hypotheses
Action based on pathogen in food	Evaluate exposure in ill and well
Assume someone broke the rules	Take action on statistics
Goal	**Goal**
Assign blame	Prevent it happening again
Treat industry as a perpetrator	Treat industry as a collaborator
Be sure your evidence holds up in court	Be sure your data are scientifically valid

Source: Hedberg CW, Rigdon CE, Osterholm MT. *White Paper on Applied Epidemiology*. Food Safety and Inspection Service.[8]

in 2002 there were 14,716 notifications of infection; of these only 24 cases were subsequently linked to outbreaks.

In early 2001, *Salmonella* Typhimurium 170 infections increased dramatically in the eastern states of Australia, with case numbers rivalling the most common *Salmonella* serovars (Figure 1). Health departments conducted intensive investigation of over 100 cases, although no source was definitively identified. Hypothesis generating interviews of cases revealed that many had putative exposures to consumption of red meat and/or poultry and livestock contact. A trace-back investigation of foods mentioned by patients failed to identify any common vehicle for the increase. State and territory health departments investigated an additional 45 clusters of salmonellosis during 2002 where no source was identified.[9] These investigations show the difficulty of identifying the cause of outbreaks, and that our strategies may need to be reviewed.

Enhancing our ability to correctly attribute infections to food vehicles and sources should also focus on combining epidemiological investigation of human cases, with data from advanced laboratory testing and hazard surveillance of animals and foodstuffs. Some potential improvements to surveillance of foodborne organisms are briefly discussed below.

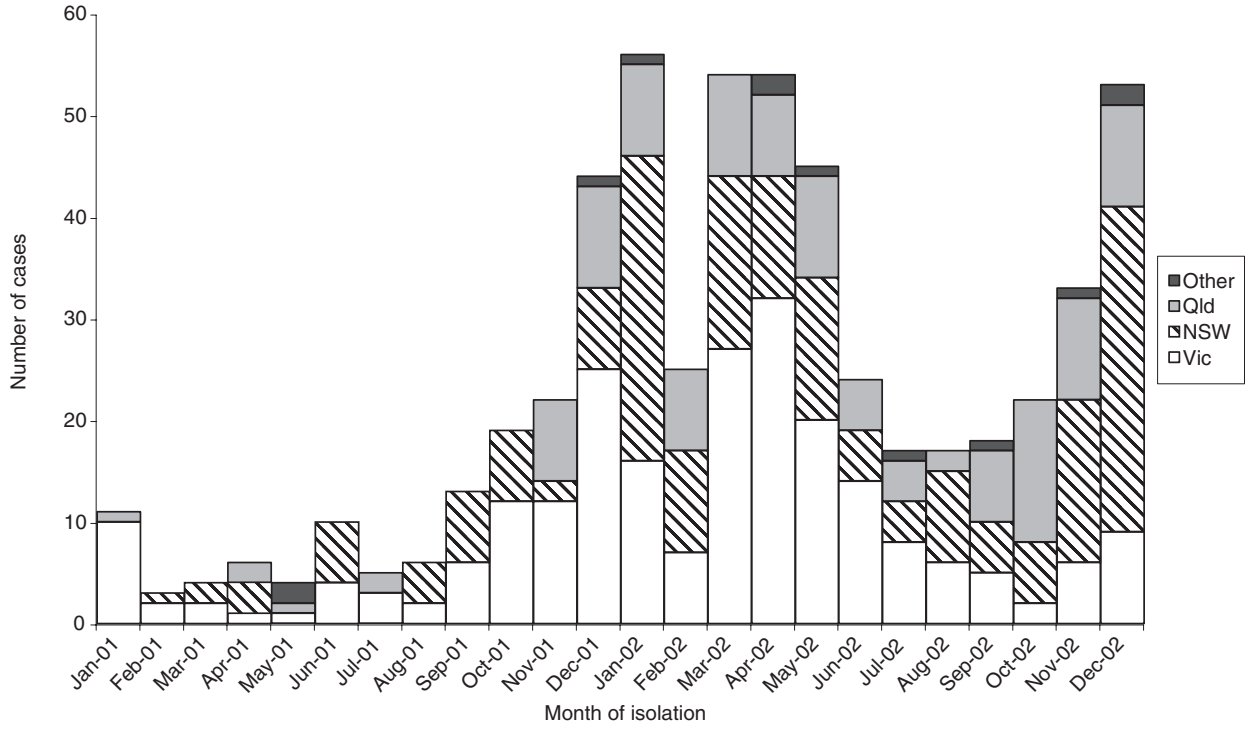
ADVANCED LABORATORY TESTING

Most state and territory reference laboratories in Australia regularly characterise pathogens using advanced methods of analysis, such as serotyping, antimicrobal testing, and gene sequencing. Despite this, for some organisms there is a lack of standardisation of testing across Australia and the results may not find their way into surveillance datasets. This leads to inadequate epidemiological information at the jurisdictional level and difficulties interpreting national data.

Typing of *Salmonella enterica* using standard panels of antisera is an example of a very successful typing scheme that assists health departments identify outbreaks of foodborne disease. This success is due to the typing scheme being able to distinguish over 2000 epidemiologically-distinct serotypes that are stable over time. The success of *Salmonella* surveillance relies on an epidemiologically robust scheme that divides *S. enterica* into serotypes and phage types. The National Enteric Pathogen Surveillance Scheme (NEPSS) has collected data on serotypes and phage types infecting humans, animals, foods, and the environment, for many years.[10] These data are a national resource that assists epidemiologists investigate outbreaks. The success of the scheme has relied on the cooperation of microbiological laboratories, and

FIGURE 1

REPORTS OF *SALMONELLA* TYPHIMURIUM PHAGE TYPE 170, BY STATE OR TERRITORY AND MONTH OF ISOLATION, JANUARY 2001 TO DECEMBER 2002



Source: Data from the National Enteric Pathogen Surveillance Scheme, March 2003.

the existence of a robust typing system for these organisms.

The Centers for Disease Control and Prevention in Atlanta have developed the PulseNet system of surveillance for typing human isolates of *Salmonella, Listeria,* and *E. coli* O157:H7 using Pulsed Field Gel Electrophoresis (PFGE).[11] PFGE is a technique based on the analysis of bacterial DNA, which allows the assignment of a characteristic genetic fingerprint. The PulseNet system relies on harmonised laboratory protocols for subtyping a restricted number of foodborne organisms, and comparing the results to detect outbreaks with a common source. The system has proven valuable for identifying the source of outbreaks and provides a library of DNA patterns for future reference.

The PulseNet system has been instituted in Canada. Europe also commonly uses PFGE as a complementary tool to traditional phenotypic methods of analysis. This has developed into a similar network—Salm-gene—to allow the exchange of PFGE profiles for common *Salmonella* serotypes across Europe.[2,13] There are current plans to increase coverage of these PFGE networks to other regions of the globe, including Asia and the Pacific.

In 2000, the Australian Public Health Laboratory Network (PHLN) conducted a trial of sharing PFGE patterns of *Listeria* isolates (personal communication from Geoff Hogg, University of Melbourne, 25 March 2003). Ideally, Australia would build on this trial to routinely type and share molecular typing information due to the serious nature of listeriosis. The rapid sharing of typing data could improve the detection of clusters of illness that are spread across different geographical regions. This would require resources and harmonisation of PFGE typing methods in state and territory public health laboratories. PFGE surveillance of these infections is desirable for the following reasons:

- *Listeria* has the potential to cause widely distributed outbreaks;
- the PHLN trial demonstrated the feasibility of such a typing network in Australia;
- the small numbers of isolates for testing each year (approximately 60) represents a manageable workload for laboratories.

The key features of this surveillance are that it should be timely, accurate, and able to assist with detection of clusters and outbreaks. Harmonised PFGE typing could also assist jurisdictions investigating multi-state outbreaks of salmonellosis.

FOOD SURVEILLANCE

While Australian health agencies regularly survey the microbiological quality of foods for human consumption, few conduct true surveillance of food hazards. 'Hazard surveillance' is defined as the 'assessment of the occurrence of, distribution of, and the secular trends in levels of hazards (such as toxic chemical agents, physical agents, biomechanical stressors, as well as biological agents) responsible for injury'.[13] It is important that collection of hazard data is ongoing and that the sampling frame is known. The aim of 'hazard surveillance' is to identify trends and emerging patterns that may be associated with human disease. However, in contrast to surveillance of human disease, 'hazard surveillance' is unlikely to identifty the occurrence of foodborne disease outbreaks.

The food supply is extremely complex and difficult to sample and test in a representative fashion. Ongoing systematic data collection describing the microbiological quality of foods is sorely lacking in Australia, despite health departments regularly conducting surveys of specific foods. One source of surveillance data on potential food hazards in Australia is the Imported Foods Database, coordinated by the Australian Quarantine and Inspection Service. Unfortunately, the usefulness of this database to health agencies is limited, due to the heterogeneous nature of imported foods tested, and because the contamination of most imported foods with human pathogens is very rare.[14]

To conduct proper hazard surveillance of foods could prove very costly. An alternative would be to conduct simple surveillance of a few foods in an ongoing fashion. This might require jurisdictions to extend targeted surveys to run over several years and widen the sampling frame to increase the representativeness of the data. Short-term surveys of retail meats focussing on the prevalence of *Salmonella* and *Campylobacter* have proven useful to support policy development.[15,16] The Australia Capital Territory Health Services conducted two surveys of retail meats in 1995–96 and 1999–2000 and found that for retail chicken meats *Campylobacter* prevalence increased from 12.3 per cent to 20.6 per cent.[15] The survey also found that 41 per cent of chicken was contaminated with *Salmonella*, although a large proportion of isolates were the Sofia serotype which is usually only mildly pathogenic for humans. Internationally, food regulators and health agencies have used the data arising from these food surveys to inform policy, particularly in relation to the development of antibiotic resistance in isolates of animal origin.[17]

This surveillance of meats at retail sale may prove to be an indicator of the quality of meats at farms and abattoir processing facilities. In the absence of animal surveillance data, information on contamination of retail foods could reflect exposure to pathogens for Australian consumers, and provide impetus for more integrated monitoring with the agricultural sector.

ANIMAL SURVEILLANCE

Many foodborne diseases have a zoonotic basis, and are commonly associated with foods of animal origin, the use of manure for fertilisers, or direct contact with infected animals. An example of this was an investigation into a

cluster of cases of *Salmonella* Typhimurium 170 occurring in October 2002. The source of the outbreak was identified as hatching chickens at a childcare centre, which was traced back to the flocks of chickens laying eggs (R. Stafford, personal communication, 12 March 2002). The investigation of this zoonotic outbreak provides important clues as to the source of the multi-state increase of these infections earlier in 2002 that was probably due to contaminated foods of animal origin (Figure 1).

Health agencies commonly use available data to inform investigations of sporadic infections and outbreaks due to these pathogens. The most commonly used data from animals, foods, and the environment, are those collected by the NEPSS and the Australian *Salmonella* Reference Laboratory databases. The National Enteric Pathogens Surveillance System dataset is housed at the Microbiological Diagnostic Unit at the University of Melbourne and is funded by the Commonwealth Department of Health and Ageing and state and territory health departments. The Australian *Salmonella* Reference Laboratory database is located at the Institute of Medical and Veterinary Science in Adelaide. These surveillance datasets record important information about the occurrence of specific *Salmonella* serovars and phage types, but the data are not representative due to the ad hoc nature of sampling, typing, and reporting. The National Enteric Pathogens Surveillance System also contains important information about antibiotic resistance of *Salmonella*, which is of vital national interest. Another important source of information can come from monitoring animal feeds, which are also collected in the National Enteric Pathogens Surveillance System.[19]

Industry groups collect information on the occurrence of potential human pathogens in animals, although these may not be available to health investigators in a timely fashion. Often the results are not typed to an adequate level, as there is no treatment benefit to the affected animals. Primary industries are changing to allow more access to their data, although there are still sensitivities surrounding the isolation of human pathogens in food-producing animals.

The Rural Industries Research and Development Corporation—a statutory authority formed in 1990 under the *Primary Industries and Energy Research and Development Act 1989*—recently commissioned a report examining surveillance and response options for the Australian egg industry to monitor for the incursion of *Salmonella* Enteritidis 4.[20] Human infections of this phage type of *S.* Enteritidis are rare in Australia, in contrast to many other countries where internal contents of eggs are contaminated. The proposed surveillance for Australia involves collecting a set number of drag swabs each month from sheds housing chickens used for breeding and laying eggs. This would only occur in larger production facilities, but the program is designed to detect contamination at moderate levels and provide insight to *Salmonella* serovars affecting the poultry and egg industries.

Ideally, government agencies with agricultural responsibilities would encourage and facilitate this type of surveillance of food-producing animals. There are significant costs associated with animal surveillance, but this may be offset by the cost savings of preventing disease in animals and humans.[21] It is important to recognise that these data on animals and the environment provide information on potential sources of foodborne disease, but rarely do they reveal specific foods responsible for outbreaks. Each investigation into foodborne disease outbreaks requires robust epidemiological assessment for possible vehicles, and a trace-back investigation to confirm the original source of contamination.[2]

INTEGRATING SURVEILLANCE

Sweden and Denmark are probably the best-known examples of countries collecting systematic data on *Salmonella* and *Campylobacter* from human, animals, and food sources over many years.[21–23] Integrating surveillance data from these three sources requires continuous intensive surveys of the microbiological quality of animal herds and foodstuffs for retail sale. The predominant serotypes or subtypes detected in these surveys are then compared to the predominant types in humans. This type of surveillance is quite costly, but yields important insights for the control of foodborne disease. Due to the intensive ongoing monitoring of *Salmonella* in foods and animal herds in these countries, they can attribute human infections with common serotypes and phage types to different commodity groups of foods. Another Nordic country—Iceland—was able to observe major declines in human *Campylobacter* infections and decreases in contamination of poultry for retail sale following significant interventions in the poultry industry and consumer education campaigns.[24]

The high incidence of *Salmonella* and *Campylobacter* infections in humans makes integration of surveillance urgent. Integration on the scale practiced by Denmark and Sweden is likely to be very difficult in Australia, due to its geographical diversity and regulatory environment. However, collecting and sharing these data in a timely fashion should be a long-term goal to control diseases transmitted by animals, foods, and environment. This goal was recently recognised as a priority by the Food Regulation Standing Committee, the peak body for Government food safety policy in Australia.

OUTCOMES

Government agencies should consider collecting new surveillance data on animals and foods, but not for regulatory enforcement purposes. The main objective for longer-term surveillance must be to support the prevention of disease in humans and control of contamination in

animals and foods. During disease investigations it is important to be aware of the potential for these diseases to have a zoonotic reservoir, which may have a complex pathway via food to humans. There are some fundamental differences in the surveillance for humans, foods, and animals, which include differences in the nature of sampling for pathogens. For Australia to improve its ability to understand and control foodborne disease, we need to work towards developing integrated surveillance and a systematic approach to molecular typing of infectious organisms that are potentially transmitted by food.

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Fred Angulo from the Centers for Disease Control and Prevention, and Mark Veitch from the Microbiological Diagnostic Unit Public Health Laboratory, provided helpful comments on a draft of this article. The OzFoodNet program of work is an initiative of the Commonwealth Department of Health and Ageing.

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EPI*REVIEW*

COMMUNICABLE ENTERIC DISEASE SURVEILLANCE, NSW, 2000–2002

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This article describes a review of communicable enteric disease surveillance, hospitalisation, and outbreak data, for NSW during the period 2000–2002.

BACKGROUND

Communicable enteric disease (CED), and in particular foodborne disease (FBD), is a major cause of illness in Australia.[1] For the purpose of this article, the term CED encompasses both diarrhoeal and foodborne diseases, which includes illness caused by toxins.

It is estimated that FBD costs the Australian community over $2.6 billion each year.[2] The incidence of FBD in Australia is increasing.[3] Salmonella infection notification rates almost doubled in NSW from 1992 (14 per 100,000) to 1998 (30 per 100,000); however, they declined slightly in 2001 (27 per 100,000).[4]

There are many factors that can influence the incidence of FBD such as: changes in the pattern of food consumption; changes in consumer demand for food; and changes in the method of manufacture, distribution, storage, and selling of food. In addition, the proportion of the Australian population susceptible to CED is increasing, as the elderly are more vulnerable.[3]

Surveillance is key to understanding the epidemiology of CED, estimating its burden on the community, controlling risks, and identifying emerging pathogens. Essential to CED surveillance is clinician and laboratory reporting, analysis, and outbreak investigation.[3]

METHODS

Surveillance

The NSW Department of Health maintains a Notifiable Diseases Database (NDD) that houses data describing communicable diseases notifiable under the *NSW Public Health Act 1991*. The CEDs included in this review were salmonellosis, listeriosis, typhoid, paratyphoid, shigellosis, hepatitis A, haemolytic uraemic syndrome (HUS), and verotoxin producing *E. coli* infections (VTEC). We reviewed notification data for CEDs for the period 2000–2002, including demographic information about cases where available. Area health services were classified as urban or rural according to the NSW Health classification.[5] Notification rates per 100,000 population were calculated using population estimates from Australian Bureau of Statistics (ABS) population data, accessed via the Health Outcomes Information and Statistical Toolkit (HOIST), a data warehouse operated by the Centre for Epidemiology and Research, NSW Department of Health, as at 30 June for each calendar year during the study period.

Hospitalisations due to CEDs were determined using the NSW Inpatients Statistic Collection (ISC) databases and the 10th Revision of the International Classification of Diseases, Australian Modification (ICD-10-AM) codes for principal diagnosis. Data was available for the period January 2000 to June 2002. Analysis was based on admission date.

Outbreaks

All NSW public health units were asked to complete an OzFoodNet outbreak reporting form for all outbreaks of CED identified for the period 2000–2002. All other reports of such outbreaks received by NSW Health for the study period were also included.

We defined institutional settings as: aged care facilities, hospitals, schools, childcare facilities, military institutions, correctional centres, organised camps, and institutional settings not otherwise specified. Non-institutional settings were defined as: restaurants, take-away outlets, fast food franchises, commercial caterers, cruise ships and airlines, grocery stores or delicatessens, fairs and festivals and other temporary–mobile services, picnics, and private residences.

A FBD outbreak was defined as a CED outbreak where two or more people experienced a similar illness after consuming a common food or meal and:

- epidemiological analyses and/or microbiological analyses implicated a food or meal as the source of illness (foodborne);
- a specific food or meal was suspected, but person-to-person transmission could not be ruled out (suspected foodborne).

Surveillance data were accessed through HOIST and were extracted and analysed in February–March 2003 using SAS version 8.[6] The outbreak data were analysed with Microsoft Access 2000 and Microsoft Excel 2000.

RESULTS

Surveillance

The notification rate of CEDs over the three-year period increased from 37.6 per 100,000 population in 2000 (*n*=2431) to 65.6 per 100,000 population in 2002 (*n*=4316).

Over the three-year period, salmonellosis was the most frequently notified CED (Table 1), with notification rates increasing from 20.6 per 100,000 population in 2000 (*n*=1,334) to 32.7 per 100,000 population in 2002 (*n*=2,153). The highest rate of salmonellosis occurred in children less than five years of age and the rate decreased steadily with increasing age. The rate was higher in rural areas than in metropolitan areas. The most frequently reported salmonella serovar during 2000–2002 was *Salmonella* Typhimurium phage type 9, which accounted for 10.3 per cent of all salmonella infections.

The rates of hepatitis A and shigellosis were highest in males, in urban area health service populations and in the 20–39 year old age group. The highest rates of typhoid were noted in urban areas and in the 5–9 and 20–39 year old age groups. The demographics of paratyphoid cases were similar, with the highest rates in the urban areas and in the 20–39 year old age group. Rates of listeriosis were highest in the elderly, in males, and in urban areas. Rates of HUS and VTEC were highest in children in the 0–4 year old age group. The rate of VTEC was higher in females than males and in urban areas. VTEC infections were identified in six of the eighteen cases of HUS.

There were 994 hospitalisations during the 30-month period, January 2000 to June 2002, for which the principal diagnosis was a CED (Table 2). The majority were due to salmonellosis (68 per cent), with the next most common hepatitis A (12 per cent). The median length of stay for patients hospitalised with CEDs ranged from three days (salmonellosis) to 17 days (listeriosis).

Outbreaks

All NSW public health units provided outbreak summary data for the period 2000–2002. There were 308 CED outbreaks reported, of which 191 (62 per cent) occurred in institutional settings, 111 (36 per cent) occurred in non-institutional settings, and six (two per cent) were community-wide. These outbreaks resulted in 6,247 individual cases of illness, 240 hospitalisations, and no deaths. For the majority of CED outbreaks (*n*=235; 76 per cent), a cause was not identified. The most commonly identified causes were enteric virus infection (*n*=45; 15 per cent) and salmonella infection (*n*=19; six per cent). Other causes include infection with campylobacter (*n*=2; one per cent), hepatitis A (*n*=1; 0.3 per cent), ciguatera poisoning (*n*=1; 0.3 per cent), giardia (*n*=2; one per cent), and *Clostridium perfringens* (*n*=3; one per cent).

Setting

Outbreaks in institutional settings

The 191 CED outbreaks in institutional settings included 4,710 individual cases of gastrointestinal illness. Among the institutional outbreaks, the most common settings were

TABLE 1

COMMUNICABLE ENTERIC DISEASE NOTIFICATIONS AND CRUDE RATE PER 100,000 POPULATION, NSW, JANUARY 2000 TO DECEMBER 2002

	Salmonellosis		Hepatitis A		Shigellosis**		Typhoid		Paratyphoid		Listeriosis		HUS***		VTEC***	
	n	r	*n*	r	*n*	r	*n*	r	*n*	r	*n*	r	*n*	r	*n*	r
Gender																
Male	2590	26.6	377	3.9	144	2.2	40	0.41	18	0.19	24	0.25	10	0.10	1	0.01
Female	2544	25.8	168	1.7	69	1.0	40	0.41	18	0.18	17	0.17	8	0.08	5	0.05
Age (Years)																
0–4	1554	110.0	15	0.7	16	1.5	6	0.24	2	0.16	1	0.07	5	0.31	2	0.16
5–9	481	30.8	31	1.9	7	0.7	13	0.75	2	0.07	0	0.00	2	0.15	0	0.00
10–19	643	20.8	57	1.8	12	0.7	14	0.41	4	0.11	0	0.00	3	0.07	2	0.07
20–39	1286	21.5	285	4.7	104	2.6	35	0.61	22	0.36	1	0.02	1	0.02	1	0.02
40–59	712	13.2	115	2.2	61	1.7	11	0.21	6	0.12	2	0.04	4	0.08	1	0.02
60+	479	14.3	43	1.3	15	0.7	2	0.06	0	0.00	37	1.10	3	0.09	1	0.03
Area Health Service																
Urban	3641	24.0	479	3.2	196	1.9	79	0.52	35	0.23	36	0.24	14	0.09	6	0.04
Rural	1509	34.4	65	1.5	19	0.6	1	0.02	1	0.02	5	0.11	4	0.09	1	0.02
Total	**5155**	**26.4**	**546**	**2.8**	**215**	**1.6**	**81**	**0.41**	**36**	**0.18**	**41**	**0.21**	**18**	**0.09**	**7**	**0.04**

Note: Totals for each condition may differ within groupings due to missing demographic values

r = average annual crude rate per 100,000 population

** for 2001 and 2002 only

*** HUS = haemolytic uraemic syndrome; VTEC = verotoxin producing *E. coli* infections.

Source: Communicable Diseases Branch, NSW Department of Health.

TABLE 2

CASES OF COMMUNICABLE ENTERIC DISEASE, HOSPITALISATIONS AND NOTIFICATIONS, NSW, JANUARY 2000 TO JUNE 2002

Condition	Hospitalisations *n*	Notifications *n*	Average annual rate of hospitalisation per 1000 notifications	Median age of patients hospitalised (years)	Median length of stay (days)
Salmonellosis	677	4299	62.99	17	3
Hepatitis A	123	486	101.23	33	3
Shigellosis[a]	59	170	231.37	32	3
Typhoid	56	73	306.85	22	6
Paratyphoid	15	29	206.90	28	5
Listeriosis	22	34	258.82	74	17
HUS *	40	15[b]	1066.67	10	5
VTEC *	2	5	160.0	45	6
Total *	**994**	**5111**	**77.79**	**-**	**-**

[a] Shigellosis only notifiable since 2001 in NSW

[b] the number of HUS notifications are underestimated.

* HUS = haemolytic uraemic syndrome; VTEC = verotoxin producing *E. coli* infections.

Source: Communicable Diseases Branch, NSW Department of Health.

aged care facilities (*n*=92; 48 per cent), childcare facilities (*n*=49; 26 per cent), and hospitals (*n*=34; 18 per cent), followed by schools (*n*=4; two per cent), organised camps (*n*=4; two per cent), military institutions (*n*=1; 0.5 per cent), correctional centres (*n*=1; 0.5 per cent), and institutions not otherwise specified (*n*=6; three per cent).

For the majority of CED outbreaks in institutional settings, person-to-person spread was identified as the most likely means of transmission (*n*=178; 93 per cent). Two (one per cent) of the outbreaks in institutional settings were of suspected foodborne transmission and one outbreak (0.5 per cent) was of suspected waterborne transmission. The mode of transmission was not known in 10 (five per cent) of these institutional outbreaks. In 45 (24 per cent) of the outbreaks, a viral cause was confirmed by laboratory tests of stool samples of those ill (Table 3).

TABLE 3

COMMUNICABLE ENTERIC DISEASE OUTBREAKS IN NSW: JANUARY 2000 TO DECEMBER 2002

Cause	Outbreaks		Cases		Hospitalised	
	n	%	*n*	%	*n*	% of cases
Viral	46	14.9	1140	18.2	58	5.1
Enteric viruses*	45	14.6	1132	18.1	58	5.1
Hepatitis A	1	0.3	8	0.1	0	0
Bacterial	24	7.8	897	14.4	58	6.5
Salmonella	19	6.2	746	11.9	58	7.8
Campylobacter	2	0.6	6	0.1	0	0
Clostridium perfringens	3	1.0	145	2.3	0	0
Protozoan	2	0.6	23	0.4	0	0
Giardia	2	0.6	23	0.4	0	0
Chemical	1	0.3	7	0.1	6	85.7
Ciguatera	1	0.3	7	0.1	6	85.7
Confirmed cause	73	23.7	2067	33.1	122	5.9
Unknown cause	235	76.3	4180	66.9	118	2.8
Total	**308**	**100**	**6247**	**100**	**240**	**3.8**

*Norwalk-like virus, rotavirus, small round-structured virus

Source: Communicable Diseases Branch, NSW Department of Health.

Outbreaks in non-institutional settings

The 111 CED outbreaks that occurred in non-institutional settings included 1,277 individual cases of gastrointestinal illness. The most common settings were restaurants (*n*=54; 49 per cent) and take-away outlets (*n*=25; 23 per cent), followed by private residences (*n*=8; seven per cent), commercial caterers (*n*=7; six per cent), fast food franchises (*n*=6; five per cent), and grocery stores or delicatessens (*n*=6; five per cent). The mode of transmission was confirmed foodborne in 18 (16 per cent), suspected foodborne in 52 (47 per cent), and suspected waterborne in one (one per cent) of these outbreaks. For all others, the mode of transmission was either unknown (*n*=37; 33 per cent) or suspected person-to-person transmission (*n*=3; three per cent).

Community-wide outbreaks

There were six community-wide CED outbreaks during the study period, which accounted for 260 individual cases of gastrointestinal illness. Four of these outbreaks were investigations conducted as a result of a temporal increase in particular *Salmonella* serovars in the community. The agents responsible for these were *S.* Typhimurium PT 9, *S.* Bovismorbificans PT24, *S.* Potsdam, and *S.* Ohio. The mode of transmission was suspected as foodborne for three of the four salmonella outbreaks. Norovirus was identified as the cause for one community-wide outbreak. The cause was not identified for the other community-wide outbreak.

Mode of transmission

Of the 308 CED outbreaks reported during the study period there were 74 (24 per cent) outbreaks in which the mode of transmission was classified as foodborne. The remainder were classified as suspected person-to-person (*n*=183; 59 per cent), suspected waterborne (*n*=2; one per cent), or unknown (*n*=50; 16 per cent).

Foodborne disease outbreaks

Of the 74 FBD outbreaks 24 (32 per cent) were classified as confirmed FBD outbreaks in which the food vehicle was identified and implicated by epidemiological and/or microbiological evidence (Table 4). The remaining 50 (68 per cent) FBD outbreaks were suspected foodborne transmission with varying degrees of evidence. There was no formal study undertaken for 33 (45 per cent) of the FBD outbreaks. A case series investigation was the most common method of investigation (*n*=16; 22 per cent), followed by a cohort study (*n*=15; 20 per cent), and a case control study (*n*=10; 14 per cent).

There were a large proportion of FBD outbreaks in which the cause was unknown (*n*=52; 71 per cent). The most common cause of all FBD outbreaks with a known pathogen was *Salmonella* (*n*=17; 23 per cent), the most common serovar *S.* Typhimurium (*n*=11), and the most common phage type STM 9 (*n*=5). Other aetiological agents include hepatitis A (*n*=1; one per cent), ciguatera poisoning (*n*=1; one per cent), and *Clostridium perfringens* (*n*=3; four per cent).

The most common settings for FBD outbreaks were restaurants (*n*=25; 34 per cent) and takeaway food outlets–franchised fast food outlets (*n*=24; 32 per cent). The most commonly reported food vehicle responsible for FBD outbreaks was poultry (22 per cent). Together, meat and poultry were responsible for 41 per cent of all FBD outbreaks (Table 4).

TABLE 4

IMPLICATED FOOD VEHICLES IN FOODBORNE DISEASE OUTBREAKS, NSW, JANUARY 2000–DECEMBER 2002

Pathogen (number of outbreaks)	Food vehicles implicated and type of evidence *					
	Meat	Poultry	Seafood	Salad or vegetables	Multiple foods	Other–miscellaneous
	n	*n*	*n*	*n*	*n*	*n*
Salmonella spp. (17)	1 L	3 1S,1L,1N	0	1 N	4 1S,1C,2N	8 a
Ciguatera poisoning (1)	0	0	1 L	0	0	0
Clostridium perfringens (3)	3	0	0	0	0	0
Hepatitis A (1)	0	0	0	0	1 N	0
Unknown (52)	10 1S,6C,3N	13 1L,1S,6C,5N	7 1S,1L,2C,3N	0	10 6C,4N	12 b
Total (74)	**14**	**16**	**8**	**1**	**15**	**20**

* L = Laboratory evidence; S = Statistical evidence; C = compelling supportive information; N = no specific evidence.

[a] = deep-fried ice-cream[S,L] (two outbreaks); tahini[L] (one outbreak); caesar dressing[L] (one outbreak); peanuts[L] (one outbreak); cream-filled cake[S] (one outbreak); baked beans–chilli con carne[S] (one outbreak); mango mousse[S] (one outbreak).

[b] = pizza[C,N,S] (six outbreaks); fried rice[N] (two outbreaks); cake[S,N] (two outbreaks); pasta[S] (one outbreak); seafood sauce[N] (one outbreak).

Source: Communicable Diseases Branch, NSW Department of Health.

The major factors identified as contributing to the outbreaks were provided for 35 (47 per cent) of the 74 FBD outbreaks. More than one contributing factor was cited in many outbreaks. Of these 35 FBD outbreaks that identified contributing factors the most commonly cited was 'insufficient cooking' (*n*=16; 46 per cent) and 'inadequate refrigeration–foods left at warm–room temperature' (*n*=16, 46 per cent), followed by 'food handler contamination' (*n*=10; 29 per cent), 'cross contamination from raw ingredients' (*n*=10; 29 per cent), 'toxic substance or part of tissue' (*n*=10; 29 per cent). Other contributing factors identified were 'inadequate hot holding temperature–delay between preparation and consumption–slow cooling' (*n*=8; 23 per cent), 'ingestion of contaminated raw products' (*n*=4; 11 per cent) and 'contaminated equipment–environment–inadequate cleaning of equipment' (*n*=4; 11 per cent).

The level of evidence varied for the factors contributing to contamination from 'assumed or suspected' to 'confirmed with measured evidence'. Of those outbreaks that cited contributing factors, only 18 (51 per cent) were confirmed with evidence, with the highest level of evidence cited as only 'assumed or suspected' in 17 (49 per cent).

DISCUSSION

This study demonstrates that there is a substantial burden of illness associated with CED in NSW. For all CED outbreaks, the most common causes were viruses and the most common settings were institutional, particularly aged care facilities, in which the mode of transmission was mostly person-to-person. For FBD outbreaks with a known pathogen, the most commonly identified cause was *Salmonella*, which is consistent with Australian and international findings.[7–9] The most common setting in which foods were prepared were restaurants and takeaways and the most commonly implicated food vehicle was poultry. Insufficient cooking, inadequate refrigeration, cross contamination from raw ingredients, and food handler contamination, were common factors associated with FBD outbreaks during this period.

There are several limitations to the surveillance and outbreak data. First, surveillance data are likely to substantially under-represent the number of people with CEDs in NSW, as many people with gastroenteritis may not present to a medical practitioner.[9] The proportion of those that do present and then have a stool sample taken is also unknown, but is likely to be small. Second, the outbreak data may be incomplete because many outbreaks, especially if small and self-limited, may not be reported to public health units.[10] Third, the detail provided on outbreaks in this review may be deficient, as many of the outbreak summary forms were incomplete due to the retrospective nature of the survey. Fourth, for the majority of outbreaks, the cause and factors contributing to FBD outbreaks were unknown, and there was a lack of epidemiological and/or microbiological evidence to confirm food vehicles and contributing factors. Finally, regulated health care settings, such as nursing homes and hospitals, may be more likely to report outbreaks than other settings because of the training of staff and their close contact with public health personnel, and because such settings often include long-term residents who are closely observed.

The results of this study are largely consistent with those reported for the whole of Australia during the same period, in particular the age distribution of cases within specific conditions.[7] In NSW, the rates of salmonellosis, shigellosis, listeriosis, and VTEC were lower, and rates of typhoid and HUS were slightly higher, than rates reported for Australia. The majority of typhoid and paratyphoid cases in Australia have acquired their condition overseas.[7] Many of these cases living in Sydney may be born overseas and have acquired the infection on return to their country of birth.[11] The higher rate of hepatitis A and shigellosis among males aged 20–39 years is believed to be largely due to a proportion of cases being men who have sex with men, who are at greater risk of contracting these conditions.[12,13] The noticeably higher salmonellosis rates in rural area health services compared to urban area health services remains unexplained.

The large proportion of institutional outbreaks that were transmitted from person-to-person suggests the need to strengthen infection control strategies in institutions. To help prevent and control outbreaks, it has been recommended that aged-care facilities have infection control guidelines and outbreak management plans in place.[14] These results also indicate food handler contamination is a major contributing factor towards FBD outbreaks, suggesting a need to better educate food handlers on the transmission of FBD and safe food practices.

There was a large amount of missing data. The quality of data obtained on FBD outbreak summary forms would improve if they were completed, by the person responsible for the investigation, within one month of the conclusion of the outbreak. Simplifying the existing data collection form may improve the completeness of data obtained from the public health unit.

Given the cost of CED to the community, and the apparent increasing incidence of FBD, ongoing surveillance and monitoring of FBD in NSW is essential. The information obtained from these outbreak investigations will assist with the identification of the underlying causes of future outbreaks and the development of systems for prevention and control.

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FACT*SHEET*

FOODBORNE DISEASE

WHAT IS FOODBORNE DISEASE?

Foodborne disease (or food poisoning) results from consuming contaminated food or drink. It is very common, with an estimated 5.4 million cases per year in Australia.

Three main types of agents may cause illness from food: bacteria, viruses, and toxins in food (either naturally occurring or introduced to a food).

Food poisoning can occur with any food, whether it is manufactured or prepared at home, at school, at the local supermarket, takeaway outlet, or restaurant. The risk of food poisoning can be reduced if the food is properly stored and prepared.

WHAT ARE THE CAUSES AND SYMPTOMS OF FOODBORNE DISEASE?

Several different diseases with different symptoms can result from eating contaminated food.

Common causes are:

- bacteria, for example *Salmonella*, *Campylobacter* and *Listeria* ;
- viruses, for example Norovirus and hepatitis A;
- toxins, for example toxins made by bacteria such as *Staphylococcus aureus* or *Bacillus cereus*, and ciguatoxin.

Symptoms will vary, depending on the cause. They may include: diarrhoea, vomiting, nausea, abdominal pain, and fever. Other symptoms may include headache, jaundice, and numbness. Symptoms can take between a few hours to a few days, or even longer, to develop and usually last for a few days, sometimes longer.

WHO IS AT RISK?

Anyone can get a foodborne disease. However some people are at increased risk of serious illness. These include:

- infants;
- elderly;
- people with suppressed immune systems;
- pregnant women.

HOW IS IT TREATED?

Many people have mild symptoms and will soon recover. People with diarrhoea and vomiting should stay home from work or school and drink plenty of fluids. People at risk of dehydration such as infants and the elderly should see their local doctor early. Antibiotics are not usually required except in complicated cases.

HOW CAN IT BE PREVENTED?

Hygiene

Wash your hands thoroughly with soap and running water for at least 15 seconds and dry them with a clean towel after using the toilet, changing nappies, and before eating or preparing food. People with symptoms of foodborne disease should not prepare food for others.

Temperature control

Storing food at incorrect temperatures can result in the multiplication of bacteria that cause food-poisoning, which grow between temperatures of 5°C and 60°C. As a precaution:

- refrigerators should not be higher than 5ºC and should have adequate air flow around food to ensure even temperature distribution;
- hot foods should be kept above 60°C;
- reheated foods should be quickly reheated until all parts of the food reach 75°C;
- frozen food should be thawed in either the refrigerator or the microwave. The longer raw food is left at room temperature the more quickly bacteria multiply and toxins may form;
- to kill germs inside food, it must be thoroughly cooked.

Storage

Raw meat, fish, poultry, and raw vegetables can contain large numbers of bacteria, and can cross-contaminate ready-to-eat food if they are not stored or handled carefully. As a precaution:

- raw foods should be stored covered or in sealed containers below other ready-to-eat foods to prevent food parts and meat juice spilling or dripping on to the other food;
- foods should be covered before storage in the refrigerator, freezer, and cupboards to protect them from contamination;
- hands should be washed immediately after handling raw foods and before handling cooked or ready to eat food;
- different chopping boards, utensils, and plates should be used for raw foods and ready-to-eat food. If the same chopping board is being used, it should be washed well in hot soapy water before re-use;
- thoroughly wash raw vegetables before preparation and eating;
- food items should be stored carefully away from toxic chemicals, insect sprays, cleaning agents, etc;
- cloth towels used for drying dishes are not to be used for wiping of hands or bench tops. These should be washed and dried regularly;
- dish cloths should be sanitised regularly or replaced.

If in doubt about the quality or safety of a particular food, the old saying applies, 'If in doubt, throw it out'.

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

January–February 2004

NSW HEALTH Working as a Team

COMMUNICABLE DISEASES REPORT, NSW, FOR OCTOBER AND NOVEMBER 2003

TRENDS

Notifications of communicable diseases through mid-spring indicated a decline in **influenza**, **invasive pneumococcal disease** and **meningococcal disease** (Figure 2, Tables 1–2). Because the surveillance case definition for **pertussis** requires, in part, the patient to have a coughing illness for 14 days, there is an inherent delay between onset of disease and notification of the case. Recent trends in case reports of pertussis are therefore likely to be substantially underestimated, and it is possible that a further rise in case reports will occur in coming months.

A large gastroenteritis outbreak caused by **Norovirus** infection was identified in October, involving over 70 people in the Greater Murray Area Health Service. The most likely cause of this outbreak was contamination of food by a food handler. Norovirus is infectious with low doses of the virus, which can survive on surfaces and in foods for long periods. People who are ill with gastroenteritis should stay home and not prepare food for anyone until 48 hours after their symptoms have completely resolved. A report of this outbreak will be published in a future issue of the *NSW Public Health Bulletin*.

For updated information, visit **www.health.nsw.gov.au** and click on the link to Infectious Diseases.

INFLUENZA SURVEILLANCE 2003

Robin Gilmour, Clayton Chiu, and David Muscatello

Enhanced surveillance for influenza in NSW indicates that the 2003 influenza season peaked in August. Most cases were caused by the influenza A virus, predominantly the A/Fujian/411/2002 strain. Little influenza B infection was reported. Preliminary analysis of emergency department data, supported by anecdotal reports from clinicians, suggests that influenza may have affected more people in 2003 than in previous recent years.

In 2003, several sources of data were included in an enhanced surveillance for influenza, including:

Sentinel general practitioners

Up to 48 general practitioners participated in weekly reporting of influenza-like illness (ILI) activity. ILI activity peaked in mid August (34.7 per 1,000 consultations). In 2002, a similar peak (36.6 per 1000 consultations) appeared in July of that year.

Virological surveillance

Six sentinel laboratories tested 10,391 respiratory samples for the presence of influenza virus, by either direct immunofluorescence (DIF) or culture. Influenza A was found in 831 samples, and this strain peaked in mid-to-late August (23.2 per 100 samples). In 2002, detection of influenza A peaked at 16.9 per 100 samples. Influenza B was found in 13 samples, and this strain peaked in early September (0.5 per 100 samples).

Serological surveillance

The same six sentinel laboratories tested 4,052 serum samples for evidence (seroconversion or rise in IgG level or high single titre) of infection with influenza. Serological diagnoses of influenza A peaked in early September, at 13.6 per 100 samples. In 2002, a similar peak (14.1 per 100 samples) appeared in late August. Serological diagnoses of influenza B were rare and no peak was identified. In 2002, the peak occurred in early July (7.8 per 100 samples).

General practitioner direct virological surveillance

In 2003, fifteen general practitioners (GPs) volunteered to provide specimens from patients who they suspected to have influenza infection for virological testing. Three-hundred-and-nine samples were taken by the GPs, of which 51 (16.5 per cent) were positive for influenza A. No samples tested positive for influenza B.

The WHO Influenza Collaborating Centre

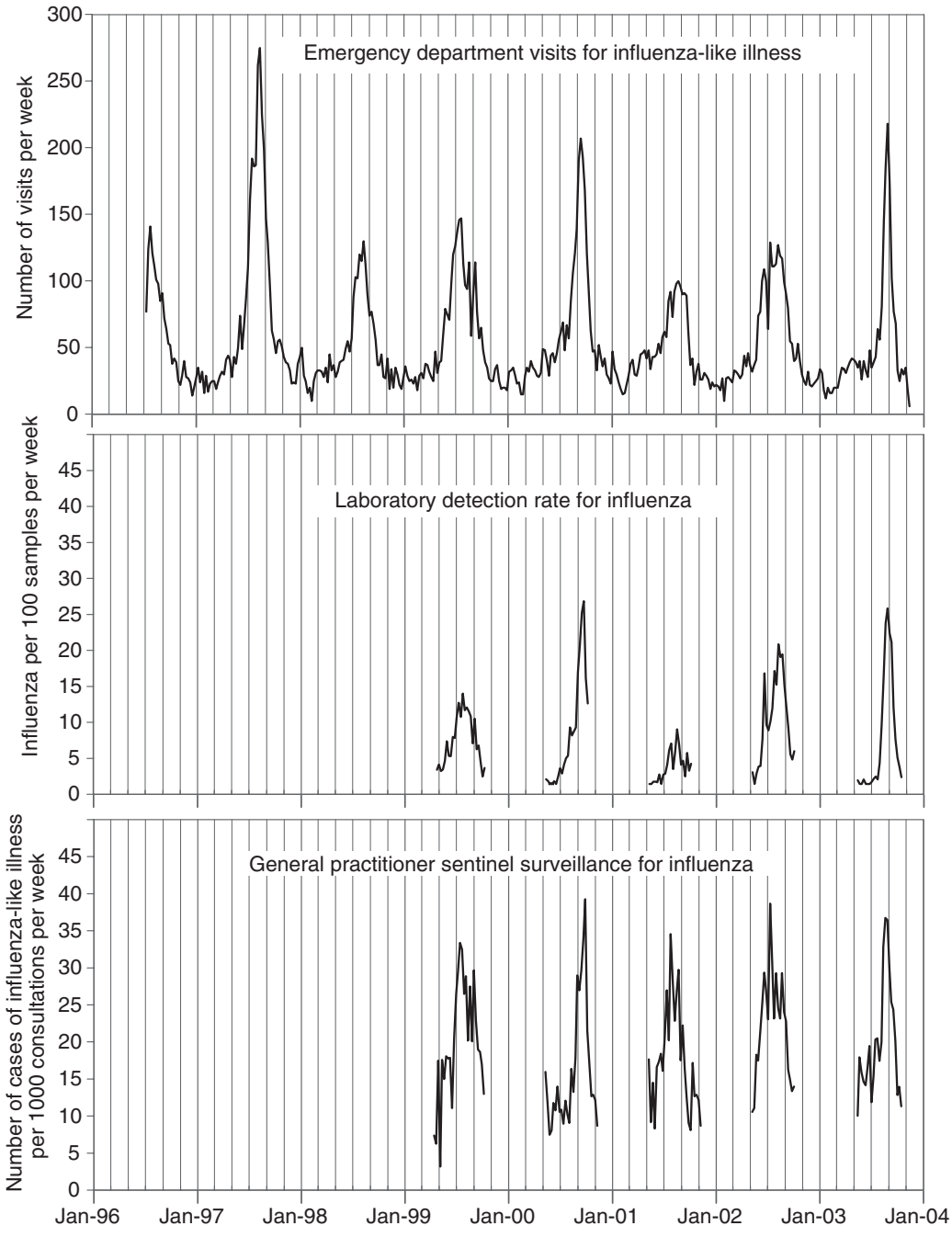
The WHO Influenza Collaborating Centre for Reference and Research on Influenza, located in Melbourne, reports that the majority of influenza A isolates identified during the peak period were A(H3) viruses of the A/Fujian/411/2002 type. This year, some antigenic drift has been detected in the virus strains circulating in Australia and New Zealand. The A/Fujian-like viruses are related to the A/Moscow-like strain included in the 2003 vaccine, and the vaccine has been demonstrated to induce antibodies to the A/Fujian-like strains but generally at a reduced level. In the last few years, dominant strains of influenza A have included A/Nanchang/95, A/Sydney/97, A/Moscow/99, and A/New Caledonia/99 (the last part of the name of each strain represents the year in which it was first identified).

Emergency department surveillance

Information on visits to NSW Emergency Departments (EDs) collected routinely by hospitals is currently being evaluated as a monitoring tool for influenza surveillance. Figure 1 compares the number of ED visits assigned a provisional diagnosis of influenza in hospitals participating in the NSW Emergency Department Data Collection with other influenza indicators currently used in NSW. Only EDs providing reasonably-complete provisional diagnosis information using the International Classification of Diseases for the period July 1996 to October 2003 were included. The collection captures approximately two-thirds of NSW Emergency Department visits. Peaks in the number of visits to EDs assigned a provisional diagnosis of influenza corresponded to peaks in reports from laboratory virology and GP sentinel surveillance. The highest peaks in the years 1997, 2000,

FIGURE 1

COMPARISON OF EMERGENCY DEPARTMENT VISITS FOR INFLUENZA-LIKE ILLNESS WITH LABORATORY-BASED DETECTION RATES AND GENERAL PRACTITIONER SENTINEL SURVEILLANCE FOR INFLUENZA, NSW, 1996–2003



Notes: Influenza-like illness in emergency departments was based on unplanned visits assigned a principal provisional diagnosis of influenza. Laboratory and general practitioner data were only available from May 1999.

Source: NSW Emergency Department Data Collection (HOIST), Centre for Epidemiology and Research, NSW Department of Health; and NSW Influenza Surveillance Program, Communicable Diseases Branch, NSW Department of Health.

and 2003 coincided with a predominance of newly emergent strains of influenza A virus among laboratory samples that had strain identification performed (Sydney/97, Moscow/99 and Fujian/2002 in each of those peak years respectively).

COMMENT

The data and information collected from these sources indicate that influenza peaks each year in winter, usually between mid-July and mid-September. In 2003, the peak was in August, and influenza activity may have been more widespread than in recent previous years.

There are several limitations to these data. First, none of the surveillance systems mentioned here are very sensitive: all collect data on only a very small proportion of people infected with influenza in NSW, and this proportion may vary over time, rendering comparisons open to bias. Second, none of the systems provide a very representative sample of influenza cases either by the demographics of the affected people, their place of residence, or severity of illness. Laboratory surveillance is based in urban hospitals, and is be more likely to include very sick children (who tend to present to hospital for testing) than the GP systems. Participating GPs are not located randomly across the state. Third, apart from the laboratory-based systems, the diagnosis of influenza-like illness is not specific, and the systems are likely to pick up a range of other respiratory conditions not caused by influenza viruses.

The apparent triennial variation in the magnitude of the influenza peaks found in Emergency Department visits appears to be temporally associated with the predominance of a newly emergent A strain among circulating strains of the influenza virus for the year. These data suggest that ED surveillance could be a useful tool for monitoring not only the occurrence of influenza epidemics in NSW but also their extent. The Centre for Epidemiology and Research has developed methods for the rapid transfer and analysis of these data for surveillance purposes.

FIGURE 2

REPORTS OF SELECTED COMMUNICABLE DISEASES, NSW, JANUARY 1996 TO NOVEMBER 2003, BY MONTH OF ONSET

These are preliminary data: case counts for recent months may increase because of reporting delays. Laboratory-confirmed cases, except for measles, meningococcal disease and pertussis.

NSW population	
Male	50%
<5	7%
5–24	28%
25–64	52%
65+	13%
Rural*	42%

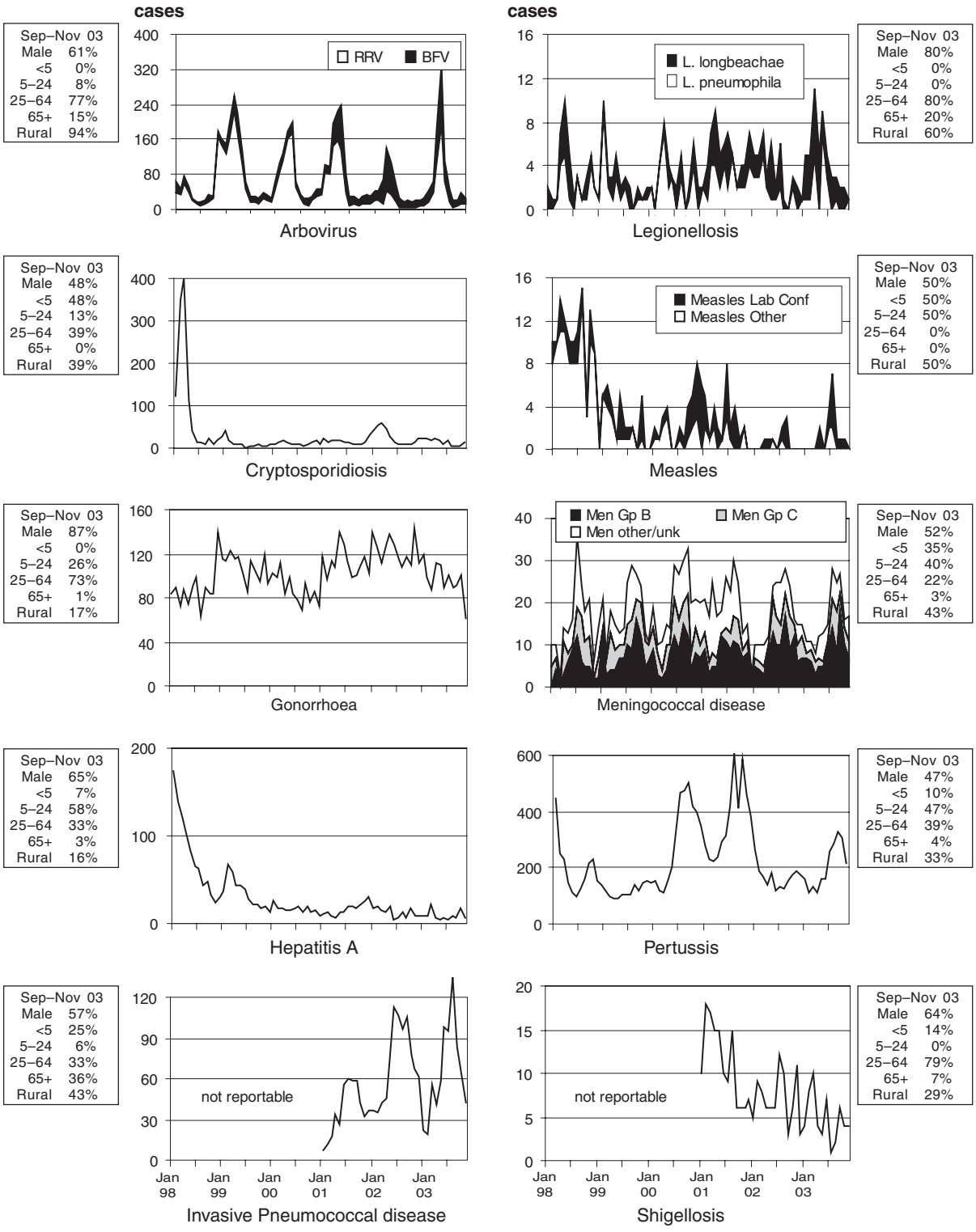


TABLE 1 REPORTS OF NOTIFIABLE CONDITIONS RECEIVED IN OCTOBER 2003 BY AREA HEALTH SERVICES

Condition	Area Health Service																		Total	
	CSA	NSA	WSA	WEN	SWS	CCA	HUN	ILL	SES	NRA	MNC	NEA	MAC	MWA	FWA	GMA	SA	CHS	for Oct†	To date†
Blood-borne and sexually transmitted																				
Chancroid*	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-
Chlamydia (genital)*	82	76	56	21	33	14	55	18	123	31	23	31	8	15	9	22	17	4	642	6,140
Gonorrhoea*	21	10	2	3	5	1	6	-	41	6	4	1	1	-	-	1	-	-	102	1,089
Hepatitis B - acute viral*	-	1	1	-	1	-	1	-	2	-	-	-	-	-	1	-	-	-	7	60
Hepatitis B - other*	36	26	31	6	35	3	5	6	39	1	3	2	-	-	3	-	-	2	198	2,795
Hepatitis C - acute viral*	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	96
Hepatitis C - other*	46	37	9	17	57	22	29	29	47	13	19	11	6	20	8	19	16	31	441	5,749
Hepatitis D - unspecified*	-	-	-	-	1	-	-	1	-	-	-	-	-	-	-	-	-	-	2	9
Syphilis	10	3	7	2	8	3	-	2	29	4	1	2	3	-	-	1	2	-	78	713
Vector-borne																				
Barmah Forest virus*	-	-	-	-	-	-	-	-	-	9	6	1	-	-	-	-	-	-	16	409
Ross River virus*	-	-	-	-	-	-	-	-	-	4	1	-	1	-	-	2	3	-	11	462
Arboviral infection (Other)*	-	2	-	-	1	-	1	-	-	-	-	-	-	-	-	-	-	-	2	71
Malaria*	-	2	2	-	-	-	-	-	1	-	-	-	-	-	-	-	-	-	5	104
Zoonoses																				
Anthrax*	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-
Brucellosis*	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	2
Leptospirosis*	-	-	-	-	-	-	2	-	-	-	-	2	-	-	-	-	-	-	4	39
Lyssavirus*	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-
Psittacosis*	-	1	-	1	-	-	2	-	-	-	-	3	-	-	-	1	1	-	9	67
Q fever*	1	-	-	-	-	-	4	-	-	-	2	5	3	-	2	-	1	-	18	264
Respiratory and other																				
Blood lead level*	-	5	-	-	2	-	4	-	1	-	1	-	-	-	7	-	1	-	21	388
Influenza*	2	7	19	5	9	2	1	2	7	2	3	4	-	-	-	-	-	-	63	890
Invasive pneumococcal infection*	6	6	6	1	3	12	13	4	8	2	2	-	1	3	-	2	-	-	69	668
Legionella longbeachae infection*	-	-	-	-	-	1	-	-	-	-	-	-	-	-	-	-	-	-	1	31
Legionella pneumophila infection*	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	19
Legionnaires- disease (Other)*	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-
Leprosy	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	1
Meningococcal infection (invasive)*	1	1	1	-	3	1	1	3	1	-	-	1	-	-	-	3	1	-	18	167
Tuberculosis	4	-	6	2	1	-	-	-	6	1	1	-	-	-	1	-	-	-	23	293
Vaccine-preventable																				
Adverse event after immunisation	-	-	2	-	3	-	3	-	-	-	-	-	2	-	-	5	-	-	15	181
H. *Influenzae b* infection (invasive)*	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	5
Measles	1	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	1	16
Mumps*	2	-	-	-	1	-	-	-	1	-	-	-	-	-	-	-	-	-	4	27
Pertussis	32	45	50	9	40	6	16	9	31	2	6	2	1	7	-	20	9	-	285	1,926
Rubella*	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	22
Tetanus	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	1
Enteric																				
Botulism	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-
Cholera*	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-
Cryptosporidiosis*	-	-	1	-	-	-	-	-	2	-	-	-	1	-	-	1	-	-	5	148
Giardiasis*	-	12	12	6	7	1	7	2	6	-	3	3	2	9	-	1	-	-	71	836
Haemolytic uraemic syndrome	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	3
Hepatitis A*	3	1	2	-	7	-	-	-	1	-	-	-	-	-	-	-	-	-	14	86
Hepatitis E*	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	1	6
Listeriosis*	-	1	-	-	1	-	-	-	-	-	-	-	-	-	-	-	-	-	2	24
Salmonellosis *	8	12	10	5	12	8	5	7	8	12	4	5	-	7	-	1	2	-	106	1,596
Shigellosis*	-	2	-	-	1	-	-	-	-	1	-	-	-	1	-	-	-	-	5	49
Typhoid and paratyphoid*	-	-	-	-	2	-	-	-	2	-	-	-	-	-	-	-	-	-	5	25
Verotoxin producing *E. coli**	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-

* lab-confirmed cases only + includes cases with unknown postcode ** HIV and AIDS data are reported separately in the *NSW Public Health Bulletin* each quarter

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SA = Southern Area
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TABLE 2 REPORTS OF NOTIFIABLE CONDITIONS RECEIVED IN NOVEMBER 2003 BY AREA HEALTH SERVICES

Condition	Area Health Service																		Total	
	CSA	NSA	WSA	WEN	SWS	CCA	HUN	ILL	SES	NRA	MNC	NEA	MAC	MWA	FWA	GMA	SA	CHS	for Nov†	To date†
Blood-borne and sexually transmitted																				
Chancroid*	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-
Chlamydia (genital)*	71	73	79	17	42	-	63	33	138	26	34	24	11	15	1	17	15	2	664	6,816
Gonorrhoea*	20	1	4	1	3	-	3	-	33	4	2	1	-	-	-	2	-	-	77	1,167
Hepatitis B - acute viral*	-	1	-	-	-	-	1	-	-	-	-	-	-	-	-	-	-	-	2	65
Hepatitis B - other*	42	36	-	4	53	-	7	1	37	3	2	7	3	1	-	2	1	5	207	3,018
Hepatitis C - acute viral*	-	-	1	-	-	-	4	-	-	-	-	-	-	-	-	-	-	-	5	112
Hepatitis C - other*	72	32	14	20	83	-	34	27	79	32	28	9	8	11	-	26	13	37	527	6,370
Hepatitis D - unspecified*	-	-	-	-	1	-	-	-	-	-	-	-	-	-	-	-	-	1	2	12
Syphilis	28	2	7	1	20	-	-	-	36	6	-	3	-	-	-	-	-	1	105	818
Vector-borne																				
Barmah Forest virus*	-	-	-	-	-	-	1	-	-	10	6	-	-	1	-	-	-	-	18	428
Ross River virus*	-	-	-	-	-	-	2	-	-	5	3	-	1	-	-	-	1	-	12	474
Arboviral infection (Other)*	-	-	-	-	1	-	-	-	-	-	-	-	-	1	-	-	-	-	2	75
Malaria*	1	-	2	1	1	-	1	-	2	2	-	1	2	-	-	1	-	-	14	118
Zoonoses																				
Anthrax*	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-
Brucellosis*	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	2
Leptospirosis*	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	39
Lyssavirus*	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-
Psittacosis*	-	-	-	-	-	-	5	-	-	1	1	2	-	-	-	-	-	-	9	76
Q fever*	-	-	-	-	-	-	-	1	-	1	-	8	7	-	-	-	-	-	17	281
Respiratory and other																				
Blood lead level*	1	-	-	-	4	-	2	1	-	-	2	1	-	-	-	1	-	-	12	405
Influenza*	1	1	7	-	5	-	-	-	8	2	-	1	-	-	-	1	-	-	26	881
Invasive pneumococcal infection*	1	8	5	5	13	-	4	4	10	1	8	-	2	1	-	-	4	-	67	738
Legionella longbeachae infection*	-	-	-	1	-	-	-	-	-	-	-	-	-	-	-	-	-	-	1	32
Legionella pneumophila infection*	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	19
Legionnaires- disease (Other)*	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-
Leprosy	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	1
Meningococcal infection (invasive)*	-	1	1	1	2	-	-	1	5	1	-	-	-	1	-	-	-	-	13	180
Tuberculosis	4	4	7	-	4	-	1	2	9	-	-	-	1	-	-	-	-	-	33	343
Vaccine-preventable																				
Adverse event after immunisation	-	-	-	-	1	-	3	-	2	-	-	-	1	2	-	-	-	-	9	192
H. *Influenzae b* infection (invasive)*	-	-	-	1	-	-	-	-	-	-	-	-	-	-	-	-	-	-	1	6
Measles	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	16
Mumps*	-	2	-	1	-	-	-	-	1	-	-	-	-	-	-	-	-	-	4	31
Pertussis	28	50	60	11	51	-	19	24	70	7	9	5	3	4	-	31	26	-	398	2,325
Rubella*	-	-	-	-	-	-	-	-	1	-	-	-	-	-	-	-	-	-	1	23
Tetanus	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	1
Enteric																				
Botulism	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-
Cholera*	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-
Cryptosporidiosis*	-	-	2	-	2	-	2	-	3	-	-	2	-	1	-	-	-	-	12	160
Giardiasis*	4	14	11	5	4	-	5	2	19	-	2	3	4	3	-	1	2	-	79	925
Haemolytic uraemic syndrome	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	3
Hepatitis A*	3	-	3	-	5	-	-	-	-	2	-	-	-	1	-	-	-	-	14	100
Hepatitis E*	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	6
Listeriosis*	-	1	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	1	25
Salmonellosis (not otherwise specified)*	8	15	14	4	9	-	12	5	16	11	1	1	1	3	-	1	1	-	105	1,705
Shigellosis*	-	1	1	-	1	-	-	-	3	-	-	-	-	-	-	-	1	-	7	56
Typhoid and paratyphoid*	-	1	-	-	-	-	-	-	2	-	-	-	-	-	-	-	-	-	3	29
Verotoxin producing *E. coli**	-	-	-	-	-	-	1	-	-	-	-	-	-	-	-	-	-	-	1	1

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