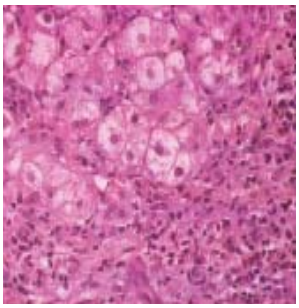
Australian Government
Department of Health and Ageing

Communicable Diseases Intelligence



Quarterly report

Volume 28
Issue no 3
2004

Communicable Diseases Intelligence

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Volume 28

Issue no 3

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ISBN 0725-3141

Communicable Diseases Intelligence aims to diseminate information on the epidemiology and control of communicable diseases in Australia. *Communicable Diseases Intelligence* invites contributions dealing with any aspect of communicable disease epidemiology, surveillance or prevention and control in Australia. Submissions can be in the form of original articles, short reports, surveillance summaries, reviews or correspondence. Instructions for authors can be found in *Commun Dis Intell* 2004;28:95–97.

Communicable Diseases Intelligence contributes to the work of the Communicable DIseases Network Australia (www.cda.gov.au/cdna/)

Website

http://www.cda.gov.au/index.htm

Subscriptions and contacts

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

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Front cover: Surveillance and Epidemiology Section, Australian Government Department of Health and Ageing.

Clockwise from top left: Professor Margaret Burgess AO, image reproduced with permission from NCIRS; horde of bats which could contain possible carriers of the rabies virus, sourced from the Centers for Disease Control and Prevention Public Health Image Library, courtesy of the Centers for Disease Control and Prevention, Atlanta, Georgia; acute hepatitis, image © Bristol Biomedical Image Archive, University of Bristol, donated by Professor Ian Lauder; aviary of budgerigars; resident in an aged care facility being immunised with influenza vaccine; tropical fish aquarium.

Printed by Union Offset, Canberra

Contents

Evaluation of Australia's National Notifiable Disease Surveillance System

Megge Miller,[1,2] Paul Roche,[2] Jenean Spencer,[2] Mary Deeble[1]

Abstract

The Australian National Notifiable Diseases Surveillance System (NNDSS) is a passive surveillance system that collects information on communicable diseases. The Australian Government manages NNDSS under the auspices of the Communicable Diseases Network Australia (CDNA). Data collected by each state and territory are collated, analysed and disseminated by the Australian Government Department of Health and Ageing. We report the first evaluation of NNDSS since it was established in 1991. Three primary stakeholder groups were surveyed: (a) CDNA members, (b) the National Surveillance Committee and (c) the readership of *Communicable Diseases Intelligence,* the primary means of data dissemination from NNDSS. The evaluation revealed that the system was acceptable, structurally simple, and that the data collected were actively used by stakeholders. However, the lack of clearly documented aims and objectives for NNDSS, inflexibility to changing needs, lack of timeliness and complexity in processes were seen as problematic. The results of this evaluation, supported by recent federal funding to enhance national biosecurity, will provide the framework for enhancing NNDSS to meet national communicable disease surveillance requirements in Australia. *Commun Dis Intell* 2004;28:311–323.

Introduction

Evaluation is an important part of communicable disease surveillance. Systematic and objective evaluation of surveillance determines the relevance, effectiveness and impact of such systems.

History of national surveillance in Australia

The occurrence of disease and death in Australia has been recorded since settlement in 1788. Each colony recorded information on an ad hoc basis[1] on the main diseases affecting the population. The *Quarantine Act,* 1832 of New South Wales was the first legislation relating to public health and was the first to introduce mandatory notification of diseases to local health authorities in Australia. Over time, the actions of New South Wales prompted other colonies to establish their own legislation for communicable disease control and reporting.[1] In 1901, the colonies of Australia joined together to form a federation, which lead to the creation of the Commonwealth Government. The new Commonwealth Constitution protected the powers and the interests of the states in relation to public health. However, the Commonwealth was given powers of quarantine for specified communicable diseases under the *Quarantine Act*, 1908. The *Quarantine Act* remains the sole legislative authority the Commonwealth has in relation to communicable diseases, to this day.

From 1917 to 1922, national data on notifiable diseases provided by the states and territories were published in the *Medical Journal of Australia*. From 1924 onwards, the Commonwealth Department of Health has published aggregated national data in various government publications.[2] In the mid 1980s, as the AIDS epidemic unfolded, the need for national surveillance was highlighted. The Communicable Diseases Network Australia New Zealand (CDNANZ) was formed in 1987, to enhance national surveillance and communicable disease collaborations. CDNANZ later became the Communicable Diseases Network Australia (CDNA), reporting to the National Public Health Partnership whose members are state and territory Chief Health Officers.

In 1988, a National Health and Medical Research Council (NHMRC) Workshop on National Disease Surveillance recommended that a Working Party be formed to establish a nationally consistent cooperative approach to surveillance.[3] The main

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issues considered by the Working Party were the list of communicable diseases to be nationally notifiable, the construction of an inventory of existing communicable disease surveillance activities in Australia, a uniform approach to a national surveillance network and uniform basic data requirements for a surveillance database.

By 1991, the National Notifiable Diseases Surveillance System (NNDSS) was established. Data on the agreed list of nationally notifiable diseases were sent via diskette or paper from the states and territories to the Commonwealth. Officers in the Commonwealth health department, now the Australian Government Department of Health and Ageing (DoHA), would collate the data and publish surveillance summaries in the fortnightly publication *Communicable Diseases Intelligence (CDI)*. In 1996, the National Communicable Diseases Surveillance Strategy was released on behalf of the Chief Health Officers of Australia. The Strategy aimed to improve communicable disease surveillance and to provide comprehensive epidemiological data on which to base risk management decisions and public health policy. The Strategy recommended that NNDSS be improved by review of data quality, timely reporting, regular review of the diseases to be notified and case definitions, and expansion of the minimum dataset for specific conditions.[4] The recommendation was adopted by both the Commonwealth department of health and CDNA.

The NNDSS database has been undergoing redevelopment since 2000. A new information technology platform has been created to automate the transmission of notification data from jurisdictions to the Commonwealth, new data fields were added to the minimum dataset and the case definitions have been under review since 2001. Throughout the development of NNDSS, the overall system has never been formally evaluated.

Aims of the evaluation

The aims of this evaluation are to systematically and objectively evaluate the attributes of NNDSS and highlight areas for improvement.

Methods

The framework detailed in the *Updated Guidelines for Evaluating Public Health Surveillance Systems*[5] was used for this evaluation, because of the comprehensive nature of these guidelines. The evaluation focussed on the national surveillance system as a whole, which included stakeholder networks as well as the database that houses notification data, the analysis and interpretation of the collated data and the feedback mechanisms to stakeholders. It was beyond the scope of this evaluation to examine enhanced surveillance for selected diseases using NNDSS. The evaluation includes a description of NNDSS and the public health importance of the events under surveillance.

The assessment of quantitative system attributes (timeliness, data quality and representativeness) was conducted by an analysis of NNDSS data including an analysis of data completeness of all 25 data fields. The sensitivity and positive predictive value (PPV) of NNDSS was beyond the scope of this evaluation. Qualitative system attributes (simplicity, flexibility and acceptability) were assessed through survey-based consultation with stakeholders. Usefulness of and accessibility to NNDSS data were also assessed using surveys. The three main stakeholder groups and the methods of consultation are described below.

Communicable Diseases Intelligence readership

A questionnaire was sent to all persons and organisations on the subscription list for *Communicable Diseases Intelligence*. The questionnaire was one page long and asked participants what their profession was, whether they use NNDSS, what they used it for, how they access the data and how easy it was to access the data. The self-administered questionnaire was distributed with a subscription renewal form for *CDI*. A postage paid envelope was included with each questionnaire.

Communicable Diseases Network Australia membership

A questionnaire was sent to selected members of the Communicable Diseases Network Australia. The survey contained quantitative and qualitative components. Participants were asked whether they use NNDSS, what they used it for, what they think the objectives of national surveillance should be and their opinions about the strengths and weaknesses of NNDSS. The members of CDNA surveyed were the Chair, the Jurisdiction Executive Group and representatives from OzFoodNet, the National Centre for Immunisation Research and Surveillance of Vaccine Preventable Diseases (NCIRS) and the National Centre for HIV Epidemiology and Clinical Research (NCHECR). Each member was interviewed by telephone. Transcripts of the interviews were analysed for key themes. A qualitative approach was chosen for this group of stakeholders to obtain a richness in detail of participants' perspectives and opinions that quantitative methods alone could not achieve.

National Surveillance Committee membership

The National Surveillance Committee (NSC) is a sub-committee of CDNA and consists of jurisdictional epidemiologists and data managers and epidemiologists from the national centres (NCIRS, NCHECR and OzFoodNet). These people have frequent interactions with NNDSS as data providers and users. Two surveys were used in the stakeholder consultation of NSC: (a) a survey for the epidemiologists (n=11) and (b) a survey for the data managers (n=8). The surveys were sent via email and respondents were asked to complete the survey electronically. Descriptive analyses were performed on the quantitative results from the NSC survey and the qualitative components were analysed for key themes.

Results

The *CDI* questionnaire was sent to 2,167 subscribers and responses were received from 537 participants. Of the subscribers to whom *CDI* is sent, 901 are institutions (i.e. *CDI* is addressed to the institution rather than an individual) and we did not expect to receive a response from them. Overseas subscribers (n=262) were not surveyed. Therefore, the total number of people from whom we could expect a response was 1,004. The response rate for the *CDI* questionnaire was 53 per cent (537/1,004).

All of the CDNA members (12/12) participated in the survey, however, four (30%) members reported that they had not prepared because they were too busy. One member did not have time for a telephone interview and sent a completed handwritten survey. Over three-quarters of the NSC members (78%, 14/18) responded to the electronic questionnaire sent to them.

Objectives and utility

Objectives of the National Notifiable Diseases Surveillance System

The objectives of NNDSS were not clearly stated in the corporate records held by the DoHA. By deduction from current system functioning, the purpose of NNDSS is to monitor trends in disease incidence nationally to help understand the epidemiology of communicable diseases Australia-wide.

When NNDSS was being established, the Working Party on National Disease Surveillance drafted a list of objectives that a national communicable disease surveillance system should have:[6] These were:

- control communicable diseases;
- alert state or territory health authorities to communicable disease episodes which require public health action across jurisdictional borders;
- coordinate national responses to disease threats; and
- act as a clearing house for the dissemination of information.

There were some differences of opinion amongst CDNA members about the objectives of a national surveillance system (Table 1). Most members thought the main objective would be to examine national trends in diseases. There were strong differences of opinion with regards to the role of NNDSS in outbreak detection, where seven members (58%) thought that national surveillance data should be used to detect outbreaks and four members (33%) thought national surveillance systems should not be used to detect outbreaks. There was recognition amongst some in this group (n=3) that NNDSS was not primarily designed to detect but to document outbreaks:

Table 1. Responses from Communicable Diseases Network Australia members on the objectives of a national surveillance system

Objective	Number who listed objective (n=12)
Examine national trends	10
Outbreak detection	7
Evaluation of interventions	7
Research	3
Meet international reporting obligations	2

'National surveillance can provide a global picture of an outbreak, but not actually identify outbreaks—that's done at a public health unit level'

Some of the other objectives for NNDSS proposed by CDNA members include economic evaluation of disease control programs such as immunisation, informing the production of disease control guidelines and providing information to inform government spending.

Uses of National Notifiable Diseases Surveillance System data

The stakeholder surveys were the main sources of data used to judge the usefulness of NNDSS. Both CDNA members and NSC members mainly used NNDSS to examine national trends and to examine trends across each state and territory (Table 2). The jurisdictional members of CDNA reported that when examining trends in other states and territories, they usually compare the rate of disease in their jurisdiction with the national rate. Within the CDNA membership, there was an obvious spectrum of enthusiasm for using NNDSS data, where one member reported:

'I feel ashamed to say it, but I don't actually use it [NNDSS]'

Another member reported:

'Yes, I use it [NNDSS] a lot…but we could do more with it, we could do a lot more with it'

Half of the CDNA members reported using NNDSS for general interest. This is an indirect indicator of engagement within the system. For example, 'general interest' describes the use of the system outside of work requirements.

Of the 537 people who responded to the *CDI* readership survey, 502 (94%) reported reading the quarterly reports of the surveillance highlights from NNDSS in *CDI*. Similarly, 502 (94%) reported reading the annual report of NNDSS. Eight-five per cent (n=454) of *CDI* readers reported using NNDSS data. The main use of NNDSS data to the *CDI* readership was general interest, followed by research (Table 3).

Of the 83 *CDI* readers who said they do not use NNDSS, nine (11%) reported they did not use NNDSS because it was too difficult to access, 22 (27%) were not aware that NNDSS data were available and 40 (48%) reported that NNDSS data were not relevant to them. The remaining 12 respondents who did not use NNDSS did not provide a reason.

In terms of guiding public health action, NNDSS data have been used mainly in the area of vaccine preventable diseases. For example, one of the CDNA members reported that the Meningococcal Vaccine Working Party and the Meningococcal Control Working Party used NNDSS data to inform policy advice for the recent introduction of the meningococcal C vaccine to the national immunisation schedule. The National Sexually Transmissible Infection Committee, the National Arbovirus and Malaria Advisory Committee, the National Tuberculosis Advisory Committee and other national expert committees also use data from NNDSS to inform policy and program decisions.

Table 2. Reported uses of National Notifiable Diseases Surveillance System by members of Communicable Diseases Network Australia and the National Surveillance Committee

Uses of NNDSS	CDNA	NSC
Monitor national trends	11	11
Examine trends in other states and territories	10	10
Research purposes	7	8
Policy development	7	7
Inform program management	6	6
General interest*	6	6
Outbreak detection	4	4
Other†	2	3

* General interest is defined as using NNDSS outside of work requirements

† Other includes teaching purposes, media and evaluation of a public health intervention

Table 3. Uses of National Notifiable Diseases Surveillance System reported by *Communicable Diseases Intelligence* readership

Use of NNDSS data	Number who use data	Percentage of all respondents
General interest	317	59
Research	180	34
Inform policy	150	28
Outbreak detection	142	26
Inform program management	125	23
Training and education	55	10

Data from NNDSS are routinely sent to the National Centre for HIV Epidemiology and Clinical Research and the National Centre for Immunisations Research and Surveillance on a monthly basis. National data are also sent to OzFoodNet, but they often contact jurisdictions for additional data on foodborne diseases. Surveillance data on zoonotic diseases are sent to the Australian Government Department of Agriculture, Fisheries and Forestry every quarter.

Monthly data for 21 diseases are also sent to the Regional Electronic Surveillance System for Notifiable Diseases in the Western Pacific Region, maintained by the World Health Organization (WHO). NNDSS is used to help Australia meet its international reporting obligations. For example, each year WHO asks the Commonwealth to send data on tuberculosis notifications, treatment and control in Australia. This information is collated with data from the rest of the world and published in the annual *Global Tuberculosis Control: Surveillance, Planning and Financing* report.[7] WHO also requests national data on leprosy, vaccine preventable diseases (e.g. polio) and quarantinable diseases (e.g. cholera, plague).

Health events under surveillance

In 2003, there were 57 communicable diseases under surveillance at a national level. AIDS and HIV data are collected, analysed, interpreted and disseminated by NCHECR.

The criteria used to determine whether a disease should be nationally notifiable are:[8]

- *Feasibility of collection*: The collection of data for the disease must be relatively simple to collect.
- *Priority*: There are diseases that are important at state and territory level, but the disease must have a demonstrated priority at a national level (i.e. disease affects most or all jurisdictions).
- *Immediacy of an intervention:* The disease requires an immediate response to prevent transmission through the community.
- *Outbreak potential of the disease*: The disease is prone to outbreaks that have a substantial burden on the community.
- *Potential for disease control programs*: The disease should be preventable through the implementation of control programs.
- *High-case fatality rate*: There is a high proportion of deaths from this disease relative to the number of cases of the disease.
- *Community or political concerns*: Some diseases may be of high concern to the community or the occurrence of the disease may have political implications.
- *International concern*: Diseases spread across international boundaries and it is important to recognise diseases that are a concern in the region.
- *Evaluation of programs*: Surveillance data can be used as a tool to evaluate existing and future communicable disease control programs.
- *Importance to Indigenous health*: Diseases that have an impact on the Indigenous communities throughout Australia should be under surveillance.

The list of nationally notifiable diseases has changed several times over the past 15 years. In 2003, the list of nationally notifiable diseases was changed to include severe acute respiratory syndrome (SARS), smallpox, tularaemia, syphilis (<2 years duration) and syphilis (>2 years duration or unknown). The general consensus from CDNA members was that the current list of nationally notifiable diseases was acceptable.

The case definitions for the nationally notifiable diseases were recently reviewed (2001 to 2003) by CDNA and are being phased in during 2004. The previous 1994 case definitions[9] were not used uni-

formly by jurisdictions as there were some deviations because jurisdictions occasionally used their own case definitions. The recent consensus review of case definitions will lead to consistent reporting throughout Australia by early 2005.

System operation

Legislation

There is no legislative requirement for states and territories to send notifiable disease data to the Commonwealth and hence, NNDSS depends on the commitment and cooperation of CDNA. There are eight separate public health Acts in Australia and each jurisdiction has its own list of notifiable diseases and reporting channels for notification written into the Acts or the Regulations under the Acts. CDNA members have different opinions about the legislative status of NNDSS. Some members do not think the variation of public health Acts is a problem and others did not even mention legislation in the interview. However, other members consider it to be a significant obstacle, with members saying:

> *'We try to cobble together a national communicable disease system out of what is essentially eight colonies. We do very well considering we all have different public health acts'.*

> *'[One of the main] weaknesses of NNDSS is its limited formal standing in Australia's legislative framework for public health'*

Data sources

Data from jurisdictions are sent to the NNDSS database in a de-identified format. The jurisdictions receive their notifications from clinical sources (e.g. general practitioners and hospitals) and from laboratories (both public and privately funded) via paper, telephone and fax. Results from the NSC survey indicate that the majority of notifications in all jurisdictions come from laboratories. Clinical notifications are sometimes received for vaccine preventable diseases, vectorborne disease and invasive meningococcal disease. Population data for the calculation of crude notification rates comes from the Australian Bureau of Statistics.

Information collected

The system collects 25 data fields for each notification of the diseases under surveillance, where relevant (Box). To protect the confidentiality of people with notifiable diseases, the NNDSS database does not receive or record any identifying personal information. A unique notification ID is provided that can be used to trace a report back to a jurisdictional health department if more information is required.

Box. Data fields reported to National Notifiable Diseases Surveillance System

Jurisdiction–source of report

Notification ID–unique identifier

Disease code–code representing a communicable disease

Organism code–code that identifies a specific organism and serogroup/subtype where applicable

Organism name–a full text scientific name of the causative organism for the specified disease

Serogroup/subtype–a full test name of the causative serogroup/subtype for the specified disease

Confirmation status–whether a case is confirmed or probable according to the case definition

Laboratory diagnosis method–one or more diagnostic methods used

Vaccination status–vaccination status of the individual with the disease

Vaccination validation–how was the vaccination information validated?

Vaccine doses–the number of doses of relevant vaccine received by the individual at the time of disease onset

Resident postcode–permanent residential postcode of the individual

True onset date–the earliest estimated date of disease onset

Specimen date–date when the first laboratory specimen was taken or when it was logged into their computer system

Notification date–date when the health professional signed the notification form or the laboratory issued the results

Notification received date–date when the notification was received by the communicable diseases section of the state or territory health authority

Date of birth–the date of birth of the individual

Age at onset–the age of the individual at the date of onset

Sex–the current sex of the individual

Indigenous status–a single character field indicating the Indigenous status of the individual (Aboriginal, Torres Strait Islander, Aboriginal and Torres Strait Islander or non-Indigenous).

Died–did the patient die from the notifiable condition?

Outbreak reference–a reference ID for a known disease outbreak that is or has been under investigation

Case found by–how was the case identified?

Imported from overseas–whether the disease was believed to been acquired in another country

The compulsory fields are jurisdiction, notification ID, disease code, resident postcode and notification received date. All other data fields are completed when the information is available.

The issue of how to record notifications of disease in persons not resident in the state or territory of disease acquisition is not clearly defined. The current provision in the data specifications is to record the postcode of residence of the case. However, this does not indicate the likely location of exposure leading to infection for diseases such as Ross River virus infection where people may visit endemic areas for recreational activities, become infected and then return home. This means that any spatial analysis of disease incidence may be misleading at the national level. The issue needs to be resolved so that location and source of infection can be appropriately assigned.

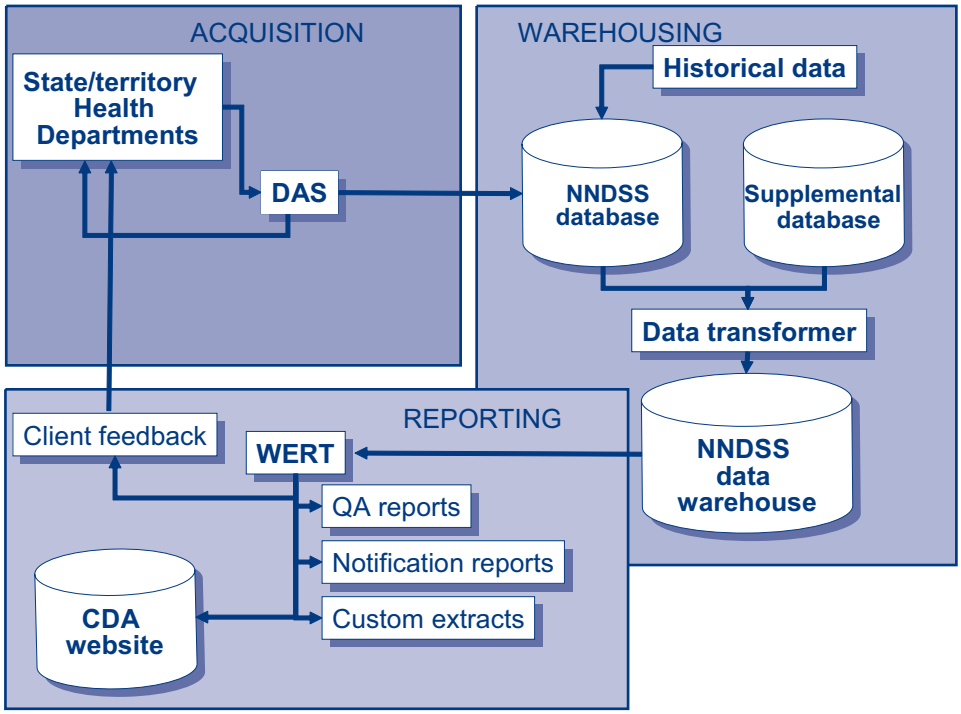
Transfer and management of information

The NNDSS database has been under going an information technology redevelopment since 2000. The main aspect of redevelopment has been improving the national collation of data from jurisdictions to the Commonwealth and the subsequent 'warehousing' of data. In the past, data from the jurisdictions were sent in paper form, on diskette or electronically and then entered into a Microsoft Access database. Since 2002, the jurisdictions and the DoHA have been working closely to implement the Data Acquisition System (DAS). An export facility was installed at the jurisdiction end and the DoHA end uses DAS to receive the data in a format that can be automatically loaded into the Microsoft Access NNDSS database.

When the DAS system is fully operational, the DoHA will acquire data on a daily basis. Four jurisdictions are currently (July 2004) transferring data daily and the remaining jurisdictions will be sending daily data before the end of 2004. Data constraints are set in DAS as a quality control measure to ensure that the data downloaded into NNDSS are valid. Updates (including notifications requiring deletion) of existing notification records can be sent at any time.

Data accepted via DAS are stored in the data warehouse. Population data loaded into the warehouse provide the denominator for automatic calculation of crude notification rates. Data can be extracted from the warehouse by each of the epidemiologists in the Surveillance Section of the Commonwealth health department using the Warehouse Extraction and Reporting Tool (WERT) to produce frequency tables and/or cross tabulations. The interaction between DAS, the data warehouse and WERT is given in Figure 1.

Figure 1. Components of the National Notifiable Diseases Surveillance System data



CDA Communicable Diseases Australia.
DAS Data Acquisition System.
NNDSS National Notifiable Diseases Surveillance System.
QA Quality Assurance.
WERT Warehouse Extraction and Reporting Tool.

The jurisdictional data managers were asked to comment on DAS in the NSC survey. Of the four data managers who participated, three were currently using DAS. These data managers reported that DAS has either made no change to the ease of operation (n=1) or has made it easier to send data to NNDSS (n=2). Two of the data managers reported that the process of changing over to DAS had been easy.

Analysis and interpretation of data

Data received from the jurisdictions are analysed by the Surveillance Section epidemiologists every fortnight. A table of the number of notifications received in the past fortnight and for the year to date (YTD) are listed for each jurisdiction and each disease. A national five-year mean of YTD notifications is also calculated for each disease. The current YTD notifications at a national level are compared against the five-year mean YTD +/- two standard deviations. Data are interpreted through the cooperation of the DoHA and jurisdictions through CDNA and the NSC.

Notification tables are published in each quarterly edition of *CDI*. Each jurisdiction is asked to comment on any outbreaks or communicable disease events in that quarter that warrant attention. The largest analysis conducted by the DoHA is done for the annual report of NNDSS. Each disease is examined in detail and the year's data are compared with historical data to determine if the epidemiology of the disease is changing. Maps showing the notification rates by Statistical Division are produced for selected diseases. Each year a surveillance survey is sent to all jurisdictions asking whether they have changed surveillance practices or implemented any disease control programs. The results from the survey enable DoHA epidemiologists to interpret the data held in NNDSS.

There are two major problems relating to the analysis of NNDSS data by the DoHA. Firstly, there has been a long history of disagreement between DoHA notification totals and jurisdictional notification totals. This usually results in a time consuming process of determining where the discrepancies might arise and trying to harmonise the data. In theory, the number of notifications should be the same. There is a requirement for state and territory data managers to have access to their own data located in the warehouse at DoHA.

The second major issue is that NNDSS data are not timely and not sufficiently detailed (e.g. serotype and phage type information is required for all *Salmonella* notifications) to detect multi-jurisdictional outbreaks. NNDSS data are published quarterly in tables and hence only serve as an historical record. This issue was cited as a major flaw by six of the CDNA members. However, it was generally recognised by the members that a lack of epidemiologists within the Surveillance Section at the DoHA has been a major limitation in the past. Currently, there are four epidemiologists at the DoHA who have contact with NNDSS. There is a need for the DoHA to conduct regular analyses to determine possible multi-jurisdictional outbreaks for appropriate diseases.

Data analyses are conducted in response to requests from the public, research organisations or community groups. The DoHA can produce basic aggregated data and provide some interpretation of the data. Requests for more detailed information, such as a list of notifications for a certain disease must be approved by CDNA. This process protects the integrity and the confidentiality of the data.

Reporting procedures

The main reporting channels of NNDSS data are through *CDI* and the *Communicable Diseases Australia* (CDA) website. The main reports published in *CDI* are the quarterly surveillance reports and the annual report of NNDSS. In terms of reporting to jurisdictions at CDNA teleconferences, the DoHA provides a fortnightly table of notifications received from each state and territory and also provides brief commentary on disease activity. The report is then put on the CDA website for public access.

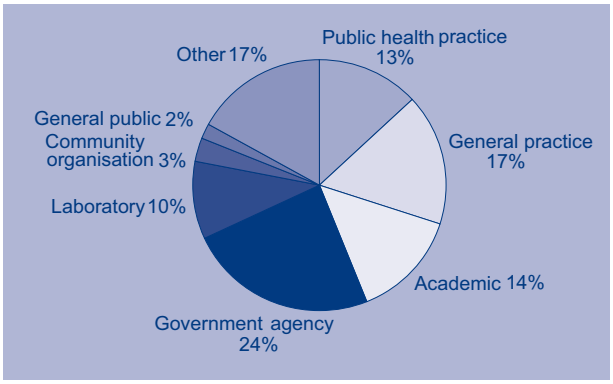
Dissemination of data

The main channels of dissemination are through the CDA website and *CDI*. The Commonwealth health department has produced *Communicable Diseases Intelligence* since 1976. *CDI* was originally published fortnightly as surveillance data with commentary, but changed to a monthly publication. Currently, *CDI* is published as a quarterly journal which also contains peer-reviewed articles on communicable diseases in Australia.

Results from the *CDI* survey suggest that *CDI* has a diverse readership. A quarter of the respondents were from government agencies and 17 per cent were from general practice (Figure 2). Some of the professions included in the 'other' category include, vaccine manufacturers, aged care facilities, health ethicists, private practitioners and infection control consultants.

The *CDI* readership were asked how they access NNDSS data. Of the 454 people who said they use NNDSS data, 451 (99%) reported using data published in *CDI*, 24 (5%) reported accessing the data through requests to the Commonwealth and 160 (35%) accessed NNDSS data through the CDA website. *CDI* readers reported that the easiest way

Figure 2. Profession of respondents to the *Communicable Diseases Intelligence* readership survey



for them to access NNDSS data was through the hard copy of *CDI* (Table 4). The CDA website, which was recently redeveloped, was reported as difficult to access. This is supported by some of the CDNA members who think that the CDA website should be redesigned to allow greater access to summary level data.

Flow of data through NNDSS

The structure and flow of data through NNDSS are shown in Figure 3. The flow of data starts from the person who presents to a health service when they are ill, to the state or territory health authority who then collates and verifies the data and responds to the report if required. The jurisdictions then send the data to the DoHA for inclusion into the nationally aggregated dataset. The data are analysed and interpreted and then disseminated through *CDI*, the CDA website and requests. CDNA provides the forum for jurisdictions and the Commonwealth, along with other institutions and stakeholders, to manage and discuss surveillance data. All levels of surveillance depicted in the flow diagram (Figure 3) represent networks of people (i.e. epidemiologists, data managers, data providers), infrastructure (i.e. offices, computers, telephones and faxes) and resources (i.e. funding).

Evaluation of system attributes

The evaluation of system attributes is a process that depends largely on the objectives of the system. Since NNDSS has no documented objectives, the evaluation of the attributes is relative to the current use of NNDSS and existing DoHA structures. Another complicating factor in evaluating the attributes of NNDSS is that there are differences in perceptions about NNDSS amongst the key stakeholders. For example, some people thought that NNDSS is just the database of notifications held at the Commonwealth. Others see NNDSS as the whole network of people and data systems that provide and/or use data to contribute to national biosecurity.

Simplicity

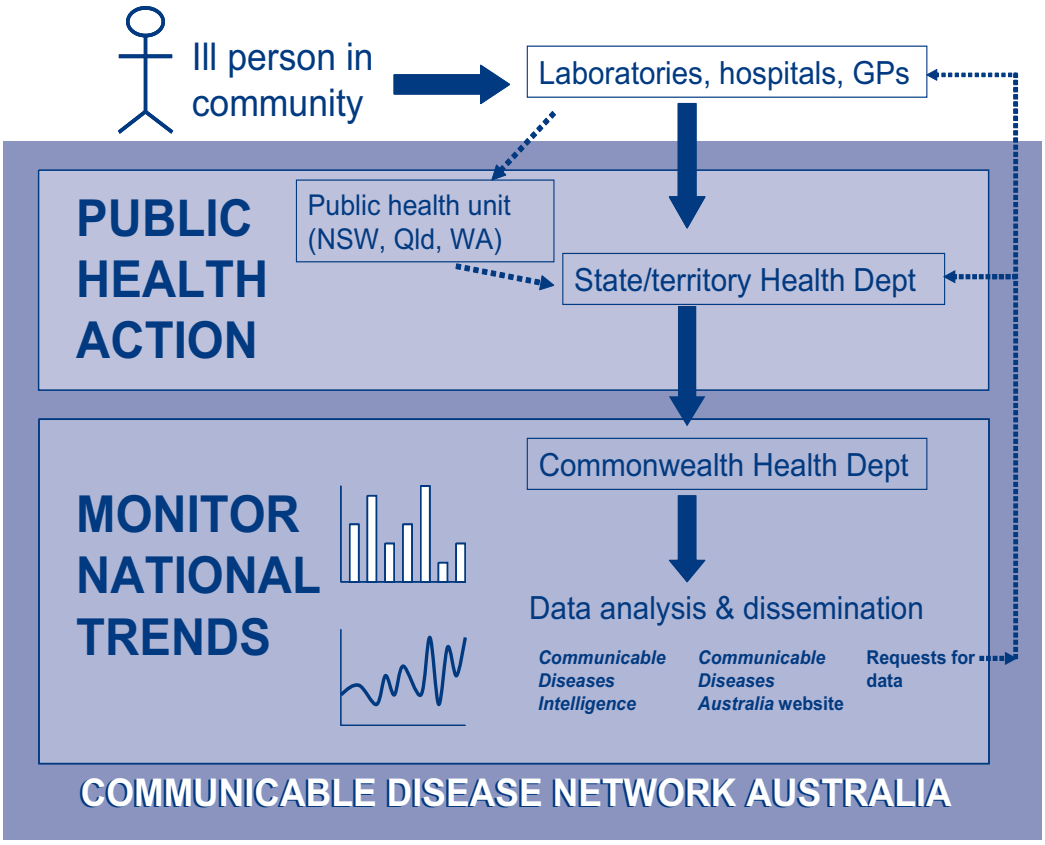
The structure of NNDSS is quite simple, as summarised in Figure 3. However, the operation and processes that govern NNDSS are complex and inefficient. Given that there are eight different jurisdictions which collect surveillance data in their own way, it has taken a long time to develop a standardised national database.

Data analysis procedures have been traditionally based in Microsoft (MS) Access, where standard queries have been written and used to extract the data into a MS Excel spreadsheet for the production of graphs. The process of analysis is currently under redevelopment. The Warehouse Extraction and Reporting Tool has the facility to extract data from the warehouse and import it into MS Excel. This feature will be used mainly for generating the standard tables in the *CDI* quarterly reports and other routine reporting (e.g. reports for expert committees). There will still be an ability to interrogate the NNDSS database using MS Access queries for ad hoc analyses.

Table 4. Accessibility of the three main forms of dissemination of National Notifiable Diseases Surveillance System data, *Communicable Diseases Intelligence* readership survey

Form of NNDSS data	Accessibility (% respondents who reported using NNDSS data)		
	Easy	Neutral	Difficult
CDI hard copy	81	18	1
CDA website	65	30	5
Request	60	24	16

Figure 3. Structure and flow of data through National Notifiable Diseases Surveillance System



Flexibility

One of the main weaknesses of the NNDSS database is its inability to implement changes in national surveillance rapidly. Although a national surveillance system does not have to be flexible, there were still several areas where the inflexibility of the database resulted in significant delays to improvement within the system. Given the federated nature of governance in Australia, it will always be difficult for NNDSS to be flexible as any proposed changes to NNDSS have to rely on consensus of CDNA members.

The inflexibility of the system was highlighted with the recent revision of the case definitions. The case definitions have been under review for the past two years. The process has been driven by CDNA, but there have been prolonged negotiations over some of the case definitions, which has delayed the process. In addition, the revision of case definitions was a low priority during the SARS epidemic.

Data quality and completeness

The quality and completeness of data sent to NNDSS through DAS from 1 January to 28 May 2002 was examined. The main issues identified were the differences between jurisdictions in the use of the names of specific organisms and their serogroup and subtype names, miscoded dates and ambiguity in the coding for unknowns for postcodes. Some data fields were not being reported at all, such as 'case found by' and 'imported from overseas'. One jurisdiction was using the outbreak reference data field, but this requires further development. One of the most poorly reported data fields was Indigenous status. There is a national effort attempting to resolve the poor reporting of Indigenous status in disease reporting through the *Improving Identification in Communicable Disease Reporting Project Steering Committee*.

Acceptability

One of the major strengths of NNDSS is its high level of acceptability. Despite the lack of a legislative requirement for jurisdictions to report to the Commonwealth and limited Commonwealth funding of jurisdiction surveillance activities, all states and territories participate in NNDSS. Participation was judged by the attendance of a jurisdictional representative at every CDNA teleconference and NSC teleconference and that each state and territory sends data to the DoHA at regular intervals. Acceptability also goes beyond jurisdictions, as members from key institutions and national centres are also active participants in NNDSS.

From the CDNA member survey, even though nearly every member reported being very busy, there was still an obvious engagement with the system and its value was recognised:

> *'In Australia, we have the continuing problem of federalism where we need to line up jurisdictional requirements for notification…problems in relation to communicable disease control are not that huge, as people are very cooperative.'*

There was a wide spectrum of engagement in NNDSS. The main reason for less enthusiastic participation in NNDSS has been competition with other priorities within the jurisdiction. One member described the situation in that they are ultimately responsible to their state or territory Health Minister, not the federal Health Minister. One member described the situation:

> *'the issue that always comes up [with national surveillance] is, what are the state/territory's priorities and what are our resourcing levels?'*

Representativeness

Given that all jurisdictions are participating in NNDSS, the entire population of Australia should be under surveillance. The representativeness of NNDSS will only ever be as good as the jurisdiction based surveillance activities. It is unlikely that NNDSS, like any other national surveillance system, will be fully representative. NNDSS does not have to be representative to be useful, but we must be aware of possible over-represented and under-represented populations. The main barrier to representativeness in Australia is geography. There are many communities living in remote and rural locations in Australia which are generally under-serviced in terms of access to health care.

Representativeness is also affected by the lack of uniformity in diagnostic and reporting practices by clinicians. For example, a person might go to their General Practitioner (GP) with gastrointestinal complaints. A GP in one area will ask for a stool sample for laboratory testing, which may lead to a notification, while a GP in another area will not take samples for testing and notification will not occur.

The Indigenous population in surveillance data is likely to be under-represented. This is due to complex socio-political factors associated with identification as an Indigenous person. The main issues are related to identification (i.e. self identifying or community acceptance) and whether there should be a requirement to ask people their Indigenous status.

Areas of better ascertainment in NNDSS are largely disease specific. For diseases that have screening programs in selected jurisdictions, the surveillance data are more reflective of the screening process and are biased towards more complete ascertainment of cases in subpopulations. For example, sexually transmissible infection screening amongst Indigenous people and screening for *Chlamydia* in people attending sexual health clinics will lead to more notifications because the health authorities are looking for the disease.

Timeliness

The timeliness of NNDSS will only be as good as the jurisdiction with the slowest reporting time to the system. An analysis presented in the *Report of the Communicable Disease Surveillance Project*[10] suggested that from 1992–1998, the average number of days from onset of illness to notification to jurisdictions (for all diseases) was 18.1 days (ranging from an average delay of 12.3 days in Australian Capital Territory to 20.9 days in Victoria). There were further delays of up to a fortnight in jurisdictions sending data to DoHA. However, the introduction of DAS has meant that jurisdictions can now send daily updates. This will enable NNDSS to detect outbreaks, if the sensitivity is high enough for the diseases of interest and if the data are of high quality.

Stability

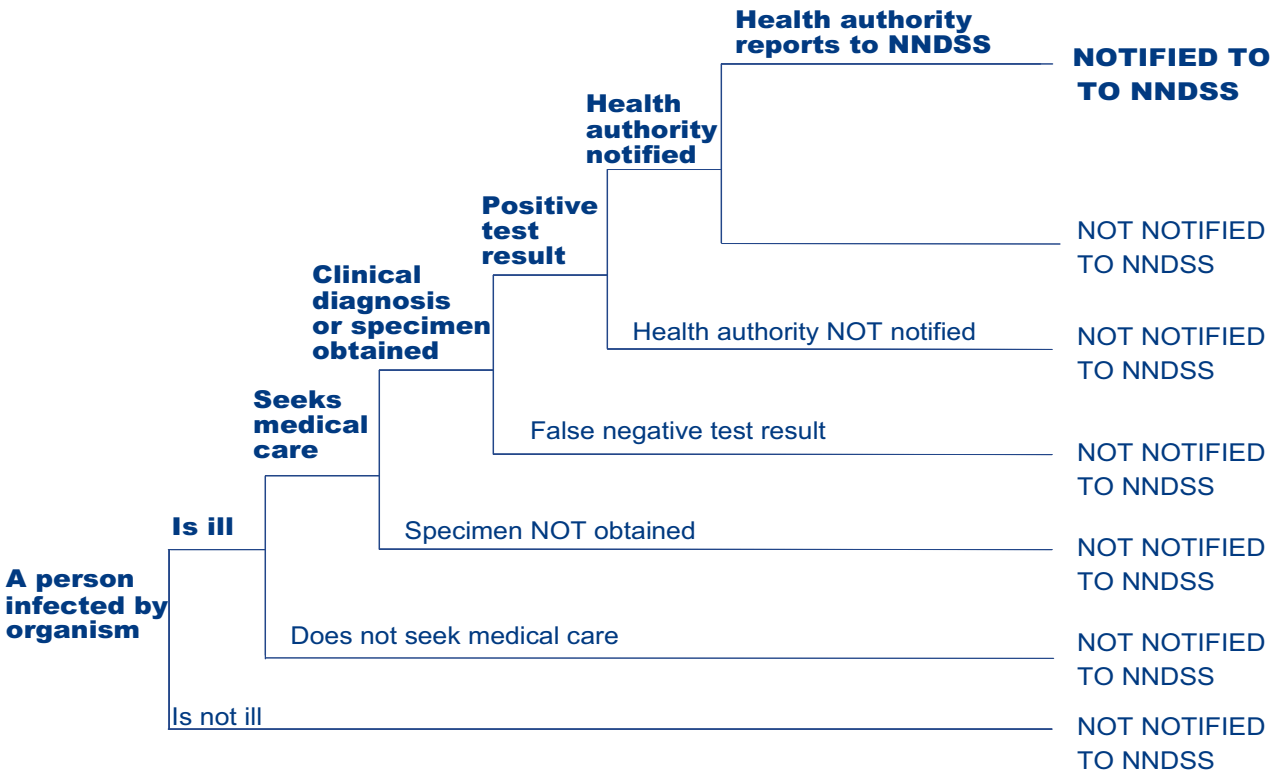
NNDSS is generally stable. Quarterly surveillance reports and annual reports are usually published on time. There have been periods in the past where annual reports have been several years out of date. The challenge is now for the DoHA to provide more frequent reports on disease activity at a national level. Epidemiologists at the DoHA can access NNDSS data whenever it's required.

Sensitivity

The sensitivity of NNDSS will vary across diseases and across jurisdictions. Figure 4 conceptualises the steps in surveillance that determine the fraction of cases that will be notified to NNDSS. The notification fraction will be different for each disease under surveillance. At a national level it is difficult to establish what proportion of true cases in the community are not notified.

Surveillance systems for most diseases do not have to detect every single case for the system to inform public health action. Varicella became notifiable in South Australia in January 2002 and sensitivity calculations indicate that only 4 per cent of cases were detected by the system.[12] Even with these data it is possible to gain sufficient information as a base line to monitor current and future trends.

Figure 4. Fraction of cases notified to National Notifiable Diseases Surveillance System [11]



Discussion

The new biosecurity environment has provided the impetus for this evaluation of NNDSS. Stakeholder interviews were structured to obtain qualitative and quantitative results, which provided essential insights into the functioning of Australia's national communicable disease surveillance system. Although NNDSS has no clearly written objectives, there was universal agreement amongst stakeholders that the following points were being addressed through NNDSS:

- NNDSS provides a nationally coordinated surveillance system for notifiable communicable diseases;
- NNDSS is enmeshed in local, state, national and international public health practice;
- through CDNA and other key stakeholders, NNDSS collects and disseminates interpreted public health information to direct action at all levels of the health system.

NNDSS has been used to inform public health action, mainly in the area of vaccine preventable diseases. To have a greater impact on communicable disease control, the data from NNDSS has to be linked to control activities. This can be done through setting national priority areas and using NNDSS to monitor progress towards controlling diseases that are priorities. The process of communicable disease control and surveillance prioritorisation could be conducted through a review of the National Communicable Disease Surveillance Strategy.

The major strength of NNDSS lies in its universal acceptability by the stakeholders and its accepted role as the primary source of national communicable disease data. Members of CDNA are committed to participation in the system, but emphasised that jurisdictional priorities come first. NNDSS was also found to be stable and simple in structure.

The foundation of notification rests in the Australian Constitution, which gives the legislative power for notification to the states and territories. Therefore, any changes to notification parameters and mechanisms require consensus from CDNA. NNDSS was found to be inflexible to rapid change and was not timely. The issue of timeliness was strongly related to whether NNDSS should be used to detect national outbreaks. There was disagreement among stakeholders about whether this should be an objective of NNDSS. If the DoHA is moving towards the daily updates of notification data from the jurisdictions, then there is no reason why, with

the appropriate information technology support, that cluster analyses and mapping cannot be conducted at a national level. Data completeness and quality will also have to be addressed for valid analyses to be conducted.

The evaluation of NNDSS had several limitations. Firstly, there was some selection bias when choosing the stakeholders to be included in the consultation. The stakeholder consultation should have included every member of CDNA, not just the jurisdictions and representatives from the national centres. Major public health laboratories should have also been included in the stakeholder consultation. Secondly, there was a poor response to the data managers survey. It would be valuable to know the barriers to participation amongst the data managers. Thirdly, the consultation process did not include all stakeholders from within the Commonwealth. However, the use of NNDSS data by Commonwealth Sections and/or expert technical committees was included in the evaluation.

In summary, NNDSS is a highly valued and important source of information on communicable disease activity in Australia. The system has undergone significant change over the past few years to improve its functioning. With the recent federal funding for biosecurity, the opportunity exists to improve NNDSS to enable it to meet its full potential within the existing federated framework of disease surveillance and control in Australia.

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Surveillance of adverse events following immunisation: Australia 2002 to 2003

Glenda Lawrence,[1] Ian Boyd,[2] Peter McIntyre,[1] David Isaacs[3]

Abstract

Reports of suspected adverse events following immunisation (AEFI) are reviewed by the Adverse Drug Reactions Advisory Committee and collated in a central database. We analysed AEFI records for vaccines administered during October 2002 to December 2003, and assessed AEFI reporting trends for 2000 to 2003. AEFI reporting rates were calculated using denominator data from the Australian Childhood Immunisation Register and the annual national influenza vaccination coverage survey. A total of 1,744 AEFI records were analysed for October 2002 to December 2003. The majority described non-serious events; 9 per cent (n=149) described AEFIs defined as 'serious'. Four deaths were reported but none were causally related to immunisation. Dose-based AEFI reporting rates were 2.1 per 100,000 doses of influenza vaccine for adults aged 40 years or over and 19.8 per 100,000 doses of scheduled vaccines for children aged <7 years. The most frequently reported individual AEFI was injection site reaction in children after a fourth or fifth dose of an acellular pertussis-containing vaccine (54 and 98 reports per 100,000 doses respectively). The most frequently suspected vaccine was meningococcal C conjugate vaccine (34% of reports—mostly injection site reactions, gastrointestinal symptoms and headaches). The average annual reporting rate was 7.0 per 100,000 population, the highest to date. The increase in the AEFI reporting rate was due to a greater number of children becoming eligible to receive a fourth or fifth consecutive dose of acellular pertussis vaccine and the introduction of the meningococcal C vaccination program in January 2003 for those aged 1–19 years. The low reporting rate of serious AEFIs demonstrates the high level of safety of vaccines in Australia. *Commun Dis Intell* 2004;28:324–338.

Keywords: AEFI, adverse events, vaccines, surveillance, immunisation, vaccine safety

Introduction

An adverse event following immunisation (AEFI) is defined as any serious or unexpected adverse event that occurs *after* a vaccination has been given which may be related to the vaccine itself or to its handling or administration.[1] An AEFI may be *coincidentally* associated with the *timing* of immunisation without necessarily being caused by the vaccine or the immunisation process. AEFI surveillance is an integral component of all vaccination programs to monitor vaccine safety and detect rare, late-onset, unexpected and population-specific adverse events that are difficult to detect in pre-licensure vaccine trials.[2,3]

In Australia, passive AEFI surveillance is conducted by state and territory health departments and the Adverse Drug Reactions Unit (ADRU), which is part of the Australian Government Therapeutic Goods Administration and provides the secretariat for the Adverse Drug Reactions Advisory Committee (ADRAC).[4,5,6] Immunisation providers, other health care professionals, vaccine manufacturers, parents and members of the public report suspected AEFIs to the ADRU, either directly or via the relevant state or territory health department as required.[5,6] At the ADRU, all reports are evaluated using internationally consistent criteria[7] and are reviewed by the ADRAC at regular meetings. The data collected by these passive surveillance methods are used to monitor trends, detect signals and generate hypotheses.

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The first AEFI surveillance report using national data collated in the ADRAC database was published in September 2003 and focussed on AEFIs reported for vaccines administered between 1 January 2000 and 30 September 2002.[4] It provided the first estimates of national dose-based AEFI reporting rates for the most commonly used vaccines in Australia and demonstrated an increase over time in reports of injection site reactions following a fourth or fifth dose of acellular-pertussis containing vaccines.

This report focuses mainly on AEFIs reported to ADRAC for vaccines administered between 1 October 2002 and 31 December 2003 and compares reporting trends for the four years 2000 to 2003. Several important surveillance and immunisation program initiatives occurred during the October 2002 to December 2003 timeframe which impact on the AEFI surveillance data presented in this report. A new data management system was implemented by the ADRU in November 2002 to replace the original system that dated from 1972. At the same time the ADRU changed from the World Health Organization Adverse Reaction Terminology (WHO-ART) coding system to the Medical Dictionary of Regulatory Activities (MedDRA®) system. This is an international medical terminology developed under the auspices of the International Conference on Harmonization of Technical Requirements for Registration of Pharmaceuticals for Human Use.[8] During implementation of the new data management system, reaction terms in reports entered into the database between January 2000 and November 2002 were converted from the WHO-ART terms to the most closely related term in the MedDRA® reaction term coding system.

In January 2003 the meningococcal C conjugate vaccine (MenCCV) was introduced into the Australian Standard Vaccination Schedule (ASVS). Children born on or after 1 January 2002 became eligible for a single dose of the vaccine at 12 months of age and a provider and school-based catch-up campaign was implemented for older children and adolescents up to 19 years of age through the National Immunisation Program.[6] The exact timing of the catch-up campaign varied between jurisdictions. In September 2003, the fourth dose of diphtheria-tetanus-acellular pertussis (DTPa) vaccine, due at 18 months of age, was removed from the ASVS.[6]

Methods

Data source

De-identified information was released to the National Centre for Immunisation Research and Surveillance for all drug and vaccine adverse event notifications entered into the ADRAC database between 1 January 2000 and 7 April 2004. The AEFI surveillance system and methods used by ADRU to evaluate AEFI reports are described in detail in the first AEFI surveillance report.[4]

ADRAC database records* were eligible for inclusion in the analysis of AEFIs if:

- a vaccine was recorded as 'suspected' of involvement in the reported adverse event; *and*
- *either* (a) the vaccination occurred between 1 January 2000 and 31 December 2003 *or* (b) if no vaccination date was recorded, the date of onset of symptoms or signs occurred between 1 January 2000 and 31 December 2003.

Study definitions of AEFI outcomes and reactions

AEFIs were defined as 'serious' or 'non-serious' based on information recorded in the ADRAC database and criteria similar to those used by the World Health Organization[7] and the United States of America (USA) Vaccine Adverse Events Reporting System (VAERS).[9] In this report, an AEFI is defined as 'serious' if the record indicated the person had recovered with sequelae, was hospitalised, experienced a life-threatening event, or died.

Typically, each AEFI record listed several symptoms, signs and diagnoses. We used the MedDRA® terms to create a set of reaction categories for analysis. Initially, reaction terms were grouped to create reaction categories analogous to the AEFIs listed and defined in the *Australian Immunisation Handbook* (7th edition).[5] Reaction categories were then created for the remaining reaction terms that were listed in more than one per cent of AEFI records. Finally, terms listed in less than one per cent of records were grouped into broader categories mainly based on the organ system where the reaction was manifested (e.g. gastrointestinal, neurological). The impact of the change in reaction term coding from the WHO-ART to MedDRA® systems was assessed by comparing the frequencies of reaction categories created for the 2,409 AEFI

* Note that the terms 'AEFI record' and 'AEFI notification' have specific meanings in this report. One 'AEFI notification' (a report to a relevant authority) may generate more than one 'AEFI record' in the ADRAC database if a number of adverse events are described in the notification (e.g. a local injection site adverse event and a systemic adverse event).[4] This report is based on 'AEFI records'

records that were coded using both systems and analysed previously.[4] The frequencies for all reaction categories based on AEFIs listed in the *Australian Immunisation Handbook*[5] were comparable for the two reaction term coding systems.

Data analysis

All data analyses were performed using the SAS version 8 computer program.[10] The distribution of AEFI records was analysed by age, gender and jurisdiction. Average annual population-based reporting rates were calculated for each state and territory and by age group using population estimates obtained from the Australian Bureau of Statistics.

The frequency and age distribution of AEFI outcomes, reaction categories and vaccines listed as 'suspected' of involvement in the reported adverse event was assessed. For each vaccine, we calculated the age distribution and the proportion of AEFI records where (i) the vaccine was the only suspected vaccine or drug, (ii) the AEFI record was assigned a 'certain' or 'probable' causality rating, and (iii) the AEFI was defined as 'serious'. Because many AEFI records listed more than one suspected vaccine and several reaction terms to describe an adverse event, column totals in the relevant tables exceed the number of AEFI records analysed.

Dose-based AEFI reporting rates were estimated for children aged <7 years for seven childhood ASVS vaccines funded through the National Immunisation Program (DTPa, DTPa-hepB, Hib, Hib-hepB, polio, MMR and MenCCV), and for adults aged 40 years and over for influenza vaccine. The number of administered doses of each of the seven childhood ASVS vaccines was calculated from the Australian Childhood Immunisation Register (ACIR), a national population-based register of approximately 99 per cent of children aged <7 years. Vaccine doses administered between 1 October 2002 and 31 December 2003 were estimated for the age groups <1 year, 1 to <2 years and 2 to <7 years (i.e. the age at vaccination). The number of administered influenza vaccine doses was estimated from the 2003 annual national influenza coverage survey[11] and mid-2003 population estimates for the 40–64 years and ≥65 years age groups. Dose-based AEFI reporting rates could not be determined for other vaccines and age groups due to the lack of reliable denominator data for the number of vaccine doses distributed or administered.

Notes on interpretation

Caution is required when interpreting the AEFI data presented in this report. Due to reporting delays and late onset of some AEFIs, the data are considered preliminary, particularly for the fourth quarter of 2003. The information collated in the ADRAC database is intended primarily for signal detection and hypothesis generation. While reporting rates of AEFIs can be estimated using appropriate denominators such as the number of vaccine doses administered, they cannot be interpreted as incidence rates due to under-reporting and biased reporting of suspected AEFIs, and the variable quality and completeness of information provided in individual notifications.[4,12]

It is also important to note that this report is based on vaccine and reaction term information collated in a database, and not on comprehensive clinical notes. Individual database records list symptoms, signs and diagnoses that were used to define a set of reaction categories based on the case definitions provided in the 7th edition of the *Australian Immunisation Handbook*.[5] However, these reaction categories are not identical to 'case definitions' of adverse events.

The reported symptoms, signs and diagnoses in each AEFI record in the ADRAC database are temporally associated with vaccination but are not necessarily causally associated with a vaccine or vaccines. The causality ratings of 'certain', 'probable' and 'possible' assigned to individual AEFI records describe the likelihood that a suspected vaccine or vaccines was/were associated with the reported reaction at the level of the individual. Factors that are considered in assigning causality ratings include the timing (minutes, hours etc) and the spatial correlation (for injection site reactions) of symptoms and signs in relation to vaccination, and whether one or more vaccines were administered.[4] Assigning a causality rating to an individual report is not the same as the epidemiological concept of 'causality' which applies at the population level and requires a specific epidemiological study with an appropriate control group to investigate. Signals and hypotheses generated from passive surveillance inform the need for such studies.

Results

Summary of data

A total of 1,744 AEFI records were entered in the ADRAC database where the date of vaccination or onset of an adverse event occurred between 1 October 2002 and 31 December 2003. This corresponded to approximately 1,575 individual AEFI notifications, with 11 per cent of AEFI notifications generating more than one AEFI record.

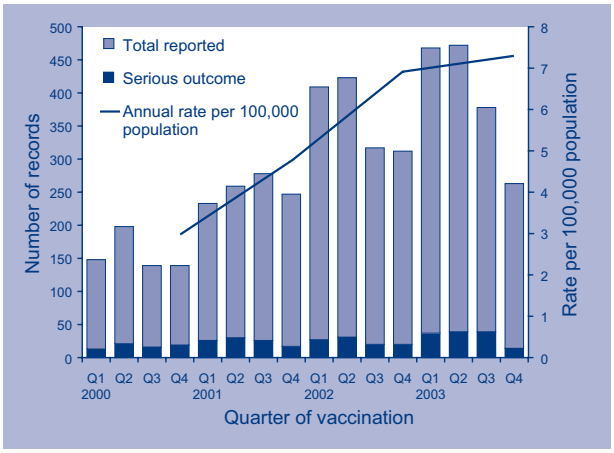
One hundred and forty nine AEFI records (9%) were defined as 'serious' (i.e. recovery with sequelae, requiring hospitalisation, experiencing a life-threat-

ening event or death). A total of 897 (51%) AEFI records were assigned causality ratings of 'certain' (n=745) or 'probable' (n=152).

AEFI reporting trends

The average annual AEFI reporting rate for the period October 2002 to December 2003 was 7.0 per 100,000 population. The rate and number of AEFIs reported in 2003 was higher than in previous years (Figure 1). The trends in AEFI notifications shown in Figure 1 are reflected in the trends in vaccines frequently suspected of involvement in reported AEFIs (Figure 2), and in the types of reactions frequently reported (Figure 3). The commencement of the MenCCV immunisation program in 2003 contributed to an overall increase in population-based AEFI reporting rates in 2003 compared with previous years.

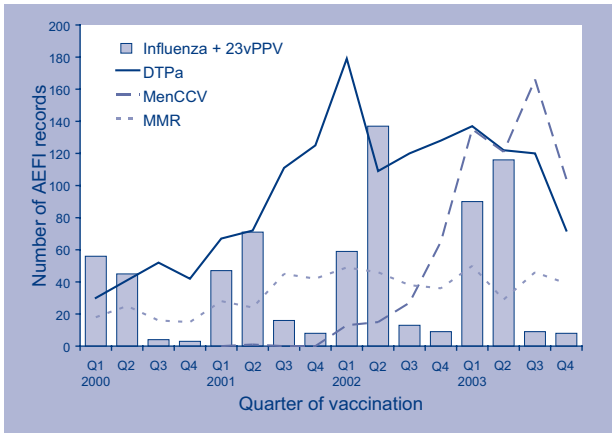
Figure 1. Adverse events following immunisation, ADRAC database, 2000 to 2003, by quarter of vaccination



Note: for reports where the date of vaccination was not recorded, the date of onset was used as a proxy for vaccination date.

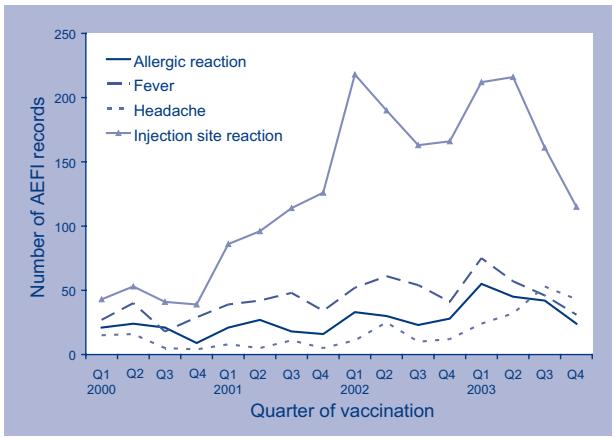
A clear seasonal pattern of AEFI reporting was apparent with the highest number of AEFI notifications for vaccinations administered in the first half of each year (Figure 1). These seasonal peaks correspond to the months when more vaccinations are administered in Australia, particularly among five year old children receiving DTPa and measles-mumps-rubella (MMR) vaccines prior to commencing school in February and older Australians receiving influenza and pneumococcal vaccines during the autumn months (March to June) (Figure 2).

Figure 2. Frequently suspected vaccines, adverse events following immunisation, ADRAC database, 2000 to 2003, by quarter of vaccination



Note: see appendix for abbreviations of vaccine names.

Figure 3. Selected frequently reported reactions, by quarter of vaccination, ADRAC database, 2000 to 2003

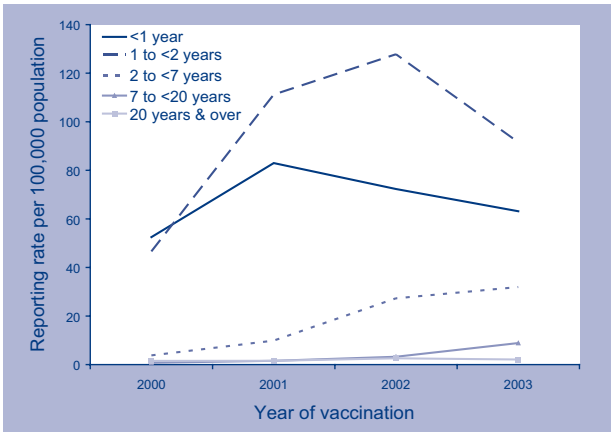


Age and gender distribution

The AEFI reporting rate in 2003 was highest among children aged <1 year and 1 to <2 years (63.1 and 91.5 per 100,000 population, respectively) (Figure 4), the age groups that receive the greatest number of vaccinations. The average annual AEFI reporting rates for these age groups decreased in 2003 compared with 2002, but increased among older age groups.

The overall male to female ratio was 1:1.2 although, as seen previously,[4] this differed by age group. There were more reports for males than females among children aged 1 to <7 years (1:0.8) and fewer reports for males than females aged 20 to 64 years (1:2).

Figure 4. Reporting rates of adverse events following immunisation per 100,000 population, ADRAC database, 2000 to 2003, by age group and quarter of vaccination



Geographical distribution

AEFI reporting rates varied between the states and territories for the period October 2002 to December 2003 (Table 1). The Australian Capital Territory and Northern Territory had the highest reporting rates (23.0 and 20.6 per 100,000 population, respectively) while Tasmania and Victoria had the lowest rates (3.9 and 4.5 per 100,000 population, respectively). In general, reporting rates of AEFIs assigned a 'certain' or 'probable' causality rating and those defined as 'serious' were less variable across jurisdictions. The greatest variation in population-based reporting rates between jurisdictions was for children aged <7 years (Table 1).

AEFI outcomes

The majority of reported AEFIs for the period October 2002 to December 2003 were defined as 'non-serious' (60%) while nine per cent were defined as 'serious' (Table 2). Fewer 'serious' AEFIs were assigned 'certain' or 'probable' causality ratings compared with 'non-serious' AEFIs (32% versus 55%). Death was recorded as the outcome in four AEFI records (Table 2). Although temporally related, none of these deaths was found to be causally related to vaccination.

Vaccines and AEFI

Twenty-five vaccines were recorded as 'suspected' of involvement in the adverse events described in the 1,744 AEFI records for the period October 2002 to December 2003 (Table 3). They included all vaccines recommended in the ASVS, plus vaccines recommended to travellers and specific risk groups.

Table 1. Adverse events following immunisation (AEFI), ADRAC database, October 2002 to December 2003, by jurisdiction

Jurisdiction	AEFI records		Annual reporting rate per 100,000 population*			
	n	%	Overall	'Certain' or 'probable' causality rating†	'Serious' outcome‡	Aged < 7 years
Australian Capital Territory	93	5.3	23.0	8.7	0.7	151.0
New South Wales	539	30.9	6.4	3.3	0.7	33.1
Northern Territory	51	2.9	20.6	9.3	2.4‖	83.0
Queensland	249	14.3	5.2	2.3	0.4	35.7
South Australia	308	17.7	16.1	9.3	0.8	155.2
Tasmania	23	1.3	3.9	2.5	0.2	14.9
Victoria	277	15.9	4.5	2.6	0.3	26.7
Western Australia	147	8.4	6.0	3.3	0.6	43.6
Other§	57	3.3	na	na	na	na
Total	1,744	100.0	7.0	3.6	0.6	45.2

* Average annual rates per 100,000 population calculated using mid-2003 population estimates (Australian Bureau of Statistics).

† See previous report[4] for criteria used to assign causality ratings.

‡ AEFI records defined as 'serious' (see Methods and Table 3).

§ Records where the jurisdiction in which the AEFI occurred was not reported (n=17) or was unclear (n=40). These included AEFIs notified by pharmaceutical companies (n = 35).

‖ Based on 6 reports.

Table 2. Outcomes shown in records of adverse events following immunisation (AEFI), ADRAC database, October 2002 to December 2003

Outcome	AEFI records		'Certain' or 'probable' causality rating†		Age group‡			
					< 7 years		≥ 7 years	
	n	%*	n	%§	n	%§	n	%§
Non-serious	1041	60	576	55	635	61	381	37
Not recovered at time of report	393	22	196	50	201	51	182	46
Not known (missing data)	161	9	77	48	105	65	53	33
Serious:	149	9	48	32	68	46	80	54
recovered with sequelae	0	0	–	–	–	–	–	–
hospital admission	132	8	45	34	61	45	71	54
life-threatening event	13	<1	3	23	5	38	7	54
death	4	<1	0	0	2	50	2	50
Total	1,744	100	897	51	1,009	58	696	40

* Percentages relate to the total number of AEFI records (n=1744).

† Causality ratings were assigned to AEFI records using criteria described previously.[4]

‡ AEFI records where age or date of birth was not recorded are not shown.

§ Percentages relate to the number of AEFI records with the specific outcome e.g. of 1041 AEFI records with a 'non-serious' outcome, 55% had causality ratings of 'certain' or 'probable' and 61% were for children aged <7 years.

The most frequently suspected individual vaccine was MenCCV with 591 (34%) of reports (Table 3). Vaccines containing pertussis, diphtheria and tetanus antigens (i.e. DTPa and DTPa-hepB) were suspected in 687 (39%) reports. The proportion of AEFI records where only one vaccine was suspected of involvement in the reported adverse event differed by vaccine, as did the proportion assigned causality ratings of 'certain' or 'probable', and the proportion defined as 'serious'.

AEFI reporting trends over time differed by vaccine (Figure 2). The number of reports of AEFIs for DTPa vaccine declined during 2003 following a peak in the first quarter of 2002. MMR reports remained relatively constant while reports for MenCCV vaccine increased following the addition of this vaccine to the National Immunisation Program and delivery through provider and school-based immunisation programs in 2003.

AEFI reactions

The distribution and frequency of reactions listed in AEFI records for October 2002 to December 2003 are shown in Tables 4 and 5. In Table 4, only the reaction categories analogous to those listed in the *Australian Immunisation Handbook*[5] are shown. In Table 5, other reaction categories are listed in descending order of frequency.

Injection site reactions were the most common category of reaction (n=870 or 50% of AEFI records) (Table 4) followed by fever (14%), allergic reactions (11%) and rash (10%). DTPa, MenCCV and 23-valent pneumococcal polysaccharide vaccine (23vPPV) were the most frequently suspected vaccines in the 870 AEFI records listing injection site reaction (50%, 21% and 12% respectively). DTPa and MenCCV were also the most frequently suspected vaccines in AEFI records listing fever (24% and 42% respectively) and allergic reaction (23% and 44% respectively).

More serious AEFIs included anaphylactic reaction (n=9), hypotonic-hyporesponsive episode (HHE, n=12), thrombocytopenia (n=6) and convulsions (n=52). The most commonly suspected vaccines in the reports of anaphylactic reaction were influenza (n=3) and MenCCV (n=3). DTPa was the most commonly suspected vaccine in reports listing HHE (10/12, 83%), while MMR was the most commonly suspected vaccine in reports listing thrombocytopenia (4/6, 67%). Of the 52 reports listing convulsions as a reaction term, 32 (62%) listed MenCCV and 13 (25%) listed DTPa as a suspected vaccine.

Of reactions not listed in the Australian Immunisation Handbook,[5] oedema (13%), headache (9%), vomiting (6%) and malaise (6%) were the most frequently recorded (Table 5). DTPa and MenCCV were the most commonly suspected vaccines in 225 AEFI records where oedema was listed as a reaction (52% and 23% respectively), while MenCCV was suspected of involvement in 73% (n=120) of the 164 AEFI records that listed headache as a reaction. MenCCV was also suspected in the majority of AEFI records that listed syncope (26/38, 68%) or loss of consciousness (15/27, 55%) as a reaction term.

Table 3. Vaccine types listed as 'suspected' in records of adverse events following immunisation (AEFI), ADRAC database, October 2000 to December 2003

Suspected vaccine type*	AEFI records	One suspected vaccine or drug only†		'Certain' or 'probable' causality rating‡		'Serious' outcome§		Age group‖			
								< 7 years		≥ 7 years	
	n	n	%¶	n	%¶	n	%¶	n	%¶	n	%¶
MenCCV	591	469	79	249	42	63	11	277	47	305	52
DTPa	579	390	67	354	61	22	4	564	97	5	1
MMR	200	32	16	23	12	16	8	189	95	7	4
Polio	180	4	2	6	3	24	13	176	98	2	1
23vPPV	151	132	87	97	64	9	6	4	3	136	90
Hib	126	11	9	12	10	17	13	125	99	0	0
DTPa-hepatitis B	108	18	17	16	15	12	11	106	98	1	1
Influenza	98	81	83	42	43	13	13	2	2	94	96
dT	73	47	64	34	47	2	3	5	7	67	92
Hepatitis B	58	39	67	27	47	10	17	18	31	39	67
Varicella	40	36	90	10	25	3	8	22	55	17	43
Hib-hepatitis B	36	4	11	3	8	10	28	36	100	0	0
Q fever	28	28	100	18	64	2	7	1	4	25	89
7vPCV	25	12	48	6	24	6	24	25	100	0	0
Hepatitis A + B	13	9	69	3	23	2	15	0	0	12	92
JE	11	9	82	4	36	3	27	1	9	10	91
BCG	6	6	100	3	50	4	67	0	0	6	100
Rabies	6	4	67	0	0	2	33	0	0	6	100
Tetanus	6	5	83	3	50	1	17	0	0	6	100
Hepatitis A	5	1	20	0	0	1	20	1	20	4	80
Men4PV	5	5	100	1	20	2	40	1	20	2	40
Typhoid	5	0	0	0	0	0	0	0	0	5	100
Hepatitis A + Typhoid	3	3	100	1	33	0	0	1	33	2	67
Anthrax	2	2	100	1	50	2	100	0	0	2	100
Yellow fever	2	1	50	0	0	0	0	0	0	2	100
Total**	1,744	1,349	77	897	51	149	9	1,009	58	696	40

* See appendix for abbreviations of vaccine names.

† AEFI records where only one vaccine was suspected of involvement in a reported adverse event.

‡ Causality ratings were assigned to AEFI records using criteria described previously.[4]

§ 'Serious' outcomes are defined in the Methods section (see Table 2 also).

‖ AEFI records not shown if age or date of birth was missing.

¶ Percentages are calculated for the number of AEFI records where the specific vaccine was suspected of involvement in the AEFI e.g. MenCCV was listed as 'suspected' in 591 AEFI records; this was the only suspected vaccine in 79% of the 591 AEFI records, 42% had 'certain' or 'probable' causality ratings, 11% were defined as 'serious' and 47% were for children aged <7 years.

** Total number of AEFI records analysed, not the total in each column.

Table 4. Reactions of interest* listed in records of adverse events following immunisation (AEFI), ADRAC database, October 2002 to December 2003

Reaction*	AEFI records	Only reaction reported†		Certain/ probable causality rating‡		Age group§			
						< 7 years		≥ 7 years	
	n	n	%‖	n	%‖	n	%‖	n	%‖
Injection site reaction	870	454	52	698	80	585	67	260	30
Fever	250	21	8	64	26	149	60	99	40
Allergic reaction	194	57	29	62	32	108	56	84	43
Rash	171	54	32	49	29	109	64	60	35
Convulsions	52	12	23	14	27	27	52	23	44
Abnormal crying	45	9	20	5	11	44	98	1	2
Arthralgia	35	2	6	13	37	2	6	30	86
Lymphadenopathy/itis¶	27	5	19	9	33	11	41	15	56
HHE**	12	2	17	1	8	11	92	0	0
hypotonia/hypokinesia**	16	0	0	3	19	13	81	3	19
Anaphylactic reaction	9	3	33	2	22	3	33	6	67
Abscess	6	4	67	5	83	3	50	3	50
Thrombocytopenia	6	3	50	0	0	5	83	1	17
Arthritis	4	1	25	0	0	1	25	3	75
Death	4	3	75	0	0	2	50	2	50
Parotitis	4	3	75	0	0	2	50	1	25
Meningitis	2	0	0	0	0	2	100	0	0
Brachial neuritis	1	0	0	1	100	0	0	1	100
Encephalitis	1	1	100	0	0	1	100	0	0
Guillain-Barré syndrome	1	0	0	0	0	0	0	1	100
Orchitis	1	1	100	0	0	0	0	1	100
Acute flaccid paralysis	0	0	0	0	0	0	0	0	0
Encephalopathy	0	0	0	0	0	0	0	0	0
Osteitis	0	0	0	0	0	0	0	0	0
Osteomyelitis	0	0	0	0	0	0	0	0	0
Sepsis	0	0	0	0	0	0	0	0	0
SSPE††	0	0	0	0	0	0	0	0	0
Toxic shock syndrome	0	0	0	0	0	0	0	0	0
Total	1,744	742	43	897	51	1,009	58	696	40

* Reaction term variables were created for the AEFIs of interest listed in the *Australian Immunisation Handbook*, (7th edition, p 22–23 and 271–275)[5] as described in Methods section.

† AEFI records where only one reaction was reported.

‡ Causality ratings were assigned to AEFI records using criteria described previously.[4]

§ AEFI records not shown if age or date of birth was missing.

‖ Percentages relate to the number of AEFI records in which the specific reaction term was listed e.g. of 870 AEFI records listing injection site reaction, 52% listed only one type of reaction while 80 per cent had causality ratings of 'certain' or 'probable' and 67% were for children aged <7 years.

¶ Includes lymphadenitis following BCG vaccination and the more general term of 'lymphadenopathy'.

** Hypotonic-hyporesponsive episode (HHE). The separate reaction term of 'hypotonia/hypokinesia' indicates records where 'HHE' was not listed but other terms describing an HHE or similar event were.

†† Subacute sclerosing panencephalitis.

Table 5. 'Other'* reactions listed in records of adverse events following immunisation (AEFI), ADRAC database, October 2002 to December 2003

Reaction*	AEFI records	Only reaction reported†		Certain/probable causality rating‡		Age group§			
						< 7 years		≥ 7 years	
	n	n	%‖	n	%‖	n	%‖	n	%‖
Oedema	225	6	3	154	68	151	67	70	31
Headache	164	25	15	59	36	15	9	145	88
Vomiting	109	11	10	28	26	60	55	46	42
Malaise	100	5	5	23	23	33	33	66	66
Pain	81	0	0	38	47	16	20	64	79
Dizziness	56	1	2	26	46	2	4	53	95
Nausea	52	1	2	12	23	4	8	48	92
Pallor	51	0	0	16	31	29	57	21	41
Irritability	48	1	2	10	21	42	88	6	13
Erythema	45	3	7	14	31	30	67	15	33
Respiratory rate/rhythm change	43	4	9	13	30	17	40	25	58
Syncope	38	3	8	22	58	8	21	30	79
Fatigue	35	1	3	10	29	11	31	21	60
Myalgia	35	0	0	16	46	3	9	29	83
Reduced sensation	33	2	6	17	52	1	3	32	97
Increased sweating	32	0	0	14	44	12	38	20	63
Anorexia	30	0	0	6	20	22	73	7	23
Diarrhoea	29	2	7	6	21	20	69	8	28
Abdominal pain	28	0	0	8	29	5	18	23	82
Loss of consciousness	27	0	0	10	37	8	30	19	70
Pharyngitis	24	1	4	6	25	5	21	18	75
Heart rate/rhythm change	23	0	0	11	48	5	22	18	78
Somnolence	20	3	15	7	35	11	55	9	45
Other									
general non-specific	108	6	6	53	49	53	49	54	50
neurological	74	10	14	28	38	25	34	47	64
respiratory	49	5	10	9	18	26	53	20	41
cardiovascular	47	5	11	21	45	14	30	32	68
musculoskeletal	46	1	2	22	48	5	11	41	89
skin	44	8	18	18	41	31	70	13	30
psychological	42	0	0	15	36	18	43	22	52
eye or ear	23	0	0	7	30	10	43	13	57
gastrointestinal	18	1	6	6	33	7	39	10	56
metabolic/endocrine	18	1	6	4	22	12	67	6	33
renal/urogenital	9	1	11	4	44	1	11	8	89
haematological	7	0	0	2	29	1	14	6	86
infection	5	1	20	0	0	5	100	0	0
miscellaneous	2	0	0	0	0	1	50	1	50
pregnancy/congenital	1	1	100	0	0	0	0	0	0

* Reaction terms not listed in the *Australian Immunisation Handbook*[5] but included in AEFI records in the ADRAC database. The top part of the table shows reaction terms included in 1% or more of AEFI records; the bottom part of the table shows reaction terms grouped by organ system that were included in <1% of AEFI records.

NOS Not otherwise specified.

Note: Please see Table 4 for a description of other footnotes.

Reactions mentioned in fewer than one per cent of AEFI records for October 2002 to December 2003 are shown grouped by higher or organ system categories in the lower portion of Table 5. The most commonly reported category was 'general non-specific'. This included broad terms that could not be assigned to an organ system (e.g. 'influenza-like illness'; 'discomfort not otherwise specified').

The trends in the most frequently reported types of reactions changed over time (Figure 3). There were fewer reports of injection site reactions and more of headache in 2003 compared to 2002. Reports of fever and allergic reaction were less variable over time.

Dose-based AEFI reporting rates

Scheduled vaccines for children aged <7 years

Dose-based AEFI reporting rates are shown in Table 6 for seven funded ASVS vaccines received by children aged <7 years between 1 October 2002 and 31 December 2003. The overall reporting rate increased to 19.8 per 100,000 doses from 14.6 per 100,000 doses for the period January 2000 to September 2002, while rates for 'certain/probable' causality or 'serious' outcomes were more stable (10.0 and 1.2 per 100,000 doses, respectively). The reporting rates for most vaccines did not differ markedly to those estimated for the January 2000 to September 2002 period. The highest AEFI reporting rates and largest changes in reporting rates for individual vaccines were for DTPa vaccine and MenCCV (Table 6).

Dose-based reporting rates of the most commonly reported reactions differed by vaccine type (Figure 5). Injection site reactions following DTPa vaccine were reported at a rate of 47.9 per 100,000 doses of DTPa vaccine, up from 27.9 per 100,000 doses for the January 2000 to September 2002 period.[4] This increase occurred among children aged 1 to <2 years and 2 to <7 years, the ages where a fourth or fifth dose of the vaccine were due (Figure 6). The reporting rates of injection site reactions for the <1 year, 1 to <2 years and 2 to <7 years age groups were 4, 65 and 80 per 100,000 doses of DTPa vaccine, respectively, for the October 2002 to December 2003 period while reporting rates of all other reactions were approximately 18 per 100,000 doses (Figure 6).

Denominator data from the ACIR allowed us to estimate reporting rates of more severe known adverse events for children aged <7 years. The reporting rate

Table 6. Reporting rates of adverse events following immunisation (AEFI) per 100,000 vaccine doses,* children aged less than 7 years, ADRAC database, October 2002 to December 2003

Suspected vaccine type† or AEFI category‡	AEFI records n	Vaccine doses n	Rate per 100,000 doses§	Difference‖
DTPa	564	876,853	64.3	+22.0
DTPa-hepB	106	560,627	18.9	-6.3
Hib	125	572,858	21.8	-1.3
Hib-hebB	36	319,626	11.3	-0.8
Polio	176	1,191,496	14.8	+2.2
MMR	189	605,611	31.2	+4.6
MenCCV	277	715,873	38.7	na
Total‡	958	4,842,917	19.8	+5.2
'Certain' or 'probable' causality rating‡	486	4,842,917	10.0	+3.6
'Serious' outcome‡	60	4,842,917	1.2	0

* Number of vaccine doses recorded on the Australian Childhood Immunisation Register and administered between 1 October 2000 and 31 December 2003.

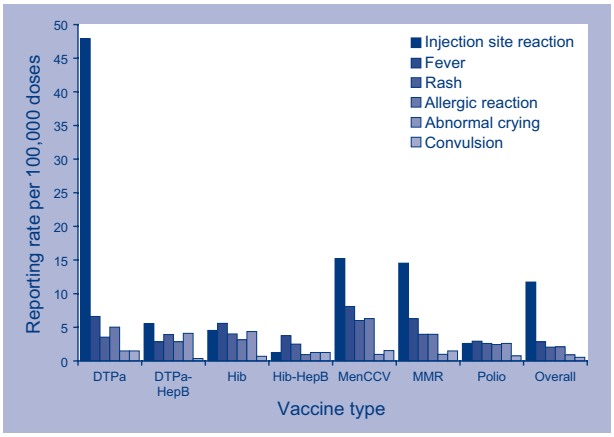
† AEFI records where the vaccine was one of those listed as 'suspected' of involvement in the reported adverse event. See appendix for abbreviations of vaccine names.

‡ AEFI category includes all records (i.e. total), those assigned 'certain' or 'probable' causality ratings, and those defined as 'serious' where at least one of the seven vaccines shown in the table was suspected of involvement in the reported adverse event. Causality ratings were assigned using the criteria described previously.[4] The definition of a 'serious' outcome is described in the Methods section.

§ The estimated rate of adverse events records per 100,000 vaccine doses recorded on the ACIR.

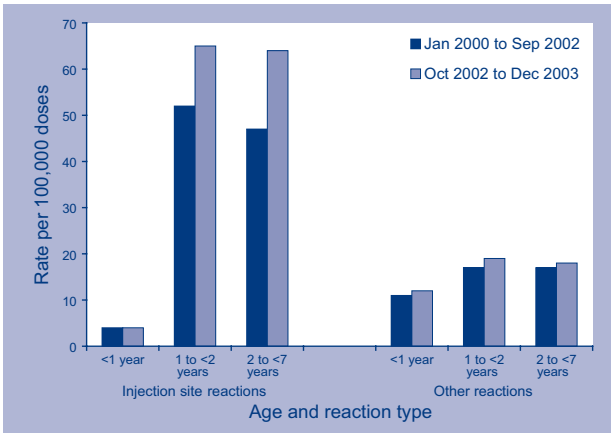
‖ Difference in reporting rate per 100,000 doses for vaccinations administered during October 2002 to December 2003 and January 2000 to September 2002.

Figure 5. Rates of frequently reported reactions per 100,000 vaccine doses administered to children aged <7 years for recommended vaccines, ADRAC database, October 2002 to December 2003



Note: see appendix for abbreviations of vaccine names.

Figure 6 Rates of injection site and other reported reactions per 100,000 vaccine doses of DTPa vaccine, ADRAC database, 2000 to 2003, by age group and year of vaccination



of HHE and convulsion following DTPa vaccination (either alone or in combination with other vaccines) was 1.14 and 1.37 per 100,000 doses respectively. The reporting rate of thrombocytopenia following MMR vaccine was 0.66 per 100,000 doses of MMR vaccine.

Influenza vaccine and adults aged ≥40 years

Influenza vaccine was suspected of involvement in 94 AEFI records for people aged ≥40 years. The dose-based AEFI reporting rates are shown in Table 7. As seen previously,[4] the AEFI reporting rates were higher among influenza vaccinees aged 40–64 years than those aged >65 years (2.8 and 1.6 per 100,000 doses, respectively). The most frequently reported adverse events were injection site reactions, fever and allergic reactions (0.6, 0.3 and 0.3 per 100, 000 doses, respectively). There was one report of Guillain-Barré syndrome following influenza vaccination (Table 4). This corresponds to a reporting rate of <0.03 per 100,000 doses and is unchanged from the previous report.[4]

Meningococcal C conjugate vaccine

A more comprehensive analysis of adverse events following MenCCV will be reported elsewhere. In summary, between licensure of the vaccine in Australia in 2000 and 31 December 2003, a total of 647 AEFI records were entered into the ADRAC database where MenCCV was suspected of involvement in the reported adverse event. The majority of reports for MenCCV were received for children aged 2 to <7 years (n=217, 34%) and those aged 7 to <20 years (n=308, 47%). There were more reports for females than males (male to female ratio of 0.8:1.0).

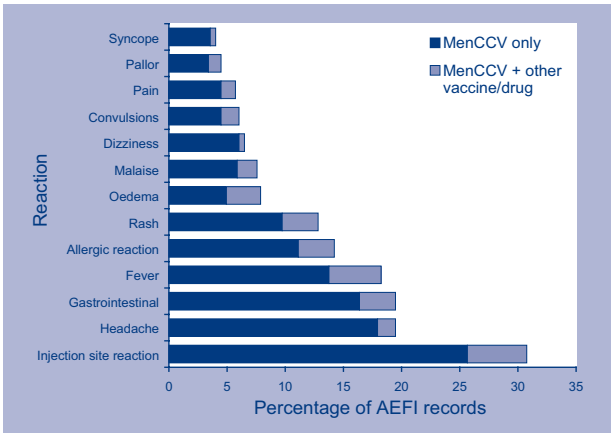
Of the 647 records, MenCCV was the only suspected vaccine in 523 (81%) records, 271 (42%) had causality ratings of 'certain' or 'probable' and 70 (11%) were defined as 'serious'. No deaths were reported. There were a total of three reports of anaphylactic reaction (one involving other vaccines in addition to MenCCV), and 39 (6%) reports of convulsion. The most frequently reported categories of reactions associated with MenCCV administration were injection site reactions (31%), headache (20%), gastrointestinal symptoms (vomiting, nausea, abdominal pain or diarrhoea, 20%), fever (18%) and allergic reactions (14%) (Figure 7).

Approximately 3.5 million doses of MenCCV had been distributed in Australia until 31 December 2003. The reporting rate of anaphylactic reactions was approximately one per one million distributed doses, convulsions at one in 90,000 distributed doses while the overall AEFI reporting rate to ADRAC, across all ages, was one in 5,400 distributed doses.

Discussion

The data presented in this report demonstrate the continued high levels of safety of the most frequently administered vaccines in Australia. In the 15 months to December 2003, over three million doses of influenza vaccine were administered to adults aged ≥40 years while more than 4.8 million

Figure 7. Frequently reported reactions following MenCCV (percentage of 647 records), ADRAC database, 2000 to 2003, by the number of vaccines suspected of involvement in reported adverse event



doses of scheduled vaccines were administered to children aged <7 years. The corresponding AEFI reporting rates were 2.1 per 100,000 doses of influenza vaccine and 19.8 per 100,000 scheduled doses for children <7 years. The majority of AEFIs notified were for injection site reactions and non-serious systemic events. Estimated reporting rates of known potentially life-threatening AEFIs such as HHE and convulsions following DTPa vaccination, and thrombocytopenia following MMR vaccination, were very low and comparable to reporting rates in other passive surveillance systems.[9,13,14]

The number of AEFIs reported to ADRAC has risen each year between 2000 and 2003 while the percentage of serious AEFIs remained stable at 9–10 per cent. The trends in age-specific and vaccine-specific reporting rates demonstrate that the annual increase in overall AEFI reporting rates is mainly due to increased reporting of injection site reactions among children aged 1 to <7 years following a fourth or fifth dose of acellular pertussis-containing vaccines, and to reporting of AEFIs following the introduction of the MenCCV immunisation program for those aged 1–19 years from January 2003. Both of these factors were expected to impact on AEFI reporting rates in 2003.

Extensive injection site swelling is a known adverse event associated with the fourth and fifth doses of acellular pertussis containing vaccines.[4,9,15,16] The higher reporting rates of injection site reactions per 100,000 doses of DTPa vaccine for children aged 1 to <2 and 2 to <7 years for October 2002–December 2003, compared with January 2000–October 2002, was expected as more children became eligible to receive their fourth or fifth consecutive dose of the vaccine in 2003. This birth cohort effect is related to changes in public funding for DTPa across Australia in 1999.[4,17] Rates of injection site reactions should decrease among children aged 1 to <2 years, and to a lesser extent among children aged 2 to <7 years, following the removal of the DTPa dose due at 18 months from the ASVS in September 2003. The impact on the younger age group is already evident with only two reports of injection site reactions following DTPa administered in the fourth quarter of 2003 compared with 33–36 per quarter for the previous quarters of 2003 (data not shown).

Whenever a new vaccine is licensed or added to an immunisation program for a large section of the population, as was the case for MenCCV in 2003, there is an appropriate and expected increase in

Table 7. Dose-based reporting rates of adverse events following immunisation (AEFI) with influenza vaccine,* 40 years and over, ADRAC database, October 2002 to December 2003

Suspected vaccine type† or AEFI category‡	AEFI records n	Rate per 100,000 doses§	Difference‖
Total‡	68	2.1	-0.1
'Certain' or 'probable' causality rating‡	27	0.8	0
'Serious' outcome‡	5	0.2	-0.1

* Number of administered influenza vaccine doses (n=3,286,400) estimated from the 2003 national influenza survey.[11]

† AEFI category includes all records, those assigned 'certain' or 'probable' causality ratings, and those defined as 'serious' where influenza vaccine was suspected of involvement in the reported adverse event. Causality ratings were assigned using the criteria described previously.[4] The definition of a 'serious' outcome is shown in the Methods section.

‡ Number of AEFI records in which influenza vaccine was 'suspected' and the vaccination was administered between 1 September 2002 and 31 December 2003.

§ The estimated reporting rate of adverse events per 100,000 administered doses of influenza vaccine.

‖ Difference in reporting rate per 100,000 doses for vaccinations administered during October 2002 to December 2003 and January 2000 to September 2002

awareness and vigilance about vaccine safety and reporting of AEFIs. Examples of the impact this has on passive AEFI surveillance data include the introduction of varicella vaccine in the United States of America (USA)[18] in the mid-1990s and the MenCCV program in the United Kingdom (UK) in 1999.[19] The profile and rates of adverse events reported to ADRAC following MenCCV are broadly similar to those reported in the UK[19,20] and South Australia[21] with injection site reactions, headaches and gastrointestinal symptoms the most commonly reported reactions. The low reporting rates of serious adverse events show that the MenCCV vaccines used in Australia have high safety levels.

The pattern in AEFI reporting rates for the states and territories for October 2002 to December 2003 is similar to that observed for the period January 2000 to September 2002.[4] The differences in reporting rates between the states and territories reflect differences in populations and, more importantly, different AEFI surveillance practices. As seen in the USA and Canada,[9,22] the less populous jurisdictions (Australian Capital Territory, Northern Territory, South Australia) generally had higher AEFI reporting rates than more populous jurisdictions (New South Wales, Queensland, Victoria).

The impact of different surveillance practices on AEFI reporting rates is highlighted by the observation that Tasmania and Victoria, which have similar reporting requirements, had the lowest population-based AEFI reporting rates (3.9 and 4.5 per 100,000 population, respectively) while the Australian Capital Territory and South Australia were among the highest (23.0 and 16.1 per 100,000 population, respectively), particularly for children aged <7 years (approximately 150 per 100,000 population). Both these jurisdictions have similar surveillance systems where AEFIs are not notifiable but direct parent reporting to the respective state or territory health department is strongly encouraged. While reporting is not mandatory in Tasmania or Victoria, both require AEFIs to be notified directly to the ADRU in Canberra and not to the state health department. In contrast, medical practitioners in New South Wales, Queensland, Western Australia and the Northern Territory are required to notify AEFIs to the local health department with other health professionals, parents and members of the public also encouraged to report AEFIs to the health department. Reporting rates were similar for the three states, both overall and for children aged <7 years (range 5.2–6.4 and 33.1–43.6 per 100,000 population, respectively) (Table 1). The higher AEFI reporting rates in the Northern Territory could be due to a number of factors including local AEFI surveillance practices and a smaller population with different characteristics, compared with other jurisdictions.

Conclusions

The benefits of immunisation in preventing disease continue to significantly outweigh the risks of immunisation-related adverse events for the Australian population. Disease notification data clearly demonstrate the impact of DTPa booster doses on lowering the incidence of pertussis among primary school-aged children[17] while deaths due to *Haemophilus influenzae* type b have declined dramatically following the introduction of Hib vaccination for all children in 1993.[17,23] The most recent example of the benefits of immunisation is the reduction in cases of meningococcal group C disease, particularly in Victoria, following the introduction of the MenCCV program in January 2003.[24] The UK has also seen a significant reduction in meningococcal type C disease among adolescents following the introduction of this vaccine in 1999.[19]

This second report of AEFIs in Australia detected through passive surveillance provides reassurance to immunisation providers, program managers and the public about the safety of vaccines in Australia. The data demonstrate that the system is sufficiently sensitive to detect both expected changes in AEFIs, such as those related to DTPa and MenCCV vaccines, and known rarer and more severe adverse events such as anaphylaxis, HHE, and thrombocytopenia. The regular analysis and reporting of national AEFI surveillance data collated in the ADRAC database is an important aspect of the management of Australia's immunisation programs. Annual AEFI surveillance reports are planned for the future.

Acknowledgements

We thank Brynley Hull for assistance in calculating vaccine doses from ACIR data. The National Centre for Immunisation Research and Surveillance of Vaccine Preventable Diseases is supported by the Australian Government Department of Health and Ageing, the New South Wales Department of Health and The Children's Hospital at Westmead, Australia.

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Appendix

Abbreviations of vaccine types

BCG	Bacille Calmette-Guèrin (i.e. tuberculosis)
dT	diphtheria and tetanus
DTPa	diphtheria-tetanus-pertussis (acellular)
DTPa-hepB	combined diphtheria-tetanus-pertussis (acellular) and hepatitis B
Hep B	hepatitis B
Hib	*Haemophilus influenzae* type b
Hib-hepB	combined *Haemophilus influenzae* type b and hepatitis B
JE	Japanese encephalitis virus
Men4PV	meningococcal polysaccharide tetravalent
MenCCV	meningococcal C conjugate
MMR	measles-mumps-rubella
7vPCV	7-valent pneumococcal conjugate
23vPPV	23-valent pneumococcal polysaccharide
polio	poliomyelitis (oral and inactivated)

Annual report of the Australian National Poliovirus Reference Laboratory, 2003

Kerri Anne Brussen,[1] Vicki Stambos,[2] Bruce R Thorley[3]

Abstract

The Australian National Poliovirus Reference Laboratory was established in late 1994, as part of Australia's commitment to the World Health Organization's (WHO) polio eradication program. The laboratory continues to play a pivotal role in maintaining Australia's polio-free status through surveillance for cases of acute flaccid paralysis (AFP), the main clinical presentation of poliomyelitis, and the testing of specimens from these cases. The annual notification rate for eligible cases of AFP in Australia for 2003 was 0.83 per 100,000 children less than 15 years of age. The annual non-polio AFP rate after classification of cases by the polio expert committee was 0.68 per 100,000, 32 per cent below WHO's annual target. While no polioviruses were isolated from the specimens tested from the 27 cases of AFP in 2003, a novel enterovirus (enterovirus 75) was isolated from one case and enterovirus 71 was isolated from another. During the same period 12 polioviruses, referred from cases other than AFP, tested as Sabin-like by the WHO approved methods of intratypic differentiation. The importation of wild polioviruses from endemic Nigeria into surrounding countries of Africa during 2003, highlights the importance of the continuation of AFP surveillance and high quality laboratory activities throughout the world until global eradication of polio is certified. *Commun Dis Intell* 2004;28:339–344.

Keywords: poliovirus, acute flaccid paralysis, surveillance, enterovirus

Introduction

In May 1988, the World Health Assembly adopted a resolution for the global eradication of poliomyelitis. The World Health Organization (WHO) implemented a program to achieve this goal through high levels of polio vaccination coverage, predominantly with the Sabin oral polio vaccine (OPV). In addition, the program initiated surveillance for cases of acute flaccid paralysis (AFP), the most commonly observed clinical manifestation of poliovirus infection, and the establishment of a global laboratory network accredited for the testing of specimens from AFP cases.[1]

The Australian National Polio Reference Laboratory (ANPRL) was established in late 1994 at the Victorian Infectious Diseases Reference Laboratory (VIDRL), as part of Australia's commitment to the WHO polio eradication program. The laboratory also serves as the national polio laboratory for the Pacific Island countries and as a regional polio reference laboratory for the WHO Western Pacific Region. Surveillance for AFP in Australia was initiated in March 1995 by the then Federal Government Department of Human Services and Health. The surveillance program has been co-ordinated at VIDRL since 2000, in collaboration with the Australian Paediatric Surveillance Unit (APSU).

Poliomyelitis is a notifiable disease in all states of Australia while AFP is also a notifiable disease in Queensland.[2] In a country that is not endemic for polio, such as Australia, the WHO indicator target for AFP cases for children aged less than 15 years, is one case per 100,000 population. For Australia, this represents 40 AFP cases per year. The WHO target for laboratory testing is that at least 80 per cent of notified AFP cases have adequate stool specimens collected (two specimens at least 24 hours apart and within 14 days of onset of paralysis) and tested in a WHO accredited laboratory.

Australia's childhood immunisation schedule includes four doses of the live, attenuated OPV. Doses are recommended at 2, 4 and 6 months of age with a booster prior to school entry.[3] OPV is a trivalent vaccine comprising all three poliovirus serotypes. After

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administration of the vaccine, the viruses multiply in the gut of the recipient and can be excreted for up to six weeks from immunocompetent individuals.[4] Longer excretion from immunocompromised recipients has been documented.[5,6] Therefore, poliovirus can be isolated from stool specimens from individuals with clinical symptoms other than AFP during routine laboratory testing. These viruses should be subjected to further testing to confirm their vaccine origin and hence considered an incidental finding. A further boost with polio vaccine is recommended for people travelling in the remaining polio endemic regions of Africa, the Eastern Mediterranean and South Asia.[3]

The activities of the ANPRL in 2003 are summarised in this annual report which includes a comparison of AFP surveillance in Australia against the major targets nominated by WHO.

Methods

AFP surveillance is co-ordinated by the ANPRL in collaboration with the APSU. Briefly, any doctor in Australia seeing a patient under 15 years of age presenting with AFP or a person of any age suspected of an acute poliomyelitis infection is requested to telephone the AFP co-ordinator at VIDRL to notify the case. The doctor is requested to collect two stool specimens, 24 hours apart and within 14 days of onset of paralysis, and forward them to the ANPRL for testing. Paediatricians also notify cases of AFP through a monthly reporting scheme to the APSU. The clinicians who notify a case of AFP are requested to complete a questionnaire, which is reviewed, in conjunction with laboratory results, by the Polio Expert Committee (PEC). Cases are classified by the committee as either (i) non-polio AFP, (ii) poliomyelitis due to wild poliovirus, vaccine-derived poliovirus (VDPV) or vaccine-associated paralytic poliomyelitis, or (iii) non-AFP.

Laboratory methods were described in detail in the 2001 ANPRL annual report.[7] In brief, stool specimens received from AFP cases are extracted with a 10% v/v chloroform solution and inoculated into WHO approved cell lines. This includes the L20B cell line, a genetically modified mouse cell line that expresses the human poliovirus receptor. All polioviruses, whether from AFP cases or other sources, are tested by enzyme-linked immunosorbent assay (ELISA) and genetic methods, (PCR), for the differentiation between wild and the OPV strains of poliovirus. The poliovirus ELISA is sensitive to antigenic drift due to mutations accumulated during viral replication. The VP1 gene is sequenced for any poliovirus with discordant results by the two methods of intratypic differentiation; that is, Sabin-like by PCR but non-Sabin-like or double reactive (equal avidity with both Sabin and non-Sabin antiserum) by ELISA. The poliovirus is reported as (i) a wild type poliovirus, (ii) Sabin vaccine-like or (iii) a vaccine derived poliovirus (VDPV) if there is more than one per cent variation in the VP1 gene sequence compared to the parental Sabin strain.

Results

Acute flaccid paralysis surveillance

According to the WHO criteria, eligible cases are patients who are Australian residents and aged less than 15 years on the date of paralysis onset. However, the PEC will review cases of suspected poliomyelitis in people of any age. Forty-four notifications of AFP in Australia were received in 2003. Duplicate notifications were received for eight cases. Thirty-three cases of AFP were from patients less than 15 years of age and three cases were patients 15 years or older. Of the 33 eligible cases, the PEC classified 27 as non-polio AFP, two cases are pending review and classification and a further four cases require more information from the referring doctor before a final classification can be made. Thus, the annual AFP notification rate for eligible cases in Australia in 2003 was 0.83 per 100,000 children less than 15 years of age, 17 per cent below the WHO target of 1.0 per 100,000 (Table 1). The annual non-polio AFP rate in 2003, after classification of cases by the PEC, was 0.68 per 100,000 population, 32 per cent below the WHO target. Twelve of the 27 cases classified as non-polio AFP by the PEC were diagnosed as Guillian-Barré Syndrome.

Laboratory testing of specimens from acute flaccid paralysis cases

The ANPRL tested 35 stool specimens and one virus isolate from 19 cases of AFP in children aged less than 15 years, in 2003. This included one case with onset of symptoms in late 2002. A further two stool specimens were referred from one case of AFP in a person greater than 15 years. The WHO criteria for adequate stool sampling of AFP cases is two specimens collected at least 24 hours apart and within 14 days of onset of paralysis in at least 80 per cent of eligible cases. During 2003, 24 per cent of eligible AFP cases had adequate stool specimens (Table 1). No polioviruses were isolated from any specimens tested from AFP cases during 2003 (Table 2).

Enterovirus 71 was isolated from two stools from one AFP case. A portion of the VP1 gene was sequenced and a search of the GenBank database with the BLAST software indicated the greatest homology to strains of genogroup C1 isolated in Malaysia in the late 1990s.

Table 1. Acute flaccid paralysis surveillance in Australia, compared with World Health Organization indicator targets for children less than 15 years, 2003

WHO* indicator target for AFP† cases of children less than 15 years	Australia's surveillance for AFP cases with onset in 2003	Australia's AFP surveillance rates for 2003
Non-polio AFP case rate of 1 per 100,000 population (40 cases for Australia in 2003)	33 cases of AFP notified	AFP notification rate: 0.83 per 100,000 population
	27 cases classified by the PEC as non-polio AFP‡	Non-polio AFP case rate: 0.68 per 100,000 population
More than 80% of notified AFP cases with 2 adequate stool specimens collected at least 24 hours apart within 14 days of onset of paralysis	8 AFP cases with 2 or more specimens per case	Referral of adequate specimens from AFP cases: 24% of case notifications (8/33)

* WHO World Health Organization.

† AFP acute flaccid paralysis.

‡ A further two cases are pending review by the Polio Expert Committee (PEC) and four cases require further information from the referring doctor before final classification.

Table 2. Testing of specimens and isolates referred to the Australian National Poliovirus Reference Laboratory, 2003

Result	Results from acute flaccid paralysis cases		Isolations from referred samples	Total
	< 15 years	≥ 15 years		
Poliovirus Sabin-like type 1	–	–	3	3
Poliovirus Sabin-like type 1 & 2	–	–	2	2
Poliovirus Sabin-like type 1 & 3	–	–	1	1
Poliovirus Sabin-like type 2	–	–	3	3
Non-polio enterovirus *	4	–	11	15
No virus isolated	32	2	13†	47
Total	36	2	33	71

* Enterovirus type 71 was isolated from two stool specimens of one AFP case and enterovirus type 75 was isolated from a stool specimen and an isolate of another case. Testing of the referred samples identified two echovirus type 25, two each of coxsackievirus types B3 and B4 and single isolations of coxsackievirus types B2 and B5. The specific identification of three isolates is pending.

† Includes specimens tested for poliovirus as part of a differential diagnosis and also for ongoing shedding from a recently immunised patient with an immune deficiency. No virus may have been isolated from the remaining referred isolates due to loss of titre in transit and/or not passaging between different cell lines.

A non-polio enterovirus was identified from the stool of another case of AFP. A comparison of the VP1 gene sequence to the GenBank database determined no significant homology to the known enterovirus prototypes. The sequence was referred to collaborators at the Centers for Disease Control and Prevention, United States of America, who reported the virus to be enterovirus type 75, a newly described enterovirus. (Dr M Steven Oberste, Research Microbiologist, Respiratory and Enteric Viruses Branch, Centres for Disease Control and Prevention, Atlanta, United States of America, personal communication).

Specimens and isolates referred for isolation and identification

Thirty-three specimens and isolates were received by the ANPRL from sources other than AFP during 2003. Six poliovirus type 1, five poliovirus type 2 and one type 3 poliovirus were isolated from nine samples (Table 2). A mixture of two different serotypes was identified from three of the samples. All 12 polioviruses isolated tested as Sabin vaccine-like by the WHO approved methods for intratypic differentiation.

Amongst the referred samples from sources other than AFP cases (Table 2), three stool specimens were from a child immunised with OPV, who was subsequently diagnosed with a T-cell deficiency. The specimens were tested to determine if ongoing shedding of the OPV virus strains was occurring, but no virus was isolated. Poliomyelitis was considered as part of the differential diagnosis of another child with ataxia. Two stool specimens were tested but no virus was isolated from either specimen.

Laboratories within Australia are encouraged to forward their untyped enteroviruses to ANPRL for identification. If they are confirmed as poliovirus they are tested by the WHO approved methods of intratypic differentiation to characterise the isolate. Of the 33 specimens and isolates tested, 18 were untyped enteroviruses from a laboratory in South Australia. Five polioviruses were identified amongst these viruses and all tested as Sabin vaccine-like.

Eight non-polio enteroviruses were identified in 2003, originating from Victoria, South Australia and Queensland. (Table 2). Two echovirus type 25, two coxsackievirus B3, two coxsackievirus B4 and single isolates of coxsackievirus B2 and B5 were identified by sequence homology by comparison of a portion of the VP1 gene to the GenBank database and the serotype was confirmed with monospecific antiserum. A further three non-poliovirus enteroviruses are yet to be identified.

A summary of enterovirus testing of specimens and isolates referred to the ANPRL from within Australia between 1995 and 2003 are presented in Table 3.

Poliovirus serology

Serum specimens were referred for poliovirus serology from two patients. One patient had an encephalomyelitis of unknown aetiology while the second patient had paraplegia. Results from both patients were inconclusive as no acute serum was available for either patient.

Regional reference laboratory activities

Three hundred and fifty specimens and isolates were referred to the ANPRL from countries of the Western Pacific Region in the laboratory's role as a WHO regional reference laboratory. As part of an ongoing laboratory quality assurance program, the laboratory received 146 specimens and isolates from the National Poliovirus Laboratory of Papua New Guinea and 120 from the National Laboratory of Viet Nam, Ho Chi Minh City. In additional roles as a National Polio Reference Laboratory, four specimens were received from two cases of AFP in Brunei Darussalam and 22 specimens from 11 cases of AFP were received from the Pacific Island countries. Echovirus type 6 was isolated from two cases of AFP in Fiji. Specimens and isolates were also referred from Malaysia, Mongolia, and the Philippines for further identification and characterisation.

A poliovirus type 1 isolated during a survey of healthy children to supplement AFP surveillance in Mongolia, was referred to ANPRL. The intratypic differentiation test result was discordant: Sabin-like by PCR and non-Sabin-like by ELISA. The complete VP1 gene sequence was confirmed by the WHO Global Specialized Laboratory at the National Institute of

Table 3. Summary of enterovirus testing at the Australian National Poliovirus Reference Laboratory, 1995 to 2003

Year	Poliovirus		Non-polio enterovirus	Non-enterovirus detected or no virus detected	Total samples tested
	Sabin-like	Non-Sabin-like*			
1995	190		200	13	403
1996	224		198	9	431
1997	124		76	0	200
1998	52		15	4	71
1999*	60	1	9	9	79
2000	45		44	47	136
2001*	46	5	33	75	159
2002†	36		21	49	106
2003	9		15	47	71

* Untyped enterovirus or uncharacterised poliovirus isolates were referred for further testing after completion of a laboratory inventory. Some of the isolates tested as non-Sabin-like and were subsequently identified as wild type poliovirus prototype strains and were destroyed.

† Two poliovirus isolates from non-AFP sources had discordant results by intra-typic differentiation. Sequencing confirmed the isolates as Sabin-like, with <1.0 per cent variation from the parental Sabin strain.

infectious Disease, Tokyo, to have 98.7 per cent homology to Sabin type 1 prototype sequence, thus classifying the virus as a VDPV (greater than 1% variation from Sabin VP1 prototype sequence). The child was later determined to have a previously undiagnosed immune deficiency and extensive surveillance activities found no evidence of circulation of the VDPV within the population.

Discussion

Australia has continued to struggle to meet the WHO standard criteria for AFP surveillance in a non-polio endemic country, of detecting at least one case of AFP per 100,000 children aged less than 15 years. Since the introduction of AFP surveillance in 1995, the target was met only in 2000 (1.15) and 2001 (1.13).[7] However, this dropped to 0.75 cases per 100,000 population less than 15 years of age in 2002[8] and to 0.68 in 2003. The rate of stool sampling in 2003 was 24 per cent, well below the WHO target of 80 per cent of AFP cases in children less than 15 years. When considering the notifications involving patients of all ages, 17 of the 36 AFP cases (47%) had at least one stool specimen collected in 2003. The 2003 rate for specimen collection was similar to that reported between 1995 and 1999 and in 2002,[7,8] while stool collection rates rose to 31 per cent and 36 per cent in 2000 and 2001, respectively.

A capture-recapture study of AFP cases determined that the incidence of AFP in Victoria was consistently under reported.[9] A more recent study was undertaken with a grant from the Australian Government Department of Health and Ageing to investigate differences in AFP notification rates by state. The study evaluated the disparity in the AFP notification rates from one state to another and concluded that the engagement of the state based departments of health to promote and monitor AFP as a public health issue, may assist in increasing Australia's AFP notification rate.[10]

The two poliovirus serology results in this report highlight the need for adequate sampling (acute and convalescent serum) for a definitive conclusion to be made from the polio antibody test. The test cannot differentiate between an immune response from recent exposure to wild poliovirus, vaccine-derived poliovirus or OPV. If an acute polio infection is suspected, stool specimens remain the specimen of choice.

The development of molecular techniques for the identification of enteroviurses has led to the description of previously untypeable enteroviruses. As yet, no antisera are available to these new enteroviruses and their identification is based on genotyping rather than the traditional method of serotyping.[11,12] The identification of enterovirus type 75 from an Australian AFP case was based on genotyping of the virus and represents, to our knowledge, the first documentation of this virus in Australia. Enterovirus type 75 has been isolated from patients with a variety of illnesses including AFP.[13]

The increase in severity of disease and outbreaks associated with enterovirus type 71 in recent years within the Asia-Pacific region, suggests the need for the specific identification and monitoring of enterovirus isolations within Australia.[14,15] The enterovirus type 71 isolated from one case of AFP in Australia in 2003, was closely related to viruses of genogroup C1 detected in the Malaysian peninsular during 1997–2000.[14] Enterovirus type 71 of genogroup C1 was determined to be responsible for the outbreak of hand, foot and mouth disease in Malaysia in 2000, and has been suggested to have the potential to cause future epidemics within the Asia-Pacific region.[15]

The number of untyped enteroviruses referred from within Australia to the ANPRL for further identification and characterisation has dwindled since 1995. The increasing use of PCR directly on extracted specimens in many laboratories has possibly decreased the number of virus isolates available for further testing. The isolation of enterovirus types 71 and 75 described in this report and the detection of polioviruses with discordant intratypic differentiation results from sources other than AFP in 2002,[8] exemplify the need for the specific identification of enteroviruses.[16]

During 2003, of the 784 wild polioviruses isolated globally, 51 were detected in eight African countries previously considered polio-free.[17] Immunisation coverage against polio is believed to have dropped recently in these countries.[17,18] The polioviruses were determined to have been imported from one of the largest remaining areas endemic for polio that spans northern Nigeria and southern Niger. For Australia to retain its polio-free status, it must maintain a high level of polio vaccination coverage until global eradication has been certified, as well as supporting the continuation of surveillance for AFP cases and laboratory testing of specimens.

Acknowledgements

We would like to acknowledge Ann Turnbull, Pauline Cardwell, Aishah Ibrahim and David Tyssen for excellent technical assistance. We thank the Australian Paediatric Surveillance Unit for their ongoing collaboration with AFP surveillance, the Polio Expert Committee for the review of AFP cases and Dr Heath Kelly as the chief investigator of AFP surveillance. We would also like to thank all notifying clinicians and laboratories for forwarding the specimens and isolates during 2003. The Australian National Poliovirus Reference Laboratory is funded by the Australian Government Department of Health and Ageing and the Victorian Department of Human Services.

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An outbreak of meningococcal disease in a secondary school — implications for public health practice

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Abstract

This report describes briefly the management of three cases of meningococcal disease which all occurred within one week at a secondary school on the Central Coast of New South Wales in late winter 2003. The Central Coast health area has a population of approximately 300,000. Between 10 and 15 cases of meningococcal disease are notified to the Central Coast Public Health Unit each year. The three cases all presented to Gosford Hospital, Cases 1 and 2, both in Year 9, on Thursday 14 August 2003 and Case 3 in Year 8 on Friday 15 August 2003. *Commun Dis Intell* 2004; 28:345–347.

Keywords: disease outbreak, meningoccocal, neisseria meningitidis

Cases 1 and 2

On admission, Case 1 was diagnosed with meningococcal disease, intubated and admitted to the Intensive Care Unit (ICU). The initial diagnosis for Case 2 was gastroenteritis but this was changed to meningococcal infection after about six hours and the patient was also admitted to the ICU. In both cases prophylaxis (rifampicin) was given to family members and close contacts. On Thursday, after discussion with several experts, it was decided to offer prophylaxis (rifampicin or ciprofloxacin) to all 220 Year 9 students on the following day.

A response team spent part of Thursday evening and Friday morning preparing a response strategy specific to the given conditions. The main concern was that the team had a very short window of opportunity, limited to Friday only, and during school hours. Since the decision to offer prophylaxis was made after school hours on Thursday, the usual practice of sending consent forms home with the students could not occur. Therefore part of Friday morning was spent telephoning parents of all Year 9 students to obtain consent for the prophylaxis, and thus further limiting time available with the students.

Before lunch on Friday, the Director (Medical Officer of Health) of the Central Coast Public Health Unit (PHU) spoke to all Year 9 students in a single group. He briefly outlined our understanding of meningococcal disease, how it is thought to be spread, the current situation with two Year 9 students in hospital, and the rationale for recommending antibiotic prophylaxis for students in Year 9. He had parental consent to give a progress report on the two cases, both of whom were stable. The risk of further cases occurring was described, and it was emphasised that family and household contacts of the cases had a much greater risk than Year 9 students, who in turn had a higher risk than the general community. Students were mainly concerned about possible side effects of the medication and these were explained. The procedure for prescribing the antibiotics was also explained. This session was delivered in an informal style with questions answered as they arose, and lasted about 40 minutes.

Medication of students on Friday could only begin at about 1400 hours and was scheduled to stop at approximately 1500 hours to allow many of the students to catch buses home. More delays occurred during the dispensing process when team staff were asked about rifampicin interactions with

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methylphenidate (ritalin), a drug not specifically mentioned in the national guidelines.1 These circumstances created some pressure on the team to maintain a steady throughput of students. However, with arrangements made to delay bus departures briefly, 202 of 220 students were able to receive prophylaxis.

The PHU Director returned to the school on the following Monday to dispense chemoprophylaxis to the remaining students who were absent on Friday. Hence 215 out of 220 students received medication. Of the remaining five, three were the cases, one had left school and one was in the process of leaving and declined treatment.

Meningococcal disease type C was confirmed by polymerase chain reaction (PCR) during the week following initial diagnosis.

Case 3

On presentation, Case 3 was also diagnosed with gastroenteritis. Because this patient was a student at the same school as Cases 1 and 2, and presented soon after they did, blood was taken to exclude meningococcal disease and intramuscular penicillin was given. This case was discharged from the Emergency Department on 16 August 2003. When meningococcal infection was confirmed by PCR on 19 August, Case 3 was recalled for ambulatory intravenous antibiotics and prophylaxis was given to family members and close contacts.

Confirmation of a third case of meningoccal infection prompted a decision to offer meningococcal C vaccine to all students at the high school. This decision simply accelerated the National Meningococcal C Vaccination Program scheduled for years 10, 11 and 12 at the school and expanded it to years 7, 8 and 9. All three cases recovered completely and no new cases have been reported following the completion of the vaccination program.

Implications for Public Health Practice

A review of the outbreak by staff of the PHU suggested that local adaptations, specific for a given situation, are needed to expand the national guidelines.[1] Such adaptations could refer to some or all of the following response processes.

PCR diagnosis

Case 3 was sent home with a provisional diagnosis of gastroenteritis. Without PCR testing for meningococcal disease, Case 3 would not have been identified. PCR tests to detect meningococcal DNA have high sensitivity and specificity. They are being used in clinical situations where meningococcal disease is suspected and blood or other cultures are negative. They are also being considered in patients with less obvious symptoms, particularly when the disease is present in the local community. This does raise some questions. Do protocols need revision in light of our increased ability to detect milder disease? Will greater recognition of milder disease escalate the public health response? This would seem likely. Will there be a better health outcome? It will be important to monitor the impact of PCR testing and the subsequent public health response.

Time constraints

Our experience indicates that it is important to determine how long the target group will be available to the response team. In this outbreak, the students were available only during school hours, excluding lunchtime. In preschools this time could be significantly shorter. Getting information and consent forms home to parents the day before the medication is to be dispensed can save hours spent telephoning parents on the clinic day.

Staffing needs

While a response team can usually be assembled easily, the response could have been streamlined by appointing an event coordinator, preferably a senior health professional with emergency management experience. In addition, sufficient staff should remain at the office to ensure other public health needs are adequately met. A person whose sole role is managing media inquiries is also desirable.

Medication needs

Arrangements need to be in place to provide adequate quantities of medication at short notice. Notifying the pharmacy early to the possibility of a mass prophylaxis event, even before a decision is made, has proved to be useful in our experience. There was no delay in providing medication in this outbreak due to the immediate and willing cooperation by pharmacy staff. However, one of the authors (TM) has experienced delay in the provision of medication while managing other outbreaks.

Fact finding and information sharing

Our experience has emphasised the need for the local public health unit to regularly share information with all stakeholders throughout the investigation. This improves cooperation and streamlines the investigation.

Reducing concern in the affected population

It is important to minimise concern in the affected population by appropriate and timely information. While the time taken to telephone parents was considerable, it did allow an opportunity to respond to any questions or concerns.

Conclusion

The national guidelines[1] state that 'A structured review should always be undertaken of each outbreak and its management with a view to improving performance' (p. 31). As the practical realisation of such advice, this report indicates that recorded experience in the management of an outbreak of meningococcal disease is likely to pay dividends in the management of future outbreaks.

Acknowledgements

We wish to thank the high school staff, students and parents; the Central Coast Health staff; and NSW Health staff.

References

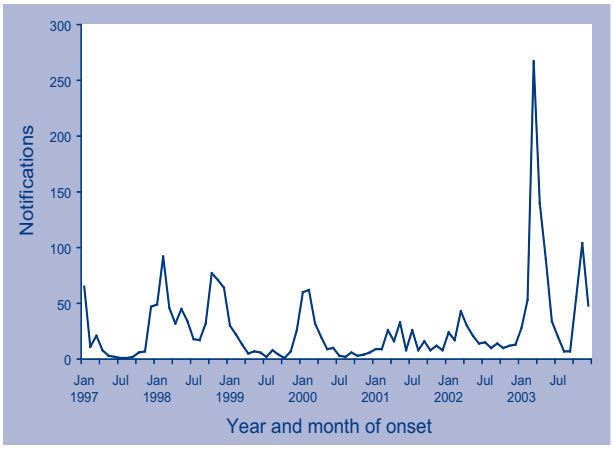
1. Communicable Diseases Network Australia. Guidelines for the Early Clinical and Public Health Management of Meningococcal Disease in Australia. Commonwealth Department of Health and Aged Care, Canberra: June 2001.

Errata

Communicable diseases surveillance — Highlights for 4th quarter, 2003

Figure 2, published in *Commun Dis Intell* 2004;28:104, showing dengue notifications in Australia 1997 to 2003, was incorrect. The correct data is shown below. We apologise to our readers for any confusion that resulted from publication of this erroneous data.

Figure 2. Notifications of dengue Australia, 1997 to 2003, by month of onset



Invasive pneumococcal disease in Australia, 2002

In December 2003, the report 'Invasive pneumococcal disease in Australia, 2002' (*Commun Dis Intell* 2004;27:466–477) recorded ten deaths from invasive pneumococcal disease (IPD) in children under 5 years of age in 2002 (Table 7, p472). The Department of Health and Ageing has reviewed these ten deaths with the states and territories and found that one of these deaths was incorrectly classified as occurring in a child, when the person was aged more than 5 years of age at the time of death. The correct figure for the number of deaths from IPD in children under 5 years of age in 2002 is therefore nine.

Errata *continued next page*

Age-related risk of adverse events following yellow fever vaccination in Australia

Tables 1 and 2, published in *Commun Dis Intell* 2004;28:244, showing reporting rates for adverse events following yellow fever vaccination, are reproduced with correct headings and footnotes.

Table 1. Reporting rates per 100,000 vaccine doses and reporting rate ratios for adverse events following yellow fever vaccination, Australia, 1993 to 2002

Age (years)	Number of doses	% of doses	Systemic adverse events*				Serious systemic adverse events*			
			n	Rate†	RRR‡	(95% CI)§	n	Rate†	RRR‡	(95% CI)§
15–24	32,423	15.4	0	0.00	0.00	–	0	0.00	0.00	–
25–44	120,552	57.2	14	11.61	1.00‖	–	3	2.49	1.00‖	–
45–64	48,697	23.1	9	18.48	1.59	(0.69–3.68)	4	8.21	3.30	(0.62–9.90)
≥65	8,984	4.3	3	33.39	2.88	(0.83–10.0)	2	22.26	8.95	(1.49–53.5)
15–44	152,975	72.6	14	9.15	1.00‖	–	3	1.96	1.00‖	–
≥45	57,681	27.4	12	20.80	2.27	(1.05–4.91)	6	10.40	5.30	(1.33–21.2)
Total	210,656	100.0	26	12.34			9	4.27		

* See Box for definitions of adverse event categories; 'serious' adverse events were those leading to hospitalisation or death.

† Rate per 100,000 doses of vaccine.

‡ Reporting rate ratio.

§ Confidence interval.

‖ Reference age group for comparison.

Table 2. Reporting rates per 100,000 doses and reporting rate ratios for adverse events following yellow fever vaccination, United States of America, 1990 to 1998[5]

Age (years)	Number of doses	% of doses	Systemic adverse events*				Serious systemic adverse events*			
			n	Rate†	RRR‡	(95% CI)§	n	Rate†	RRR‡	(95% CI)§
15–24	189,991	13.2	3	1.58	1.01	(0.28–3.6)	2	1.05	3.70	(0.52–26)
25–44	702,783	48.7	11	1.57	1.00‖	–	2	0.28	1.00‖	–
45–64	442,605	30.7	12	2.71	1.73	(0.76–3.9)	5	1.13	3.97	(0.77–20)
≥65**	108,307	7.5	9	8.31	5.31	(2.2–12.8)	5	4.62	16.2	(3.2–84)
15–44**	892,774	61.8	14	1.57	1.00‖	–	4	0.45	1.00‖	–
≥45**	550,912	38.2	21	3.81	2.43	(1.2–4.8)	10	1.82	4.04	(1.3–12.9)
Total	1,443,686	100.0	35	2.42			14	0.97		

* See Box for definitions of adverse event categories; 'serious' adverse events were those leading to hospitalisation or death.

† Rate per 100,000 doses of vaccine.

‡ Reporting rate ratio.

§ Confidence interval.

‖ Reference age group for comparison.

** Reporting rates and reporting rate ratio values for these age groups were calculated from published data.[5]

Festschrift for Professor Margaret Burgess AO

Compiled by: Julia ML Brotherton on behalf of the staff of the National Centre for Immunisation Research and Surveillance of Vaccine Preventable Diseases and the presenting speakers.

Abstract

In honour of the retirement of our director Margaret Burgess, National Centre for Immunisation Research and Surveillance (NCIRS) held a Festschrift on 5th to 6th February 2004. The themes of the event were Vaccines for the 21st Century and Congenital and Neonatal Infections. International guests attended the Festschrift, as well as over 180 colleagues and co-workers from across Australia. A summary of the presentations over these two fascinating days is provided herein. *Commun Dis Intell* 2004;28:349–355.

Day One Presentations

Session 1: The Children's Hospital at Westmead Grand Rounds
Chair: Professor Kim Oates

Congenital rubella in Australia 1941 to 2004: Professor Margaret Burgess

Margaret described the important role researchers in Australia, and especially those from the Royal Alexandra Hospital for Children, have played in the story of rubella in pregnancy. In 1941, Norman Gregg, an ophthalmic surgeon at the hospital, was the first to report an association between children born with cataracts and a history of rubella in the mother during the early part of the pregnancy. Later, Australian researchers Swan (1943) and Lancaster (1951) reported that deafness in children was associated with rubella infection during pregnancy. The long-term outlook of children with congenital rubella has also been explored by Australian researchers in three reviews (in 1966, 1991 and 2001) of Gregg's patients born in 1941. The 50 patients were found to have an increased risk of diabetes, thyroid disorders and early menopause compared with the Australian population of the same age, and several had glaucoma and hypertension. Despite this, they were well adjusted socially.

Margaret also highlighted the impact of vaccination in Australia. Rubella vaccine was licensed in Australia in 1970 and was first used in a targeted school-girl vaccination program. Now the vaccine is given to children at one and four years of age and immunity is high in children and women of child-bearing age. However there remains a cohort of young adult males who are still susceptible and Asian born pregnant women presently have a 5–10 times higher risk of being seronegative compared with Australian born women. Vaccination has led to a remarkable reduction in rubella, but two cases of congenital rubella in 2003 (the first locally acquired cases since 1997) remind us that we need to remain vigilant.

Rubella — the global picture: Professor Felicity Cutts

Professor Cutts began her presentation by discussing the global burden of rubella and congenital rubella syndrome (CRS). In 2001, 123 countries (57%) performed surveillance for rubella and 51 (24%) had a surveillance system in place for CRS. However surveillance, especially for CRS and during non-outbreak periods, is very insensitive, so serosurveys have been performed in some countries to obtain better estimates of the burden. Estimates of the prevalence of CRS from these serosurveys range from 0.7 (in parts of Europe) to 1.75 (in parts of the Americas) per 1,000 live births. The serosurveys also provided estimates of the level of vaccination coverage required in each country to achieve herd immunity (e.g. 85–91% in Ethiopia).

Rubella vaccination has been reported to be cost-effective in Latin American and Caribbean countries, as well as in developed countries. However, Professor Cutts cautioned that this does not mean infant vaccination should be introduced in all countries. Infant vaccination shifts the average age of infection upwards and may increase the risk of infection in women of child-bearing age (and the risk of CRS) so it should only be introduced if sufficient coverage can be achieved. The required level of coverage depends on the intensity of rubella transmission and is about 80 per cent in developed

* Full paper is available from the National Centre for Immunisation Research and Surveillance, The Children's Hospital at Westmead, Locked Bag 4001, Westmead NSW 2145.

countries. If the required level can not be reached with an infant program, then selective vaccination of adolescent and adult females will also reduce the risk of CRS, but will take longer to have an impact and will not affect the transmission of rubella. For rapid elimination of CRS, routine infant vaccination is supplemented by vaccination of susceptible adult women (e.g. post-partum vaccination). To eliminate rubella transmission, most industrialised countries now offer a second opportunity for rubella vaccination to school-aged children, while Latin America is pioneering the use of large-scale mass vaccination campaigns of adult men and women. For example, an outbreak of rubella that resulted in 46 documented cases of CRS (0.5/1,000 live births) occurred in Costa Rica in 2000, after almost 30 years of routine infant rubella vaccination. Following the mass campaign in which 95 per cent of men and women aged 15–39 years were vaccinated, the last case of rubella was reported in August 2001.

Vaccines in the 21st Century: Professor Stanley Plotkin

Emeritus Professor Stanley Plotkin reflected on the history of vaccination and outlined his perception of six revolutions in vaccine development. The first revolution described was the development of attenuated vaccines made in the laboratory: animal viruses as vaccines (vaccinia), physical attenuation (rabies, anthrax), and passage in animals or *in vitro* (yellow fever, bacille Calmette-Guerin.) The second revolution was the discovery that inactivated organisms or sub-units of organisms could function as vaccines, leading to vaccines based upon polysaccharide capsules, proteins or toxoids. After World War II, the development of vaccines through the passage of organisms in cell culture *in vitro*, produced the third revolution. The fourth revolution was the development of molecular biology, which has resulted in a new array of strategies for vaccine development starting from information on the microbial genome (DNA, cDNA, or RNA.) Examples of such strategies are recombinant protein production, prime boost using DNA and/or vectors and reverse genetics. The fifth revolution has been the development of methods to induce cell mediated immune responses, such as through the use of live microbes, live vectors, alphavirus replicons, DNA, lipopeptides and Th1 adjuvants. Professor Plotkin believes that the sixth revolution in vaccination will relate to the development of new strategies in vaccine delivery. Non-parenteral methods of vaccination being investigated include intranasal (influenza), aerosol (measles/rubella), transcutaneous, oral (e.g. transgenic plants) and rectal (sexually transmitted infections).

Professor Plotkin also provided updates on several vaccines in advanced states of development including live influenza, rotavirus, measles-mumps-rubella-varicella, meningococcal conjugate against groups A/C/W-135/Y, meningococcal group B outer membrane protein and human papillomavirus vaccines. Particular challenges and trends in vaccination in 2004 described by Professor Plotkin included the need for new combination paediatric and adult vaccines, the rise of adolescent vaccines, and in new vaccination targets such as for hospitalised patients, for pregnant women, against bioterrorism threats and against chronic infections. The main threats to vaccination in the 21st Century were identified as cost, supply, safety and anti-vaccinationism. Professor Plotkin reflected that we are in the golden age of vaccine development, in which it is feasible to produce any antigen we want for use in a vaccine, but that we currently lack sufficient knowledge of pathogenesis and immunology to choose the best antigens and methods of vaccination.

Session Two: The Australian Contribution to Vaccine Research
Chair: Professor Sir Gustav Nossal

Historical background: Professor Sir Gustav Nossal

Professor Sir Gustav Nossal outlined the history of vaccine development in Australia from World War I to the present. Over that period, a number of scientists working at notable institutions, such as the Commonwealth Serum Laboratories (CSL) and the Walter and Eliza Hall Institute (WEHI), made important achievements in vaccine technology and delivery. Early collaborations between CSL and WEHI, under their respective directors Penfold and Kellaway, resulted in Australia-wide availability of passive immunisation for snake and spider bites through the provision of antivenene. A strong collaboration on influenza work saw the popularisation and improvement of Goodpasture's technique for growing viruses in hen's eggs. In the middle of last century the Commonwealth Serum Laboratories were in charge of vaccine production. This included providing Salk vaccine (inactivated polio vaccine) for the nation and, working with Burnett during World War I, live attenuated intranasal influenza vaccine was given to 20,000 army recruits. Another major contribution of CSL was providing antiD for the active immunisation of Rhesus negative women.

Advances in animal vaccine development in Australia have been made by CSIRO's Animal Health Division, especially through the work of Lionel Bull e.g. bovine pleuropneumonia vaccine. Progress in the development of human vaccines advanced by scientists around Australia has included vaccines for cholera, tuberculosis, Q fever, human papillomavirus, *Helicobacter pylori* and malaria. These contributions will not go unrewarded in the ongoing effort to reduce the burden of these diseases.

Vaccine trials in Australia: Professor Terry Nolan

Professor Terry Nolan outlined the recent history of vaccine trials in Australia from the 1980s until the present. A range of governmental initiatives in the 1980s paved the way for increased trials and a broad range of vaccines were successfully trialed. Australia is an attractive site for industry sponsored trials because it is seen as good value economically, has a rigorous regulatory environment, accepted internationally, access to a large population in the Southern hemisphere with alternate seasonal cycles, good recruitment capabilities and high quality researchers. Recent threats to future trials were outlined, including complicated and lengthy ethics procedures, competitive recruitment between countries, and funding restrictions. The successful creation of the Indigenous trial network, although expensive to set up, is an excellent initiative with an exciting future. The road ahead promises new vaccines trialed by enthusiastic and dedicated researchers.

Collaborative Research Centre for Vaccine Technology: Professor Anne Kelso

The Collaborative Research Centre (CRC) for Vaccine Technology was established in 1993 with the aim of maximising the economic and social benefits of publicly funded vaccine research and design through collaboration between researchers, government and industry. In the past decade, the CRC has taken many novel ideas and transformed them into potential commercially successful products and along the way considerably enhanced the numbers of commercially aware PhD students and research managers in Australia. Highlighted concepts included new platform technologies to combine multiple T cell epitopes and methods to enhance immunogenicity. The CRC's vaccine targets include malaria (2 potential candidates), Epstein-Barr virus, cytomegalovirus (CMV) and Group A *Streptococcus* and a highly publicised animal immunocastration vaccine. Different pathways to protect and develop resulting CRC intellectual property were outlined including the creation of a start up company, VacTXPty Ltd. Finally, Professor Kelso noted that the CRC for Vaccine Technology will wind down in June 2006, leaving a lasting legacy of durable networks and will be replaced by the CRC for Immunological Principles.

The contribution of Professor Margaret Burgess: Professor Kim Oates, Associate Professor Peter Shaw, Dr Mary Bergin, Associate Professor Peter McIntyre

Professor Kim Oates summarised the early years of Professor Margaret Burgess' professional life. Margaret attended Fort Street Girls' High School, Sydney and was school captain in 1954. She completed her Bachelor degree in Medicine and Surgery in 1961 (University of Sydney), being awarded the Dagma Berne Prize for first place among women candidates and Distinction and first place in Surgery. She trained in Paediatrics and became a Fellow of the Royal Australasian College of Physicians in 1972. Between 1965 and 1984, she worked as a Research Fellow (1965–1970) at the Children's Medical Research Foundation, Royal Alexandra Hospital for Children, Sydney and then as a Norman Gregg Senior Research Fellow (1970–1984), also at the Children's Medical Research Foundation. She gained a MD in 1971 (University of Sydney). Her major work was on the epidemiology of congenital malformations and their relation to infection in pregnancy, particularly congenital rubella. Later on, her research focussed on vaccine preventable diseases. In the early 1970s her team of researchers carried out the first clinical trials of rubella vaccines in Australia.

Associate Professor Peter Shaw and Dr Mary Bergin then summarised Margaret's involvement with the Oncology Department at Royal Alexandra Hospital for Children (RHAC), Sydney. From 1972 until 1995, her work was shared between clinical work in the Oncology Department and research into vaccine preventable diseases. Margaret was appointed Senior Staff Physician at Royal Alexandra Hospital for Children in 1984. Her colleagues from Oncology remember her not only as a rigorous scientific researcher, but also as an accomplished clinician and an invaluable mentor. She contributed to a number of research projects, including the documentation of a high prevalence of growth failure and growth hormone deficiency in children who had been treated for acute lymphoblastic leukaemia, and the morbidity and mortality associated with varicella-zoster infection in the Oncology Unit.

In 1995, she became the Director of the Centre for Immunisation Research (CIR at the University of Sydney and RAHC). In 1997, under her leadership, the Centre was successful in securing the Commonwealth's tender to become the National Centre for Immunisation Research and Surveillance of Vaccine Preventable Diseases (NCIRS).

Associate Professor Peter McIntyre wrapped up the session with an impressive list of Margaret's achievements as Director of CIR/NCIRS. Early work by the Centre included the demonstration of the successful use of measles and mumps vaccines in Australian children aged as young as 12 months and the conduct of a very large hepatitis B serological study of 3,000 Sydney schoolchildren. Recent research has focused on barriers to immunisation uptake in Australia, the epidemiology of varicella-zoster, rotavirus, measles and pertussis, and trials of new vaccines including multivalent vaccines for children and pertussis vaccines for adults.

In 1998, Margaret became Professor of Paediatrics and Preventive Medicine at the University of Sydney. She was awarded an Order of Australia in 2003 for services to public health in Australia and overseas, particularly through the provision of policy advice to government and research into vaccine preventable diseases. She has been a member of various committees and working parties at the state, national and international levels such as the Australian Technical Advisory Group on Immunisation, Department of Health and Ageing and National Health and Medical Research Council and the Strategic Advisory Group of Experts to the Department of Vaccines and Biologicals, World Health Organization (WHO) (1999–2002). She is a patron of the NSW Deaf Children's Association. Margaret has authored at least 185 peer-reviewed articles and numerous books and book chapters. Peter finished with a poem particularly well suited to 'our' Margaret – 'To Margaret' by Charles Lamb (1775–1834).

To Margaret

Margaret, in happy hour
Christen'd from that humble flower
Which we a daisy call!
May thy pretty name-sake be
In all things a type of thee,
And image to thee in all.

Like *it* you show a modest face,
An unpretending native grace;
The tulip, and the pink,
The china and the damask rose,
And every flaunting flower that blows,
In the comparing shrink.

Of lowly fields you think no scorn,
Yet gayest gardens would adorn,
And grace, wherever set,
Home-seated in your lovely bower,
Or wedded - a transplanted flower-
I bless you, Margaret!

Charles Lamb (1775–1834)

Day two presentations: Vaccines for the prevention of congenital and neonatal infections

Session One: Developmental immunology and vaccines
Chair: Dr Alyson Kakakios

Clinical principles: Associate Professor Susan Prescott (School of Paediatrics and Child Health, University of Western Australia)*

Professor Prescott outlined the general concepts behind the immunological immaturity seen in the perinatal period, how the humoral and cellular immune system develops in the post natal period and how microbial products can modify the immune system's development. Interestingly there may be very good, as yet incompletely understood reasons, for the newborn's dampened and skewed immune response in 'letting the immune system off the leash slowly'. Current research looking at whether this is an intrinsic immaturity or active regulation and what factors are in play were highlighted. In addition, Associate Professor Prescott discussed different vaccination strategies to overcome this early hyporesponsiveness including using bacterial antigens and the potential concerns and limitations of these strategies. In the future improved understanding of the influence of perinatal exposures will assist in the development of vaccines aimed at allergic diseases, autoimmune diseases and malignancies.

Immunologic principles: Professor Patrick Holt (Institute for Child Health Research)

Professor Holt's presentation elaborated on some of the problems highlighted by the previous presenter. Evidence showing important immunological maturation between 12 to 18 months of age and some of the possible reasons to account for it were discussed, including genetic differences and environmental factors. Data yet to be published were presented showing that delayed immune maturation correlated with a family history of atopy. Professor Holt noted that perinatal immunology is a developing and exciting field and each new answer leads to new questions important for future vaccine initiatives.

Developmental immunology and vaccines in premature infants: Professor Don Roberton*

Professor Roberton presented a range of studies which showed that premature infants have lower antibody responses to many, but not all, immunisation antigens. For some of these antigens, the depressed response persists well into childhood. However, few studies have been performed to look at functional antibodies in this vulnerable group of children. One of the strategies to address this problem might be to combine immunisation of infants with boosting the immunity of pregnant women. This has been shown with Hib-PRPT vaccine to result in increased antibodies in breast milk. He concluded that clinical trials of vaccine efficacy in premature infants were needed, but highlighted the logistic difficulties associated with doing such trials. Based on current knowledge, the recommendation for premature infants is to vaccinate at chronological age, except for hepatitis B vaccine, which should be delayed until 30 days or discharge from hospital.

Session Two: Vaccines for neonatal viral infections
Chair: Prof David Isaacs

Human cytomegalovirus: Professor Stanley Plotkin

Professor Plotkin presented data showing that approximately one per cent of live births are affected by congenital CMV infection, of which about 20 per cent die or have long term sequelae. Congenital CMV is the leading infectious cause of neurological damage in infants. An effective CMV vaccine would not only prevent congenital infection but also the problem of CMV infection in transplant recipients. CMV vaccines currently in development include a live attenuated virus, a live attenuated/virulent recombinant virus and protein subunit vaccines consisting of glycoproteins or other proteins known to be important in immune responses. Several of the CMV vaccines have entered phase I or phase II trials. They have been shown to be safe and effective in inducing neutralising antibodies and cell-mediated immunity. One of the major hurdles to be overcome in phase III trials is recruitment of enough women to demonstrate protection from congenital infection in their babies.

Vaccines against Neonatal Herpes: Dr Cheryl Jones*

Neonatal herpes occurs in about one in 25,000 births in Australia with a quarter of infected babies dying shortly after birth. The most common source of the infection is from the mother who develops primary genital herpes infection in the later stages of pregnancy, often acquired from an asymptomatic partner. The most effective way of preventing neonatal herpes would be to immunise women against genital herpes with a long lasting broadly immunogenic vaccine. This may be best achieved by immunisation during early adolescence. Candidate vaccines that have been tested in clinical trials include live attenuated strains, replication-defective strains and protein sub-unit vaccines. A recent international multicentre efficacy trial of recombinant HSV-2 glycoprotein D sub-unit vaccine combined with the adjuvant monophosphoryl lipid have shown it to be partially protective in women who were seronegative for both herpes symplex virus (HSV) strains against genital disease (approximately 74% effective) and infection (approximately 40% effective), but not in men, or in women who were seropositive for HSV type 1. Future goals in the development of an effective vaccine to prevent neonatal herpes include better understanding of the immunological correlates of protection.

Hepatitis B virus: Professor Felicity Cutts*

Professor Cutts described the worldwide epidemiology and natural history of hepatitis B infection (HBV), which is responsible for approximately 750,000 deaths per year due to the development of cirrhosis and hepatocellular carcinoma. In relation to perinatal transmission, 70–90 per cent of infants born to mothers with a high HBV viral load (eAg +ve) and less than 10 per cent of infants born to mothers with low viral load (eAg -ve) become chronic carriers. In utero transmission is infrequent (<2%) and there is no evidence of transmission of hepatitis B through breastmilk. In areas of high hepatitis B

endemicity (≥ 8% infected) perinatal and early childhood infections are common. Primary vaccination (3 doses) will result in protective antibody levels in at least 95 per cent of infants.

In 1987 WHO recommended that by 1997 universal hepatitis B vaccination programs should be implemented. Unfortunately, by 2003 not all countries had programs in place with cost a significant barrier. The necessity for implementing a birth dose, rather than an alternate schedule, is variable according to the epidemiology, with a birth dose being less necessary in Africa where a lower proportion of infections are acquired perinatally than in South-East Asia where a high proportion are acquired perinatally. There are examples of the successful implementation of birth dose programs in developing world settings (e.g. Lombok). As the vaccine is extremely heat stable innovative strategies are possible in developing settings. Professor Cutts concluded that hepatitis B vaccine is a very effective vaccine, which is almost as cost-effective as measles vaccine.

Developing a live respiratory syncytial virus vaccine: Dr E David McIntosh (for Dr Valerie Randolph and Dr Frank Malinoski)*

Respiratory syncytial virus (RSV) is the leading cause of serious bronchiolitis and pneumonia in infants and young children, causing an estimated 4,500 deaths a year in the United States of America and 90,000 hospitalisations. There have been various attempts to develop a safe and effective RSV vaccine for infants since 1966. Currently, candidate live attenuated vaccine strains given intranasally are undergoing clinical testing, with promising early results. The recombinant approach to development has yielded a number of candidates, containing various mutations and deletions. Sub-unit vaccines are also undergoing clinical trials: a purified fusion protein-2 vaccine has shown promising results when administered to pregnant women.

Rotavirus: Professor Graeme Barnes*

Rotavirus infection is a significant problem in developing countries, with many hospitalisations for the disease occurring in young infants aged under six months. This epidemiology clearly favours neonatal immunisation. Neonatal rotavirus infection (in the first days/weeks of life) occurs in two circumstances: with community strains or with adapted nursery strains (nosocomial, with only one in 30 cases being symptomatic.) Cohort studies have demonstrated that whilst early infection with nursery strains does not offer protection against subsequent infection with rotavirus, it does offer protection against developing disease. Vaccines based upon neonatal strains are currently in development.

Session Three: Vaccines for other neonatal infections
Chair: Professor Don Roberton

Neonatal immunisation for pneumococcal disease: Dr Peter Richmond*

The burden of pneumococcal disease in early childhood is particularly high in Aboriginal and Torres Strait Islander populations in Australia, and in developing countries, where *Streptococcus pneumoniae* is the most common cause of meningitis and septicaemia in children aged less than three months. Infection and carriage in the first three months of life are linked to higher risk of pneumococcal disease in childhood and adulthood. The development of immunological tolerance during neonatal infection may be the cause of this higher risk. While early vaccination may prevent early infection, not enough is currently known about the impact of earlier 7-valent pneumococcal conjugate vaccination on immunological memory, response to infection or other vaccines, serotype replacement or adverse events. An Australian-funded study is currently underway in Papua New Guinea, and another in Kenya, to compare the outcomes of commencing vaccination at birth, one month and two months of age.

Is neonatal group B streptococcal disease vaccine preventable? Professor Lyn Gilbert*

Group B streptococcal disease emerged in the 1970s in Australia as the most common cause of neonatal sepsis and infectious stillbirth, probably through the emergence of a newly virulent serotype III. Since then, intrapartum antibiotic prophylaxis of pregnant women, identified through screening for carriage or clinical risk factors, has been effective in reducing the incidence of neonatal sepsis. Several vaccines have been developed over the years but none has progressed to phase III trials, because of the relatively low incidence of disease and reservations about vaccinating pregnant women. In future, improved methods are needed for identifying women at risk and virulent clones, to enable more targeted use of antibiotics or new generation vaccines.

Neonatal pertussis: Associate Professor Peter McIntyre*

Most cases of pertussis now occur in adolescents and adults. However, almost all deaths and 80 per cent of hospitalisations occur in those aged under three months. Three strategies have been considered to prevent neonatal disease—maternal, neonatal and parental immunisation. There is evidence to support the effectiveness of maternal immunisation and neonatal immunisation with DTPa. Immunisation of both parents is likely to be logistically more difficult and less cost effective. Analysis so far has shown a birth dose to be the most cost effective of the three options, but all are relatively expensive. However, it is likely that the currently available data underestimate the burden of disease, and therefore the cost-effectiveness of these strategies.

Immunisation for the prevention of neonatal tetanus: Professor Kim Mulholland

The incidence of neonatal tetanus is estimated by WHO to be approximately 200,000 cases per year. It occurs almost exclusively in developing countries in circumstances where most births are unassisted, and over 90 per cent of cases are fatal, due to lack of access to assisted ventilation. In 1989 the World Health Assembly adopted a resolution for the elimination of neonatal tetanus. Since then the incidence is estimated to have decreased by two-thirds. Current WHO policy is for a comprehensive strategy which includes the promotion of clean deliveries, disease surveillance and both childhood (3 doses of DTP in infancy and a booster 1 year later) and maternal immunisation. Tetanus vaccination coverage for the first three doses in infants and two doses in women has recently been estimated at around only 50 per cent in high-risk areas. A single-dose vaccine would significantly improve the prospects for elimination, but attempts to develop it have so far been unsuccessful, as boosting is needed. Work is progressing on a microcapsule delivery system for gradual vaccine release.

Tuberculosis: Professor Warwick Britton*

Bacille Calmette-Guerin vaccine has been used for the prevention of tuberculosis (TB) for over 50 years. It is most effective for the prevention of primary disease including miliary TB, but only 50 per cent effective against re-activation. It is therefore widely used for the prevention of serious disease in children in high prevalence countries, but has limited use in controlling TB incidence and is rarely used in adults. It has also been shown to have an adjuvant effect, boosting responses to the oral polio vaccine and hepatitis B vaccine, although the clinical significance of this is not clear. A recent study in Sydney found BCG to be effective in preventing asthma in children with a family history of atopic disease. Recent work focuses on the development of recombinant vaccines (against the sub-unit) and live attenuated vaccines. Future trials should be combined with HIV vaccine trials, given the importance of HIV-TB co-infection.

Conclusion

This colloquium allowed us to look back at the contributions many clinicians and researchers have made nationally and internationally to the development of vaccines and vaccination programs. In the near future infections such as measles and rubella will be little more than of historical interest in Australia. The colloquium also illustrated, sometimes in fascinating detail, the progress which is being made in basic research and in vaccine development by contemporary scientists. We heard about the wide range of organisms and diseases which have become or are likely to become amenable to prevention or treatment using vaccines.

We were left with several important challenges: how to appropriately evaluate and introduce some of the ‘old’ and many of the newer costly vaccines into developing countries, how to develop and deliver effective vaccines to neonates and how to work cooperatively in the early stages of vaccine research and development to make sure that the vaccines most needed globally (e.g. HIV) are fast tracked. The generous contribution of the international speakers to these discussions with our Australian experts helped all who attended to anticipate the likely future successes and disappointments.

Creutzfeldt-Jakob disease surveillance in Australia January 1970 to December 2003

Genevieve M Klug,[1] Victoria Lewis,[1] Alison Boyd,[2] James S Lee,[1] Colin L Masters,[3] Steven J Collins[3]

Abstract

The Australian National Creutzfeldt-Jakob Disease Registry (ANCJDR) was established by the Commonwealth Government in October 1993 in response to the recognition of four probable human pituitary hormone related Creutzfeldt-Jakob disease (CJD) deaths. An inquiry[1] into CJD in Australia and the use of human pituitary hormones under the Australian Human Pituitary Hormone Program suggested the expansion of some activities of the Registry to include retrospective case ascertainment from 1 January 1970. In parallel with monitoring possible medically acquired (iatrogenic) cases of CJD, the ANCJDR prospectively monitors and investigates all suspect cases of transmissible spongiform encephalopathies occurring within the states and territories of Australia, including sporadic and familial, and the potential occurrence of variant CJD. The ANCJDR also actively participates in an international surveillance consortium. This brief report summarises methods of classification and ascertainment as well as current epidemiological findings and new surveillance techniques that are being adopted to improve case ascertainment. *Commun Dis Intell* 2004;28;356–358.

Key words: Creutzfeldt-Jakob disease, transmissible spongiform encephalopathies, surveillance

Introduction

Transmissible spongiform encephalopathies (TSE) comprise a group of rare neurodegenerative disorders that are invariably fatal and develop in both animals and humans. Creutzfeldt-Jakob disease (CJD) is the most common human TSE and occurs sporadically, secondary to prion protein gene (*PRNP*) mutations or through medical interventions using contaminated therapeutics/equipment. Variant CJD (vCJD) is zoonotically linked to bovine spongiform encephalopathy (BSE), the commercial bovine livestock form of prion disease, of which Australia remains free as of December 2003.[2,3]

Classification and notification methods

The Australian National Creutzfeldt-Jakob Disease Registry (ANCJDR) utilises internationally recognised case definitions for classification of definite, probable or possible cases.[4,5] An incomplete status is given to suspect cases where investigation is pending. Definite cases are those that have been confirmed neuropathologically. Probable cases are classified on the basis of clinical profile and a characteristic electroencephalogram (EEG) and/or a positive 14-3-3 cerebrospinal fluid (CSF) test. Possible cases fulfil the same clinical profile in the absence of an EEG or with an atypical EEG and either no 14-3-3 CSF test or a negative result. The method of classification of possible cases is in accordance with the EUROCJD diagnostic criteria and has been adopted since 1 January 2001.

Case ascertainment relies on the notification of suspect cases to the registry by numerous methods. The most numerically important method for ascertaining cases overall has been personal communication from medical practitioners (41.6% of registry cases). Previous sources of suspect cases include hospital and health department records searches, death certificates searches, communication from the Pituitary Hormone Taskforce, the CJD counselling service and families. Since 1997, requests to the ANCJDR for diagnostic testing by assessing the presence of

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the CSF 14-3-3 protein has led to an increase in the notification of suspect cases, accounting overall for 19.9 per cent of all registry cases. At present, this source is the most important ongoing mechanism for referral of cases.

Surveillance summary to 31 December 2003

As of 31 December 2003, there were 519 cases on the register with 274 definite cases and 180 probable cases (Table). There were six cases of possible CJD of which five were of sporadic and one iatrogenic. A total of 59 cases were incomplete with 27 of these cases still alive. Four hundred and nine suspect cases have been excluded after detailed follow-up. As of December 2003, no further cases of iatrogenic CJD have been detected since 2000 and Australia remains free of vCJD.

There has been a steady increase in the annual incidence of spongiform encephalopathies since 1970, with rates stabilising in the last few years (Figures 1 and 2). This is consistent with, and analogous to, the experience of other CJD surveillance programs, with the increase probably reflecting case ascertainment bias stemming from improved recognition, reporting, investigation and case confirmation. The average annual age-adjusted mortality rate during the period from 1970 to 2003 is 0.83 deaths per million per year. During the prospective period of ANCJDR surveillance from 1993 to 2003, the average annual rate of mortality was 1.19 deaths per million population. This epoch is considered a more reliable period for analysis because prospective ascertainment was employed, and standardised approaches to case classification and ascertainment were implemented nationally.

Figure 1. Definite and probable cases of Creutzfeldt-Jakob disease on the Australian National Creutzfeldt-Jakob Disease Registry, 1 January 1970 to 31 December 2003

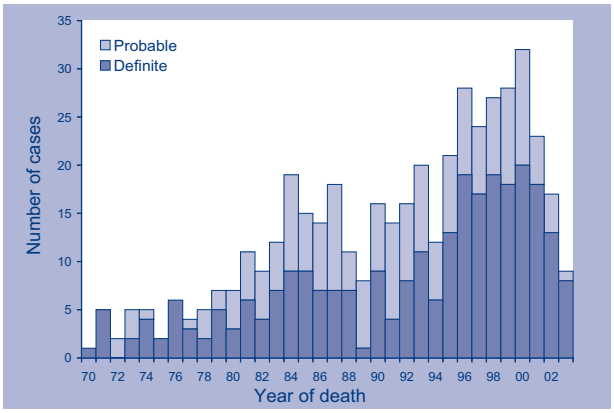
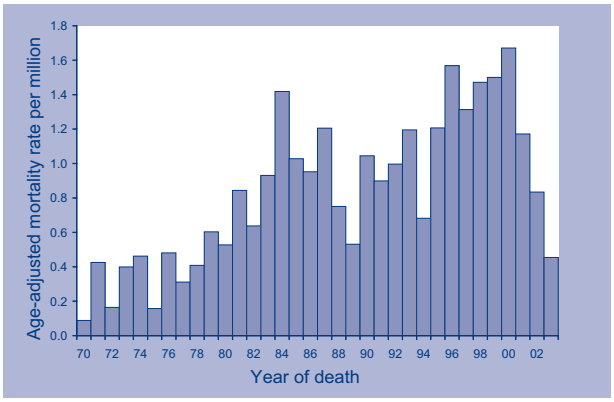


Figure 2. Age-adjusted mortality rates* of definite and probable cases of Creutzfeldt-Jakob disease, 1 January 1970 to 31 December 2003



* Mortality rates were calculated using the Australian Bureau of Statistics 2000 resident population estimates for Australia.

Table. Classification of cases on the Australian National Creutzfeldt-Jakob Disease Registry, 1 January 1970 to 31 December 2003

Classification	Sporadic	Familial	Iatrogenic	Variant CJD	Unclassified	Total
Definite	244	25	5*	0	0	274
Probable	168	8	4	0	0	180
Possible	5	0	1	0	0	6
Incomplete	0	0	0	0	59†	59
Total	417	33	10	0	59	519

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

† Includes 27 living cases.

The majority of Australian definite and probable CJD cases are sporadic (90.9%). Familial and iatrogenic cases constitute 7.3 per cent and 1.8 per cent, respectively, of all definite and probable cases. The percentage of familial cases observed in Australia is less than the 12–14 per cent reported by European surveillance programs.[6] An explanation for this difference most likely relates to the non-systematic approach to *PRNP* testing adopted in Australia, which from 1997 to 2002 was undertaken in 31 per cent of all definite and probable cases.

The duration of illness for CJD cases varies depending on aetiology. The median length of illness duration for all CJD cases was four months. For sporadic cases, median duration was found to be four months (range, 0.9–60 months), for iatrogenic cases 6.25 months (range, 2–25 months) and for familial cases seven months (range, 1.5–192 months). Familial CJD was found to be associated with a significantly greater survival time in comparison to sporadic CJD ($p<0.0001$ by log rank test).

In sporadic CJD, no significant gender differences have been observed. Overall, 47.6 per cent of cases were male and 52.4 per cent were female. The average age of death in sporadic cases by gender was 65 years (range, 25–88) for males and 67 years (range, 33–89) for females. Over the period 1970 to 2003, there was no difference between the average age-specific mortality rates of males (0.75 deaths/million/year) and females (0.77 deaths/million/year). In males, the peak mortality rate occurred between 70–74 years (4.0 deaths/million/year) and in females between 65–69 years (4.5 deaths/million/year).

In comparison to sporadic cases, the average death age of familial cases was 52 years (range, 20–82 years) in males and 61 years (range, 42–82 years) in females. Peak mortality rates occurred in the 65–69 year age group in both males (0.27 deaths/million/year) and females (0.35 deaths/million/year) and in iatrogenic cases, the average death age was 45 years (range, 27–62 years) for males and 39 (range, 26–50 years) for females.

Analysis of the occurrence of sporadic cases by state of residence showed that the rate of death was not significantly different in any state or territory compared to the rate in the Australian general population. Furthermore, sporadic CJD does not exhibit a significant association with region of birth or travel history of Australian-born cases.

In order to facilitate optimal surveillance, the Communicable Diseases Network Australia agreed to designate TSEs as a notifiable disease. At the time of writing, CJD was notifiable in Tasmania, Victoria, Western Australia and New South Wales with the remaining states and territories to follow.

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Foodborne disease investigation across Australia: Annual report of the OzFoodNet network, 2003

The OzFoodNet Working Group

Abstract

In 2003, OzFoodNet conducted enhanced surveillance of foodborne diseases across Australia, which covered all states and territories. During 2003, there were 23,250 notifications of eight potentially foodborne diseases, of which 67 per cent and 30 per cent were due to *Campylobacter* and *Salmonella* infections respectively. The most common *Salmonella* serotype was Typhimurium, as in previous years. Most *S.* Enteritidis were acquired overseas, except for Queensland where 52 per cent of infections were acquired locally. Locally acquired *S.* Enteritidis infections in Australia were predominantly due to phage type 26. The most common serotype of Shiga toxin producing *E. coli* was O157, although for 49 per cent of notified infections serotype was unknown due to the use of polymerase chain reaction based screening tests. There were 12 materno-foetal listeriosis infections in 2003, which was an increase compared to recent years. During 2003, there were 444 outbreaks of gastroenteritis and foodborne disease recorded. Ninety-nine of these were of foodborne origin affecting 1,686 persons, hospitalising 105 and causing six deaths. A wide range of agents and foods caused these outbreaks, with *Salmonella* Typhimurium being the most common pathogen. Outbreaks associated with fish and seafood dishes, poultry meat, and Asian style and imported foods were common. Four outbreaks with international implications were reported: an outbreak of *Salmonella* in Montevideo involving contaminated tahini from the Middle East and three outbreaks of norovirus infection associated with imported Japanese oysters. Outbreak data indicated a need to monitor food safety in aged care settings, restaurants and catering. Eighty-nine investigations into clusters of gastrointestinal illness where a source could not be identified were conducted, including multi-state outbreaks of salmonellosis. One multistate investigation of antibiotic resistant *Salmonella* Paratyphi b Java identified 18 cases who had recent exposure to tropical fish aquariums. Ninety-seven per cent of *Salmonella* notifications on state and territory surveillance databases have complete information on serotype and phage type. In 2003, OzFoodNet demonstrated the benefits of national collaboration to control food borne disease. *Commun Dis Intell* 2004;28:359–389.

Keywords: surveillance, foodborne disease, disease outbreak, Salmonella*, Enteritidis,* Campylobacter*,* Listeria*,* Yersinia*,* Shigella*, typhoid*

Introduction

Foodborne disease surveillance is a fundamental part of ensuring a safe food supply.[1] Many countries have conducted passive surveillance of foodborne diseases through statistics of patient encounters with health systems.[2] These systems have several limitations, particularly where the data are based on syndromic diagnoses rather than isolation of micro-organisms. To improve the capacity to interpret surveillance data, some countries have collected extra data on patients infected with foodborne illness, or have collected complementary data from animals and foods.[3,4]

The Centers for Disease Control and Prevention in the United States of America (USA) established the FoodNet active surveillance system in 1995.[3] FoodNet consists of 10 sentinel sites across the USA where laboratories report weekly cases of infections that may be transmitted by food. The system has

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helped to quantify the burden of foodborne illness and understand its causes. FoodNet has provided a major platform for special research into foodborne diseases.

In 2000, the Australian Government Department of Health and Ageing established the OzFoodNet network to enhance surveillance for foodborne disease.[5,6] This built upon an 18-month trial of active surveillance in the Hunter region of New South Wales.

OzFoodNet was modelled on the FoodNet surveillance system, although it differs in some important respects.[3] OzFoodNet:

- does not actively contact laboratories for reports of individual infections (active surveillance), but relies upon Australia's laboratory-based notification system;
- coordinates investigations into outbreaks of national significance;
- collects data on outbreaks of foodborne and gastrointestinal illness due to all modes of transmission;
- covers the whole Australian population; and
- conducts studies of locally important pathogens in different jurisdictions.

OzFoodNet is primarily a national network of epidemiologists that conducts investigations and applied research into foodborne disease. The network involves many different organisations, including the National Centre for Epidemiology and Population Health, and the Public Health Laboratory Network. OzFoodNet is a member of the Communicable Diseases Network Australia (CDNA), which is Australia's peak body for communicable disease control.[7] The Australian Government Department of Health and Ageing funds OzFoodNet and convenes committees to manage the network and oversee the scientific quality of its work.

This is the third annual report of OzFoodNet and covers data and activities for 2003.

Methods

Population under surveillance

In 2003, the coverage of the network included the entire Australian population, which was estimated at mid-year to be 19,662,781 persons (Australian Bureau of Statistics (ABS), June 2004).

OzFoodNet sites were located in every state in 2003, there was an OzFoodNet site in the Hunter Area Health Service of New South Wales, which complemented foodborne disease surveillance across New South Wales. The Hunter site conducts thorough local investigation and provides a baseline for foodborne disease incidence in New South Wales. In 2003, the population covered by the Hunter site was estimated to be 544,623 persons.

Data sources

Rates of notified infections

All Australian states and territories require doctors and/or pathology laboratories to notify patients with infectious diseases that are important to public health. Western Australia is the only jurisdiction where laboratory notification is not mandatory under legislation, although most laboratories still notify the health department. OzFoodNet aggregated and analysed data on patients notified with the following diseases or conditions, a proportion of which may be acquired from food:

- *Campylobacter* infections;
- *Salmonella* infections;
- *Listeria* infections;
- *Yersinia* infections;
- Shiga toxin producing E. coli infections and haemolytic uraemic syndrome;
- typhoid; and
- *Shigella* infections.

To compare the current rates of disease with previous levels, OzFoodNet compared crude numbers and rates of notification to the means of the previous five years. Where available, numbers and rates of notifications for specific sub-types of infecting organisms were compared to notifications for the previous year.

To calculate rates of notification the estimated resident populations for each state or territory for June 2003, or the specified year, were used (ABS, June 2004). Age specific rates for notified infections in each state or territory were also calculated.

The date that notifications were received was used to analyse notification data. These data are similar to those reported to the National Notifiable Diseases Surveillance System (NNDSS), but may differ for methodological reasons.

Gastrointestinal and foodborne disease outbreaks

OzFoodNet collected information on gastrointestinal and foodborne disease outbreaks that occurred in Australia during 2003. The reports collate summary information about the outbreak setting, the date, the aetiological agent, the number of persons affected, the type of investigation, the level of evidence and the food vehicle. Data on outbreaks due to transmission from animals and cluster investigations were also summarised.

Risk factors for infection

To identify risk factors for foodborne infection in Australia, OzFoodNet reviewed summary data from outbreaks that occurred in 2003 and compared them to previous years. Data from several complementary OzFoodNet studies of foodborne illness in Australia were also examined.

Surveillance evaluation and enhancement

OzFoodNet compared the results of surveillance across different sites, including rates of reporting outbreaks, and investigation of clusters of *Salmonella*. To measure the quality of national surveillance data, OzFoodNet examined the completeness of information on state and territory databases in 2003. The proportions of *Salmonella* notifications with serotype and phage type information were compared with results for the previous three years.

Results

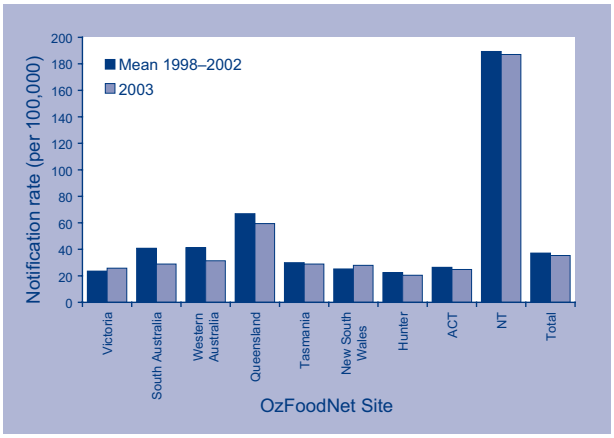
Rates of notified infections

In 2003, OzFoodNet sites reported 23,250 notifications of eight diseases that were potentially foodborne. This was a 5.5 per cent increase from the mean of 22,035 notifications for the previous five years. Reports for these eight diseases make up almost a quarter of notifications to the NNDSS Diseases Surveillance System.[14] A summary of the number and rates of notifications by OzFoodNet sites is shown in Appendix 1.

Salmonella *infections*

In 2003, OzFoodNet sites reported 7,032 cases of *Salmonella* infection, which indicated a rate of 35.4 cases per 100,000 population and a decrease of 4.8 per cent from the mean for the previous five years (Figure 1). The rates ranged from 20.4 cases per 100,000 population in the Hunter region to 187.1 cases per 100,000 population in the Northern Territory.

Figure 1. Notification rates of *Salmonella* infections for 2003 compared to mean rates for 1998 to 2002, by OzFoodNet site



Overall, notification rates of salmonellosis for 2003 were increased in New South Wales (11.2%) and Victoria (9.2%) compared to historical means. There were moderate declines in the notification rate of *Salmonella* in all other states and territories, with more significant declines in South Australia (–29.3%) and Western Australia (–24.0%).

The overall ratio of male to female cases was approximately 1:1, ranging from 1.2:1 in Tasmania to 0.8:1 in South Australia and Western Australia. The median age of cases ranged between 17 and 23 years at all OzFoodNet sites, except for the Northern Territory and Queensland where the median ages were three and 12 years respectively. There were no major changes in the median ages of salmonellosis cases from 2002 to 2003.

The highest rate of *Salmonella* infection was 189.5 cases per 100,000 population in males aged 0 to 4 years of age. The rate was highest in this age group for all sites and ranged from 76.8 cases per 100,000 population in the Australian Capital Territory to 1,432.8 cases per 100,000 population in the Northern Territory. Notification rates were also high in the 5–9 year age group in all jurisdictions. In most jurisdictions there was also a secondary peak in notification rates in the 20–29 year age range for males and females, which was most noticeable in the Northern Territory.

Rates of salmonellosis were highest in northern areas of Australia. The highest rate is consistently reported in the Kimberley region of Western Australia.[8,9] Western Australia reported that the Kimberley region had a rate of 314 cases per 100,000 population, with the majority of infections in Indigenous people. In Western Australia, rates of salmonellosis were higher in Indigenous people in all age groups, particularly in children aged 0–4 years

of age. Thirty-nine per cent (128/330) of *Salmonella* notifications in the Northern Territory were in persons of Aboriginal or Torres Strait Island origin. As in previous years, OzFoodNet sites reported that notification rates of salmonellosis increased from south to north along the eastern seaboard of Australia. The rate of notification increased from 25.8 per 100,000 population in Victoria to 106 per 100,000 population in far north Queensland.

During 2003, the most commonly reported *Salmonella* serotype was *S.* Typhimurium. There were 714 notifications of *Salmonella* Typhimurium 135 (including 135a) to OzFoodNet sites making it the most common infection (Table 1). Eighteen per cent (125/714) of these related to a single outbreak in Victoria. There were 678 notifications of this phage type last year. There were 405 notifications of *S.* Typhimurium 9 in 2003 compared to 583 for the previous year, which represents a 31 per cent drop in this common phage type. *S.* Typhimurium 170 and *S.* Typhimurium 108 continued to emerge as a significant phage type around Australia. In 2002, NSW investigators recognised that these two phage types were in fact the same organism after human specimens went to one laboratory for typing and food samples went to another. This explains why certain states and territories never reported cases of *S.* Typhimurium 170 and others never reported cases of *S.* Typhimurium 108. In the remainder of this report infections due to this organism are referred to as *S.* Typhimurium 170/108. There were 382 cases of *S.* Saintpaul, making it the most common *Salmonella* serovar following *S.* Typhimurium. The highest specific rates for single serotypes reported in OzFoodNet sites were *S.* Ball and *S.* Saintpaul in the Northern Territory and *S.* Mississippi in Tasmania with rates of 22.2, 14.1 and 14.7 per 100,000 population respectively.

Salmonella *Enteritidis*

S. Enteritidis is a serotype that can infect the internal contents of eggs through the oviducts of infected chickens, predominantly with *S.* Enteritidis phage type 4. People may become infected with this serotype after eating raw or undercooked eggs. This phage type has caused major problems in the northern hemisphere where it has become established in commercial egg laying flocks, although the incidence has declined in many countries.[10] Australia is largely free of *S.* Enteritidis phage type 4 except in people infected overseas. There are other phage types of *S.* Enteritidis that are acquired locally in Australia, although the causes of these local infections are largely unknown.

OzFoodNet has been conducting a case control study of all locally acquired *S.* Enteritidis infections in Australia to determine the risk factors for infection. The case control study was established in 2001 and assesses food-based and zoonotic risk factors for infection. These are compared to the exposure histories for up to three age-matched controls per case. Cases infected while overseas are not enrolled in the study, but they are asked about the countries they visited.

During 2003, OzFoodNet sites recorded 227 cases of *S.* Enteritidis, of which 63 per cent (142/227) had travelled overseas (Table 2). Travel history was unknown for 15% (33/227) of cases, while 23% (52/227) reported no travel out of Australia. Relevant travel histories were difficult to obtain, as people have often travelled to several countries before visiting Australia. Asian countries were commonly mentioned, reflecting that they are common travel destinations for Australians. In the Asian region, cases of *S.* Enteritidis infection reported travelling to Indonesia and Bali (46%), Singapore (11%), Malaysia (8%), Thailand (7%), and other Asian destinations (6%). Approximately 20 per cent of people acquiring their infection overseas reported travelling to Europe.

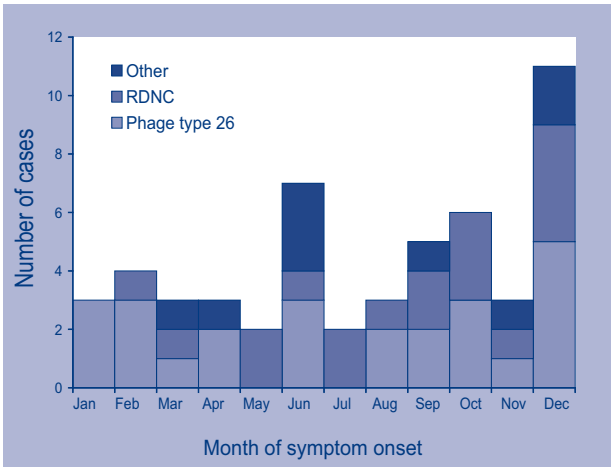
The most common phage types isolated varied with the region that the person travelled to. For people returning from Bali and Indonesia, the most common phage types were 6a, 4, and 4b. In Malaysia and Singapore the most common infecting phage types were 1 and 6a, with no phage type 4 reported at all. Travellers to Thailand were also infected with phage types 1 and 6a, along with phage type 4. Where cases had returned from Pacific countries, phage type 26 predominated. For travellers returning from Europe, phage types 1, 4 and 6 were most common.

OzFoodNet Sites reported a decrease in the total number of overseas-acquired *S.* Enteritidis infections, particularly as fewer travellers visited Bali, where this serotype is endemic. There was also a shift in the numbers of different phage types being notified, with *Salmonella* Enteritidis 4 declining and other phage types such as 6a increasing. Phage type 4b was recognised for the first time in 2002 after reference laboratories commenced testing for this particular phage in September 2001. Isolates of Phage type 4 typed prior to September 2001 have not been retested to determine whether a proportion of them are 4b. It is not possible to say whether there has been a real shift in phage types, from 4 to 4b or whether it is just a result of changed typing methods or changes in travel patterns.

Overall, 23 per cent (52/227) of patients infected with *S.* Enteritidis acquired their infection in Australia (Figure 2). The median age of cases was 24 years old (age range 0–76 years) and 50 per cent were male. Locally-acquired *S.* Enteritidis infections were

much more common in Queensland than in Victoria, with 52 per cent (39/75) versus four per cent (3/49) locally acquired. Most locally acquired infections in Queensland were due to phage type 26 (Table 3). There was a temporal clustering of cases of *S.* Enteritidis Reaction Does Not Conform (RDNC) in December 2003, although no common sources were identified (Figure 2). There were no locally acquired cases of *S.* Enteritidis in Tasmania or the Northern Territory.

Figure 2. *Salmonella* Enteritidis infections acquired in Australia by phage type and month of notification, 2003



RDNC: Reaction Does Not Conform.

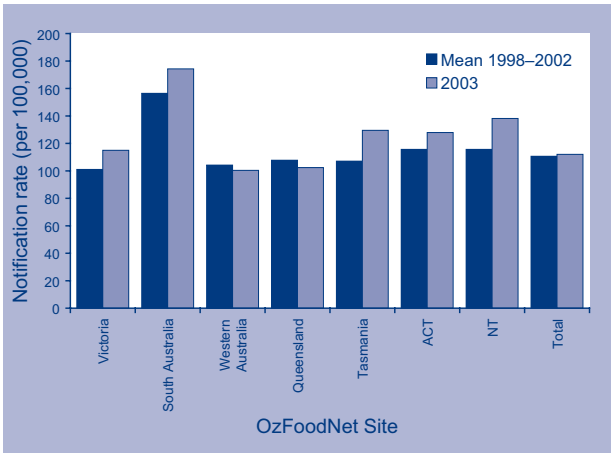
Salmonella *Clustering*

In total, state and territory health departments conducted 73 investigations into clusters and point source outbreaks of salmonellosis during 2002. A source of infection was identified for 47 per cent (34/73) of these investigations. Approximately 55 per cent (40/73) of these investigations were due to various phage types of *S.* Typhimurium.

Campylobacter *infections*

Data for campylobacteriosis were not available for New South Wales, including the Hunter Health Area. With this exception, in 2003 OzFoodNet sites reported 15,464 cases of *Campylobacter* infection, which indicated a rate of 112 cases per 100,000 population. This rate represented a 1.2 per cent increase over the mean for the previous five years (Figure 3). Tasmania, Northern Territory, Victoria and South Australia all recorded a greater than 10 per cent increase in rates of infections compared to the mean of the previous five years. Queensland and Western Australia reported slightly lower rates than for previous years. The highest rates of *Campylobacter* notification were in South Australia (174.2 per 100,000 population) and the lowest rates were in Western Australia (100.3 per 100,000 population).

Figure 3. Notification rates of *Campylobacter* infections for 2003 compared to mean rates for 1998 to 2002, by OzFoodNet site excluding New South Wales



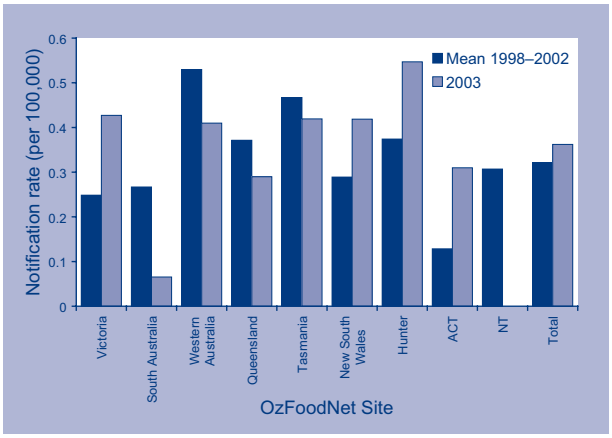
The ratio of male to females cases ranged from 1.1:1 in the Australian Capital Territory and Tasmania to 1.3:1 in the Northern Territory. The median ages of cases ranged from 17 to 30 years, except in the Northern Territory where it was five years of age. The highest age specific rates were in male children in the 0–4 year age group, with a secondary peak in the 20–29 year age range for males and females. The highest age specific rates were in males in the 0–4 year age group in the Northern Territory (958 cases per 100,000 population) and South Australia (433 cases per 100,000 population). There were four identified outbreaks of *Campylobacter* during 2003, two of which occurred in association with visits to farms where school students drank unpasteurised milk and had close contact with animals.

Listeria *infections*

OzFoodNet sites reported 72 cases of listeriosis in 2003, which represents a notification rate of 0.4 cases per 100,000 population (Figure 4). This was a 17 per cent increase in the number of notifications compared to the historical mean. There were no common source outbreaks of listeriosis detected during the period, although sites investigated instances of temporal clustering of cases using Pulsed Field Gel Electrophoresis testing of isolates.

Twelve infections in 2003 (17%) were materno-foetal infections, giving a rate of 4.7 cases per 100,000 live births.[11] This represents a considerable increase from two materno-foetal infections in the previous year. Victoria reported five materno-foetal infections during 2003, compared to a total of three cases in the previous three years. Amongst the Victorian cases, 80 per cent (4/5) of the mothers had previously received information about *Listeria*

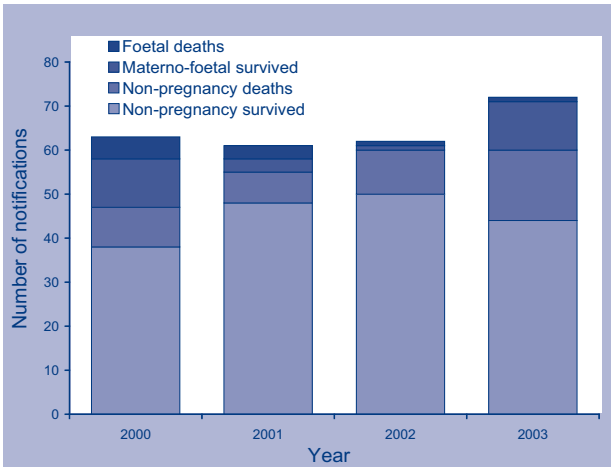
Figure 4. Notification rates of *Listeria* infections for 2003 compared to mean rates for 1998 to 2002, by OzFoodNet site



infection from their doctors. Western Australia had the highest rate with three notifications, although small numbers make rates unstable. The case fatality rate of eight per cent (1/12) for materno-foetal infections was considerably lower than for previous years (Figure 5).

Amongst non-pregnancy related cases, the male to female ratio was 1.1:1. OzFoodNet sites reported that the median ages of non-pregnancy associated cases were between 57–76 years old. The highest age specific rate of 2.1 cases per 100,000 population was in males over the age of 60 years. Twenty seven per cent (16/60) of non-pregnancy associated cases died.

Figure 5. Notifications of *Listeria* infections showing non-pregnancy related infections and deaths and materno-foetal infections and deaths in Australia, 2000 to 2003



Yersinia *infections*

In January 2001, the CDNA agreed to stop reporting notifications of *Yersinia* infections to the NNDSS, due to declines in incidence and lack of identified outbreaks. Victoria has revised regulations to remove yersiniosis from the list of reportable conditions and *Yersinia* is also not notifiable in New South Wales.

In 2002, OzFoodNet sites reported 117 cases of yersiniosis, which equated to a rate of 1.7 notifications per 100,000 population (Figure 6). The overall rate declined 20 per cent from previous years, when adjusted for the absence of reporting from Victoria and New South Wales. Queensland and South Australia recorded the highest rates of infection, with 2.5 and 1.2 notifications per 100,000 population respectively. Queensland reported 80 per cent (94/117) of all cases and the rates of yersiniosis were similar in all three Queensland health zones. The male to female ratio was approximately 1:1 and the highest age specific rate was 16.6 per 100,000 in 0–4 year old Queensland infants.

There was one cluster investigation into four cases of *Yersinia pseudotuberculosis* in South Australia in November 2003. All four children affected were from metropolitan Adelaide and presented with severe abdominal pain. Three underwent surgical intervention resulting in appendectomies. An investigation did not identify any common food or environmental exposure among the infected patients.

Figure 6. Notification rates of *Yersinia* infections for 2003 compared to mean rates for 1998 to 2002, Australia excluding Victoria and New South Wales, by OzFoodNet site

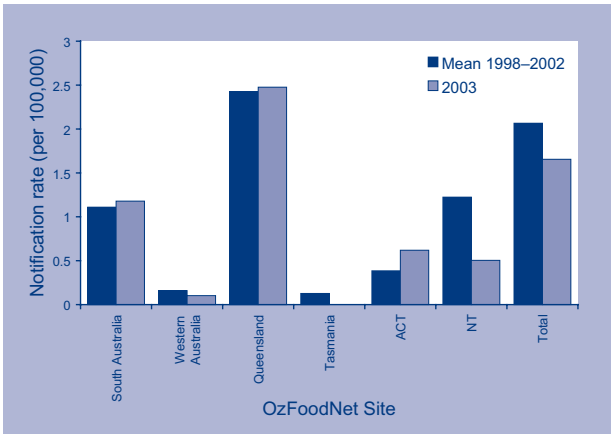


Table 1. Numbers, rates and proportions of the top 10 *Salmonella* infections, 2002 to 2003, by OzFoodNet site*

OzFoodNet site	*Salmonella* type (sero/phage type)	Top 10 infections					
		2003 n	2003 rate†	Proportion (%)‡	2002 n	2003 rate	Ratio§
Australian Capital Territory	Typhimurium 135	25	7.7	31.3	9	2.9	2.8
	Typhimurium 170	4	1.2	5.0	4	1.3	1.0
	Typhimurium 6var1	4	1.2	5.0	0	0.0	–
	Typhimurium 9	4	1.2	5.0	17	5.4	0.2
	Typhimurium U290	4	1.2	5.0	3	1.0	1.3
	Infantis	3	0.9	3.8	1	0.3	3.0
	Bovismorbificans 14	2	0.6	2.5	0	0.0	–
	Mississippi	2	0.6	2.5	0	0.0	–
	Saint Paul	2	0.6	2.5	0	0.0	–
Hunter	Typhimurium 104L	2	0.6	2.5	0	0.0	–
	Typhimurium 170	10	1.8	8.9	7	1.3	1.4
	Typhimurium 4	10	1.8	8.9	2	0.4	5.0
	Infantis	9	1.6	8.0	0	0.0	–
	Typhimurium U290	8	1.5	7.1	8	1.5	1.0
	Bovismorbificans 14	7	1.3	6.3	2	0.4	3.5
	Typhimurium 9	7	1.3	6.3	14	2.6	0.5
	Montevideo	6	1.1	5.4	21	3.9	0.3
	Birkenhead	3	0.5	2.7	2	0.4	1.5
	Chester	3	0.5	2.7	4	0.7	0.8
	Typhimurium 135a	3	0.5	2.7	2	0.4	1.5
	Typhimurium 197	3	0.5	2.7	4	0.7	0.8
New South Wales	Typhimurium 170	233	3.5	12.5	148	2.3	1.6
	Typhimurium 135	134	2.0	7.2	189	2.9	0.7
	Typhimurium 9	133	2.0	7.1	255	3.9	0.5
	Infantis	87	1.3	4.7	38	0.6	2.3
	Birkenhead	68	1.0	3.6	94	1.4	0.7
	Typhimurium 197	68	1.0	3.6	61	0.9	1.1
	Virchow	60	0.9	3.2	74	1.1	0.8
	Chester	40	0.6	2.1	28	0.4	1.4
	Typhimurium 12	38	0.6	2.0	19	0.3	2.0
	Typhimurium 135a	37	0.6	2.0	48	0.7	0.8
Northern Territory	Infantis	12	0.6	2.0	18	0.9	0.7
	Bali	44	22.2	11.9	49	24.8	0.9
	Saintpaul	28	14.1	7.5	19	9.6	1.5
	Anatum	22	11.1	5.9	15	7.6	1.5
	Typhimurium 135	17	8.6	4.6	9	4.6	1.9
	Chester	16	8.1	4.3	18	9.1	0.9
	Muenchen	14	7.1	3.8	14	7.1	1.0

Table 1. Numbers, rates and proportions of the top 10 *Salmonella* infections, 2002 to 2003, by OzFoodNet site* *continued*

OzFoodNet site	*Salmonella* type (sero/phage type)	Top 10 infections					
		2003 n	2003 rate†	Proportion (%)‡	2002 n	2003 rate	Ratio§
Northern Territory *continued*	Havana	11	5.5	3.0	3	1.5	3.7
	Subsp 1 ser 16:1,v	11	5.5	3.0	6	3.0	1.8
	Adelaide	10	5.0	2.7	5	2.5	2.0
	Weltevreden	10	5.0	2.7	5	2.5	2.0
Queensland	Saintpaul	167	4.4	7.4	227	6.3	0.7
	Virchow 8	165	4.3	7.3	279	7.7	0.6
	Typhimurium 135	155	4.1	6.9	110	3.0	1.4
	Birkenhead	109	2.9	4.8	136	3.7	0.8
	Chester	98	2.6	4.3	84	2.3	1.2
	Typhimurium 197	90	2.4	4.0	31	0.9	2.9
	Aberdeen	75	2.0	3.3	112	3.1	0.7
	Hvittingfoss	72	1.9	3.2	114	3.1	0.6
	Typhimurium 170	70	1.8	3.1	138	3.8	0.5
	Muenchen	55	1.4	2.4	60	1.7	0.9
South Australia	Typhimurium 108	32	2.1	7.3	25	1.7	1.3
	Typhimurium 9	28	1.8	6.3	24	1.6	1.2
	Chester	24	1.6	5.4	11	0.7	2.2
	Typhimurium 4	23	1.5	5.2	7	0.5	3.3
	Infantis	20	1.3	4.5	9	0.6	2.2
	Typhimurium 135a	18	1.2	4.1	15	1.0	1.2
	Typhimurium 135	17	1.1	3.9	13	0.9	1.3
	Typhimurium 12	15	1.0	3.4	17	1.1	0.9
	Typhimurium 12a	15	1.0	3.4	15	1.0	1.0
	Saintpaul	13	0.9	2.9	11	0.7	1.2
	Anatum	13	0.9	2.9	1	0.1	13.0
Tasmania	Mississippi	70	14.7	50.7	78	16.6	0.9
	Typhimurium 9	7	1.5	5.1	11	2.3	0.6
	Typhiumurium 135	6	1.3	4.3	15	3.2	0.4
	Saintpaul	5	1.0	3.6	3	0.6	1.7
	Typhimurium 170	5	1.0	3.6	0	0.0	–
	Typhimurium U290	5	1.0	3.6	2	0.4	2.5
	Typhimurium 4	4	0.8	2.9	1	0.2	4.0
	Infantis	3	0.6	2.2	1	0.2	3.0
	Typhimurium 126	3	0.6	2.2	4	0.9	0.8
	Typhimurium 12a	3	0.6	2.2	0	0.0	–

Table 1. Numbers, rates and proportions of the top 10 *Salmonella* infections, 2002 to 2003, by OzFoodNet site* *continued*

OzFoodNet site	*Salmonella* type (sero/phage type)	Top 10 infections					
		2003 n	2003 rate†	Proportion (%)‡	2002 n	2003 rate	Ratio§
Victoria	Typhimurium 135‖	233	4.7	18.4	177	3.7	1.3
	Typhimurium 9	160	3.3	12.6	151	3.1	1.1
	Typhimurium 170	125	2.5	9.9	162	3.4	0.8
	Typhimurium U290	88	1.8	6.9	39	0.8	2.3
	Infantis	54	1.1	4.3	22	0.5	2.5
	Typhimurium 197	21	0.4	1.7	10	0.2	2.1
	Stanley	19	0.4	1.5	12	0.2	1.6
	Typhimurium 12	19	0.4	1.5	8	0.2	2.4
	Typhimurium 126	18	0.4	1.4	61	1.3	0.3
Western Australia	Saintpaul	17	0.3	1.3	43	0.9	0.4
	Typhimurium 135a	41	2.1	6.7	63	3.3	0.7
	Chester	36	1.8	5.9	34	1.8	1.1
	Saintpaul	30	1.5	4.9	42	2.2	0.7
	Typhimurium 135	30	1.5	4.9	30	1.6	1.0
	Muenchen	28	1.4	4.6	27	1.4	1.0
	Oranienburg	21	1.1	3.4	6	0.3	3.5
	Mbandaka	20	1.0	3.3	5	0.3	4.0
	Typhimurium 9	20	1.0	3.3	45	2.4	0.4
	Typhimurium 126	17	0.9	2.8	5	0.3	3.4
	Senftenberg	15	0.8	2.5	8	0.4	1.9
	Anatum	12	0.6	2.0	14	0.7	0.9

* Where there were multiple tenth ranking *Salmonella* types all data have been shown, giving more than 10 categories for some sites.

† Rate per 100,000 population.

‡ Proportion of total *Salmonella* notified for this jurisdiction in 2003.

§ Ratio of the number of reported cases in 2003 compared to the number reported in 2002.

‖ *S.* Typhimurium 135 also includes cases of *S.* Typhimurium 135a.

Table 2. Numbers of *Salmonella* Enteritidis infections acquired overseas and in Australia in 2003, by OzFoodNet site

OzFoodNet site	History of travel overseas			Total
	Yes	No	Unknown	
Australian Capital Territory	2	1	0	3
New South Wales	23	2	15	40
Northern Territory	1	0	0	1
Queensland	19	39	17	75
South Australia	17	5	1	23
Tasmania	3	0	0	3
Victoria	47	2	0	49
Western Australia	30	3	0	33
Total	142	52	33	227

While *Yersinia* notifications have decreased in recent years, continued surveillance for yersiniosis is important to monitor for foodborne outbreaks and the effect of zoonotic control programs. In Queensland, the incidence of yersiniosis has increased each year since 2001 when the lowest rate of 1.5 per 100,000 population was reported.

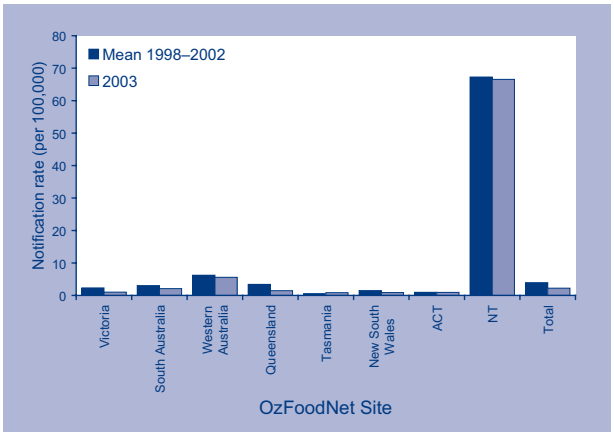
Shigella

OzFoodNet sites reported 443 cases of shigellosis during 2003, which was a notification rate of 2.2 cases per 100,000 population (Figure 7). This was a 43 per cent decrease in the rate of notification compared with historical averages, after adjusting for the introduction of notifications from New South Wales in January 2001.

The highest rate of notification was in the Northern Territory (67 cases per 100,000 population), which was 30 times the overall Australian rate. Rates of shigellosis are considerably higher in Indigenous communities. In Western Australia, the rates of shigellosis approached 300 cases per 100,000 population in Indigenous children aged 0–4 years of age.

Overall the notification rate for shigellosis was 43 per cent lower than the mean of the previous five years, and this observed consistently across jurisdictions. The male to female ratio of shigellosis cases was 1:1. The highest age specific rates were in males (12 cases per 100,000 population) and females (11 cases per 100,000 population) in the 0–4 year old age group.

Figure 7. Notification rates of *Shigella* infections for 2003 compared to mean rates for 1998 to 2002,* by OzFoodNet site



* Shigellosis became notifiable in New South Wales from 2001 onwards.

There was an outbreak of *Shigella flexneri* 2a reported at a school in Victoria in August 2003. There were also community increases of *Shigella sonnei* biotype A in central Australia during February and March 2003. These increases were noted in Western Australia, South Australia and the Northern Territory. There were no confirmed links with food in any of the outbreaks. In Australia, the majority of shigellosis infections probably were acquired by person-to-person transmission or overseas.

Table 3. Number of locally acquired *Salmonella* Enteritidis infections in 2003, by phage type and state or territory

Phage type	ACT	NSW	QLD	SA	VIC	WA	Total
26	1		24				25
RDNC*			5	4		1	10
RDNC/12			7	1			8
6a						1	1
Untypable			1		1		2
13					1		1
21			1				1
11b						1	1
1b		1					1
21b var			1				1
4b		1					1
Total	1	2	39	5	2	3	52

* 'Reaction Does Not Conform' (RDNC) represents phage type patterns that are not yet assigned.

No cases were reported from the Northern Territory or Tasmania

Typhoid

OzFoodNet sites reported 54 cases of typhoid infection during 2003, representing an overall notification rate of 0.3 cases per 100,000 population (Figure 8). The number of notifications was similar to previous years. The highest rates were reported in Western Australia and Victoria with rates of 0.5 and 0.4 cases per 100,000 population respectively. Tasmania, the Northern Territory and the Hunter sites did not report any cases.

Travel status was unknown for six cases. Information on phage type was reported for 78 per cent (42/54) of isolates. Where travel status was known, sites reported that 78 per cent (42/54) of cases of typhoid had recently travelled overseas (Table 4). Twenty-two per cent (12/54) of these cases had recently travelled from Indonesia or Bali and the predominant phage types was D2 (6 cases). Fifteen cases had travelled to the Indian subcontinent and the predominant phage type of *S. Typhi* was degraded (5 cases).

Figure 8. Notification rates of typhoid infections for 2003 compared to mean rates for 1998 to 2002, by OzFoodNet site

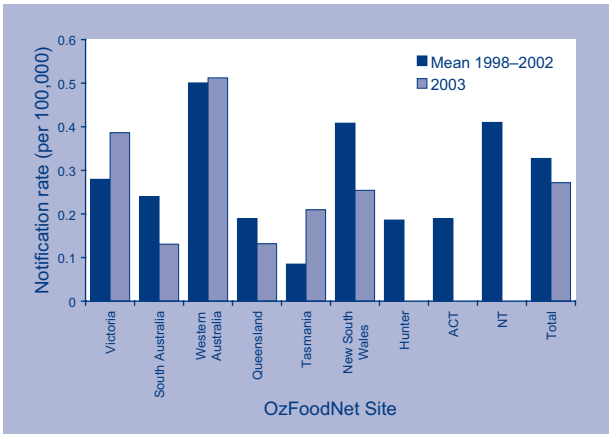


Table 4. Travel status for typhoid cases, Australia, 2003

Country	Number of cases	Phage types
Afghanistan	1	E1a (1)*
Bali	1	D2 (1)
Bangladesh	1	O (1)
India	11	E1a (1), B1var (1), Degraded (5), 1a (1), O (1), Untyped (1), Untypable (2)
Indonesia	10	D2 (5) Untypable (3), Untyped (2)
Indonesia/Singapore	1	Untyped (1)
Kenya	1	Untyped (1)
Lebanon	2	Untyped (2)
Nigeria	1	Degraded (1)
Pakistan	2	M1 (1), Untypable (1)
Philippines	3	B degraded (3)
Thailand	1	M1 (1)
United States of America	1	Degraded (1)
Asymptomatic carrier	4	C4 (1), A (1), A degraded (1), E1a (1),
Locally acquired	4	E9 (1), 40 (1), Untypable (2)
Infected by a carrier	4	C4 (4)
Unknown	6	Untyped (5) E1a (1)
Total	54	

* Numbers in parentheses represent number of cases infected by the phage type.

There were several cases of typhoid infection that were locally acquired in Australia during 2003. These occurred in Western Australia (5), Victoria (5) and New South Wales (2). This included four cases of *S.* Typhi C4 infection in Victoria that were contracted from an asymptomatic carrier who prepared food. Three infections in Western Australia were long-term carriers, while one was locally acquired and another case was suspected to have been infected in a laboratory.

Shiga toxin producing E. coli *infections*

OzFoodNet sites reported 53 cases of Shiga toxin producing *E. coli* (STEC) infection during 2003, compared to 59 for 2002 (Figure 9). This number does not include cases of haemolytic uraemic syndrome where a Shiga toxin producing *E. coli* was isolated. The notification rate of 0.3 cases per 100,000 population was a 13 per cent increase over the mean rate for previous years. South Australia (38 cases) reported the majority of cases and had the highest rate of notification of 2.5 per 100,000 population. All sites reporting cases had an increase in the number of cases notified, except for Victoria and Queensland where there were 38 per cent and 12 per cent declines respectively. There were no cases reported from Tasmania, the Australian Capital Territory or the Northern Territory during 2003. The male to female ratio of cases was 1.1:1, contrasting with a male:female ratio of 0.3:1 in 2002. In 2003, the highest rates of reported infection were in children aged 4–9 years of age.

E. coli O157 was the most common (25%) serotype isolated in 2003, compared to 34 per cent of isolates in 2002. *E. coli* O111 was the second most common and was responsible for 15 per cent of reports in 2003 (Tables 5 and 6). Almost all cases of *E. coli* O111 occurred in females in South Australia, which were notified between February and May 2003 (Figure 10). This temporal clustering of cases was related to infections occurring in an aged care facility, which was suspected to be person-to-person spread. Another cluster of *E. coli* O157 occurred around the same time in metropolitan Adelaide, although no links with any specific foods were identified. Cases of *E. coli* O157 infection were notified throughout the year. Male cases were significantly more likely than females to be recorded as 'toxin producing *E. coli* untyped' (Table 5, Odds ratio 5.3, 95% C.I. 1.4–22.9).

The majority of these untyped infections were the result of positive polymerase chain reaction (PCR) tests for the presence of toxin producing genes, but no culture of *E. coli* was obtained or specific serotype identified. In South Australia all stools containing macroscopic blood are screened for genes encoding for the production of Shiga toxins 1 and 2. Positive specimens are then tested using a multiplex PCR test for the O111, O157 and O28 serotypes, along with various virulence factors. This PCR method is highly sensitive and consequently cultures are often not obtained for further typing. Sixty three per cent (24/38) of cases in South Australia were detected by PCR and no typing details were available. (Table 6)

Figure 9. Notification rates of Shiga toxin producing *E. coli* infections for 2003 compared to mean rates for 1998 to 2002, by OzFoodNet site

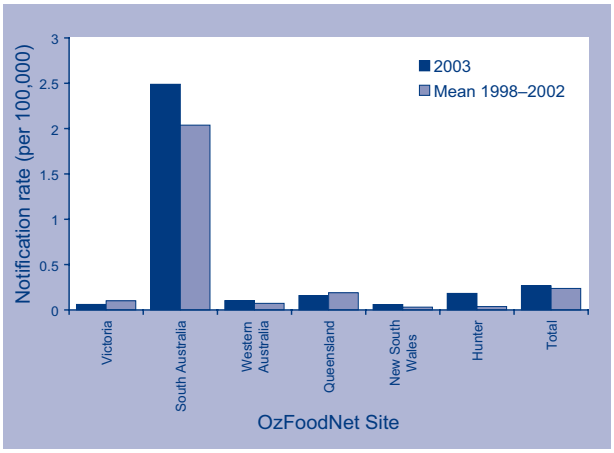
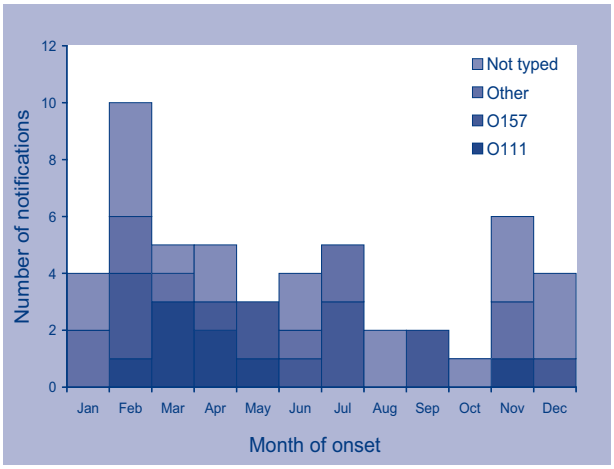


Figure does not include Tasmania, the Australian Capital Territory and the Northern Territory, as they did not report cases of Shiga toxin producing *E. coli* infections during 2003.

Figure 10. Numbers of notification of Shiga toxin producing *E. coli* infections, by month of onset and serotype, Australia, 2003



Does not include two cases, one of which was asymptomatic and another with onset of illness in late 2002.

Table 5. Number of notified cases of Shiga toxin producing *E. coli*, by sex and serotype in 2003, Australia

Serotype	Number of cases		Total
	Female	Male	
O157	8	5	13
O111	7	1	8
O130	0	1	1
O28	1	0	1
O5	0	1	1
Untypeable	1	1	2
Unknown	3	3	6
Not Typed	5	16	21
Total	25	28	53

Table 6. Number of notified cases of Shiga toxin producing *E. coli*, by State and serotype in 2003, Australia

Serotype	Number of cases					Total
	NSW	Qld	SA	Vic	WA	
O157	0	2	8	1	2	13
O111	1	0	7	0	0	8
O130	0	1	0	0	0	1
O28	0	0	0	1	0	1
O5	0	0	0	1	0	1
Untypeable	0	0	2	0	0	2
Unknown	3	3	0	0	0	6
Untyped	0	0	21	0	0	21
Total	4	6	38	3	2	53

H typing information was available for only 12 per cent (6/50) of all cases in 2003. There were three *E. coli* O157:H- infections, one each of serotypes O28:H-, O5:H- and O130:H11.

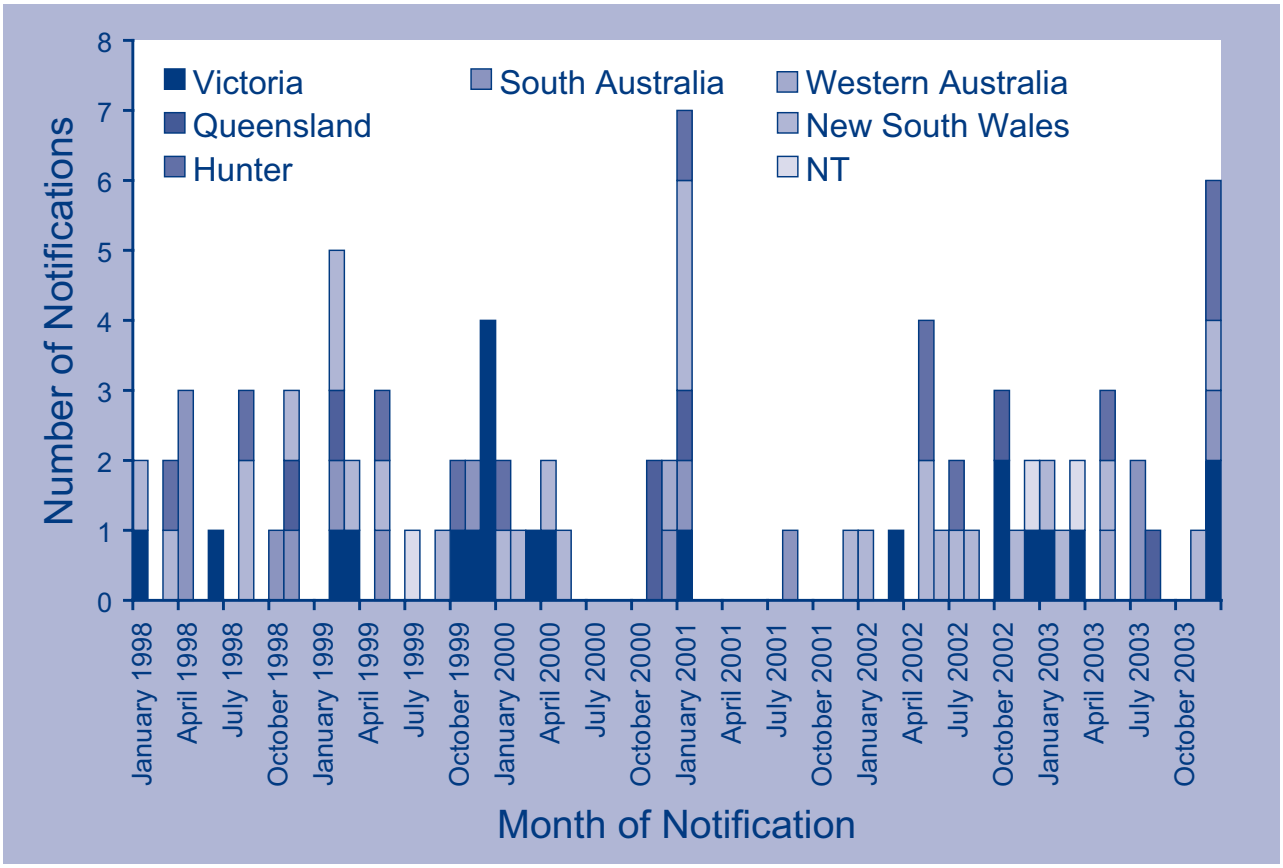
Surveillance for STEC is strongly influenced by screening practices at laboratories. South Australia has the highest rates of infection with STEC because it screens far more bloody diarrhoea specimens using sensitive PCR tests. The proportion of faecal specimens that is positive for STEC is remarkably similar between state public health laboratories in Australia regardless of the method of detection used.[12] In Australia, it is likely that many pathology laboratories do not routinely screen faeces for STEC using Sorbitol MacKonkey agar. Where this agar is used, only *E. coli* that do not ferment sorbitol are detected. In many other countries the predominant *E. coli* serotype—O157:H7—is routinely detected on this agar, although it is less common in Australia. *E. coli* that do not ferment sorbitol only represent a small proportion of this species that produce toxin-based infections.

Haemolytic uraemic syndrome

There were 15 cases of haemolytic uraemic syndrome (HUS) reported during 2003, corresponding to an overall rate of 0.1 case per 100,000 population. This compared to 13 cases of HUS in 2002. New South Wales reported five of these cases, three of which were notified in the Hunter OzFoodNet Site. Victoria reported four cases, South Australia three cases, and Western Australia, Queensland and the Northern Territory each reported a single case (Figure 11).

The male to female ratio of cases was 1:2. The highest rate of infection was in females aged 5–9 years old and males aged 0–5 years old, which were both 0.5 cases per 100,000 population. Sites reported that STEC were isolated from faeces in 20 per cent (3/15) of cases. One case was due to the O157 serotype, while two others were STEC unspecified. There was no obvious clustering of cases in 2003.

Figure 11. Numbers of notifications of haemolytic uraemic syndrome, by month of notification and jurisdiction, Australia, 1998 to 2003



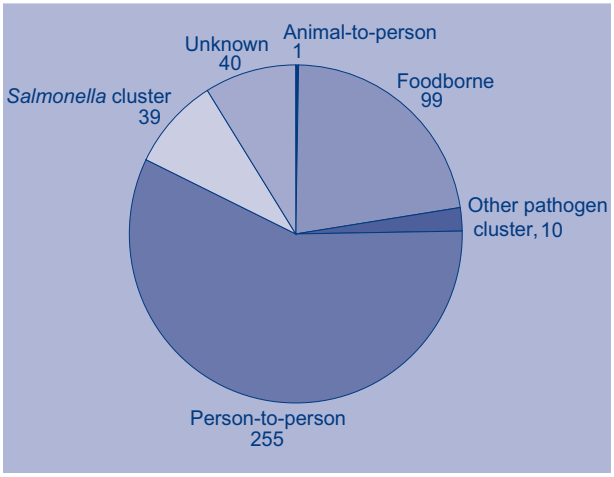
Gastrointestinal and foodborne disease outbreaks

During 2003, OzFoodNet sites reported 444 outbreaks of gastroenteritis illness affecting 10,368 persons. Five-hundred and one people were hospitalised and 10 people died as a result of these outbreaks. Fifty seven per cent (255/444) of outbreaks were suspected to be spread from infected persons to other people (Figure 12).

Outbreaks of gastroenteritis spread by person to person contact were responsible for 71 per cent (7,388/10,368) of all persons affected by gastroenteritis outbreaks, and three deaths. Fifty three per cent (135/255) of person to person gastroenteritis outbreaks were reported in aged care facilities, while 16 per cent (41/255) and 11 per cent (27/255) of outbreaks were reported in hospitals and childcare settings. Forty three per cent (109/255) of person-to-person outbreaks were due to norovirus, while 48 per cent (123/255) were of unknown aetiology many of which would have been viral.

Sites conducted investigations into 89 different clusters where the mode of transmission was not determined, or a foodborne source was not identified.

Figure 12. Outbreaks of gastrointestinal and foodborne disease, Australia, 2003



Foodborne disease outbreaks

In 2003, 99 foodborne disease outbreaks affected 1,686 persons, hospitalised 105 persons and caused six deaths (Table 7). This equates to an overall rate of 5.0 outbreaks of foodborne disease per million population. Appendix 2 shows a summary description of each outbreak.

Table 7. Outbreaks of foodborne disease in Australia, 2003, by OzFoodNet site

State	Number of outbreaks	Number affected	Hospitalised	Deaths	Mean number cases per outbreak	Outbreaks per million population
Australian Capital Territory	3	35	7	1	11.7	9.3
New South Wales	29	521	29	1	18.0	4.3
Northern Territory	7	110	4	0	15.7	35.3
Queensland	30	311	28	2	10.4	7.9
South Australia	1	6	1	0	6.0	0.7
Tasmania	1	22	2	0	22.0	2.1
Victoria	20	499	27	1	25.0	4.1
Western Australia	8	182	7	1	22.8	4.1
Total	99	1,686	105	6	17.0	5.0

Queensland reported the largest number of outbreaks, which represented 30 per cent (30/99) of all outbreaks reported (Table 7). The reporting rates of foodborne outbreaks for different OzFoodNet sites ranged from 0.7 per million population in South Australia to 35.3 per million population in the Northern Territory. The majority of outbreaks occurred in summer and autumn (Figure 13).

Aetiological agents

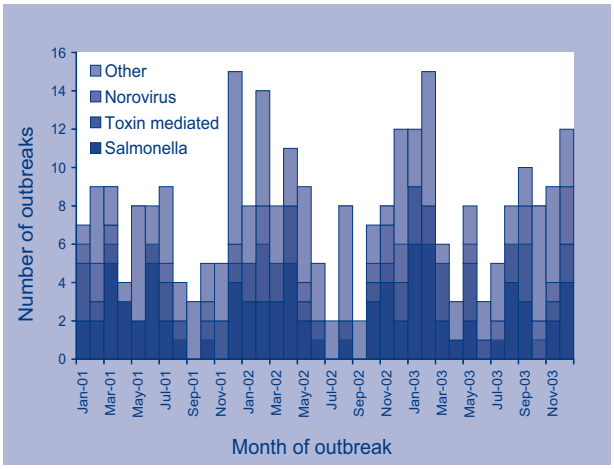
The most common agent responsible for foodborne disease outbreaks was *Salmonella*, which caused 31 per cent (31/99) of outbreaks (Table 8). These outbreaks affected a total of 710 persons with a hospitalisation rate of 12 per cent (82/710). S. Typhimurium was responsible for 81 per cent (25/31) of foodborne *Salmonella* outbreaks. Five of 6 fatalities were reported from four separate outbreaks of *S.* Typhimurium.

Many of the 21 outbreaks of illness due to toxins in 2003 were associated with contaminated fish. While ciguatera poisoning caused 48 per cent (10/21) of these outbreaks, all were small with a mean of 4.7 persons affected and a hospitalisation rate of 11 per cent. In comparison, four outbreaks of ciguatera poisoning in 2002 resulted in 50 per cent (7/14) of cases requiring admission to hospital. Histamine

Table 8. Aetiological agents responsible for foodborne disease outbreaks showing number of outbreaks and numbers of persons affected in Australia, 2003

Agent	Number of outbreaks	Number affected	Average size of outbreak	Hospitalised	Deaths
Campylobacter sp.	3	34	11.3	1	0
C. perfringens	5	116	23.2	0	1
Ciguatera	10	47	4.7	5	0
Escolar	2	23	11.5	0	0
Hepatitis A	2	24	12.0	2	0
Histamine poisoning	4	29	7.3	2	0
Norovirus	9	258	28.7	0	0
S. aureus	2	21	10.5	4	0
S. Other	6	38	6.3	4	0
S. Typhimurium	25	672	26.9	78	5
Sorbic acid	1	23	23.0	0	0
Unknown	30	401	13.4	9	0
Total	99	1,686	17.0	105	6

Figure 13. Outbreaks of foodborne disease, by selected aetiological agents, Australia, 2001 to 2003



poisoning from contaminated fish caused four outbreaks affecting 29 people. There were two outbreaks of kerriorhea due to consumption of Escolar fish, which affected 23 people. Escolar fish contain high levels of indigestible wax esters, which result in excessively oily stools. They are not considered toxins per se, but may result in outbreaks of oily diarrhoea.

There were five outbreaks *Clostridium perfringens* intoxication and two of *Staphylococcus aureus* intoxication. One outbreak of *C. perfringens* resulted in a death in a nursing home resident. There were three outbreaks of *Campylobacter* that affected 34 people.

There were eleven outbreaks of known viral aetiology, nine of which were due to norovirus. These outbreaks of norovirus affected 258 persons, but no one was hospitalised. The other outbreaks of viral illness were due to hepatitis A, which affected 24 persons. Thirty per cent (30/99) of outbreaks were of unknown aetiology, which affected 401 persons including nine cases who were hospitalised.

Food vehicles

There was a wide variety of foods implicated in outbreaks of foodborne disease during 2003 (Table 9), although investigators could not identify a vehicle for 34 per cent (34/99) of outbreaks. Contaminated fish was the most common food vehicle and was responsible for 17 per cent (17/99) of outbreaks. Poultry were responsible for or suspected as the cause of eight outbreaks, while pork was responsible for four outbreaks. Egg dishes, oysters, sandwiches, rice dishes and mixed foods were implicated in three outbreaks each. There was one outbreak associated with Vietnamese pork rolls and one associated with contaminated tahini from the Middle East. Outbreaks involving egg dishes had a hospitalisation rate of 20 per cent (23/117) and resulted in two deaths.

Outbreak settings

The most common setting for the occurrence of outbreaks was at restaurants (34%), followed by the home (20%), events catered for by professional companies (14%) and aged care facilities (9%) (Table 10). There were six outbreaks in school camps or excursions and three outbreaks each community settings and take away food stores.

Investigative methods and levels of evidence

States and territories investigated 26 outbreaks using retrospective cohort studies and nine outbreaks using case control studies. Cohort studies were conducted for only 31 per cent (8/26) outbreaks of unknown aetiology, compared to 50 per cent in 2002. Twenty-seven per cent of investigations using cohort studies were for norovirus outbreaks.

To attribute the cause of the outbreak to a specific food vehicle, investigators obtained analytical evidence from epidemiological studies for nine outbreaks. Microbiological evidence of contaminated food was found in nine outbreaks, with a further seven outbreaks investigations obtaining both microbiological and analytical evidence. Investigators obtained analytical and/or microbiological evidence for 39 per cent (12/31) of *Salmonella* outbreaks. Seventy-four per cent (74/99) of outbreaks relied on descriptive evidence to implicate a food or foodborne transmission.

Significant outbreaks

There were five outbreaks affecting 50 or more persons in 2003, compared to six in 2002. Three were due to *Salmonella* Typhimurium, one to norovirus and one was of unknown aetiology. Three of the outbreaks occurred at restaurants, one was associated with a bakery and one with a commercial caterer. One of the outbreaks of *S.* Typhimurium

occurred at a restaurant and was associated with dishes containing eggs, while another was associated with Vietnamese rolls from a bakery. The third outbreak was due to pigeon meat contaminated with with *S.* Typhimurium 99. Apple strudel served at a restaurant was responsible for a large outbreak of norovirus. The catering associated outbreak did not identify a food vehicle or aetiological agent.

There were 23 outbreaks affecting between 20 and 50 persons. Three of these outbreaks occurred in aged care facilities and were due to *C. perfringens* or *Salmonella.* Seafood was implicated in four of these outbreaks, including two due to oysters from Japan contaminated with norovirus. Nine outbreaks were due to *Salmonella* Typhimurium, of which phage types 135 (4 outbreaks) and 108 (2 outbreaks) were the most common causes. Two of these *S.* Typhimurium outbreaks were due to roast pork, while three were related to Asian foods.

The Tasmanian Department of Health and Human Services investigated an outbreak of hepatitis A following a four-day festival in the Northern Territory in April 2003. Four notifications of hepatitis A infection triggered an investigation involving OzFoodNet

Table 9. Categories of food vehicles implicated in foodborne disease outbreaks in Australia, 2003

Vehicle category	Number of outbreaks	Number affected	Hospitalised	Deaths
Beverage	1	19	0	0
Cakes	2	73	1	0
Cheese	1	23	0	0
Dessert	1	31	0	0
Egg dishes	1	52	4	0
Escolar fish	3	45	0	0
Fish (other)	14	57	7	0
Mixed vehicle	3	62	10	0
Oysters	3	100	0	0
Pasta	2	29	0	0
Pizza	1	18	0	0
Pork	4	57	3	0
Pork rolls	1	213	22	1
Poultry	5	98	8	0
Red meat/meat products	1	7	0	0
Rice dish	3	47	5	1
Sandwiches	3	38	0	0
Seafood	2	21	0	0
Sesame seed products	1	3	0	0
Unknown	34	483	18	2
Unpasteurised milk	1	13	0	0
Asian foods	2	40	3	0
Salad	2	26	1	0
Sliced meats	1	1	0	0
Suspected poultry	3	37	2	0
Suspected egg dishes	2	65	19	2
Raw vegetables	2	28	2	0
Total	99	1,686	105	6

Table 10. Categories of settings where food was prepared or consumed for foodborne disease outbreaks, Australia, 2003

Setting category	Number of outbreaks	Number affected	Hospitalised	Deaths
Aged care facility	9	167	17	3
Bakery	1	213	22	1
Community	4	35	3	0
Home	20	115	12	0
Hospital	2	22	10	1
Institution	2	49	7	0
Restaurant	34	619	25	1
School	1	19	0	0
Take away	4	67	0	0
Camp/Excursion	6	80	6	0
Caterer	14	264	3	0
Childcare	2	36	0	0
Total	99	1,686	105	6

Sites in Queensland, New South Wales, Victoria, Western Australia and Tasmania. A retrospective cohort study of 213 out of 350 people attending the event identified 21 cases of hepatitis A. People who consumed cordial or coleslaw were at higher risk of developing hepatitis A. All food handlers tested were found to be negative for hepatitis A IgM and environmental investigations did not reveal any cause of the outbreak.

There were four outbreaks involving foods imported into Australia, highlighting the international implications of foodborne disease. One outbreak of *S.* Montevideo in Victoria affected 3 persons and was linked to tahini from Lebanon. This followed a previous outbreak in New South Wales in 2002, with cases of *S.* Montevideo being reported well into 2003. In total, there were 58 cases of *S.* Montevideo from these two separate outbreaks associated with sesame seed products in New South Wales and Victoria. As a result there were several local recalls of contaminated tahini and helva along with an international alert to investigators. The international alert identified a further ten human infections in New Zealand and assisted food safety agencies in Canada and the United Kingdom to identify contaminated food products, with subsequent recalls of contaminated tahini and helva in these countries.

Salmonella contamination of sesame seed based products continues to be a problem worldwide. Australian and New Zealand food safety authorities have implemented routine microbiological testing for imported foods containing crushed sesame seeds. However, since detecting low concentrations of sporadic contamination with *Salmonella* from random testing is very difficult, human health surveillance of *Salmonella* infections plays a vital role in ensuring the safety of these types of products.

There were three other outbreaks with international implications in Western Australia[2] and Northern Territory[1] in November 2003 that were associated with oysters. The oysters were Individually Quick Frozen (IQF) meat imported from Japan, although they were different importers and brands. The labelling on some of these oyster products indicated the need to 'cook before consumption'. Despite this, the two outbreaks in Western Australia were both due to caterers using the oyster meat uncooked in 'oyster shooter' cocktails. The third outbreak in the Northern Territory occurred in a popular restaurant where the oysters were cooked for 8–10 minutes. Norovirus was detected in patients' faeces in two of these outbreaks and suspected as the cause of the third. Norovirus was also detected in the oyster meat in the outbreak that occurred in the Northern Territory. Traceback investigations identified that all oyster products were supplied by a single company in Japan and the two batches were harvested at similar times.

Cluster investigations

A cluster is defined as an increase in infections that are epidemiologically related in time, place or person where investigators are unable to implicate a food vehicle or determine a mode of transmission. An example is a temporal or geographic increase in the number of cases of a certain type of *Salmonella* serovar or phage type. In this category, some outbreaks where the mode of transmission was indeterminate have been included.

During 2003, states and territories conducted 89 investigations of clusters of enteric diseases that affected 1,298 people, hospitalising 88 people and causing one fatality. Investigators were unable to determine the mode of transmission or source of infections for these clusters, which were due to organisms such as *Salmonella*, *Campylobacter* and hepatitis A. These clusters do not include all investigations conducted at the State, Territory or public health unit level, but the number is indicative of the effort to investigate enteric diseases in Australia. Forty-four per cent (39/89) of these investigations related to clusters of *Salmonella,* which affected 427 persons with 33 cases hospitalised. *S.* Typhimurium was responsible for 36 per cent (14/39) these *Salmonella* cluster investigations. Of the remaining 25 investigations, 17 other different *Salmonella* serovars were involved.

Many of the cluster investigations were suspected to be related to animal or food-based exposures, which could not be confirmed. An example was an investigation into four cases of *S.* Reading in Queensland in February 2003. It was likely that most of the infections were acquired by zoonotic transmission, as all four cases had contact with farm animals including calves, pigs and chickens in the week prior to the onset of illness. Another example was a small household cluster of *S.* Typhimurium 108 in Tasmania suspected to be related to a cat with diarrhoea.

In April 2003, a laboratory reported a 64 per cent increase in *Campylobacter* cases in a regional area of Victoria compared to the same time period in 2002. Local government personnel interviewed 29 cases of *Campylobacter* infection, which showed that a higher proportion of cases had consumed chicken fillet and had contact with pets compared to community based controls from an earlier study. Molecular typing of 27 *Campylobacter jejuni* isolates revealed 16 different patterns, making it unlikely that cases consumed the same type or batch of food product. It was discovered that the primary pathology laboratory in the area had changed its laboratory methods for *Campylobacter* detection in late November 2002. Plates were incubated for three rather than two days, and this methodological change coincided with the increase in *Campylobacter* cases notified from December 2002 onwards. While this may not be the sole reason for the increase, as the laboratory did state that higher numbers of faecal specimens had been submitted, it may explain some of the increase.

An investigation of an apparent cluster of *S.* Enteritidis PT 21b amongst eight people in Queensland took place in early September 2003. Further investigation revealed that all of the positive specimens came from the same pathology laboratory and all except one were collected on the same day. Two of the eight cases had a history of overseas travel, which was not consistent with a single outbreak source. Hypothesis-generating interviews did not suggest any common exposures among the cases. Following discussions with the pathology laboratory, further investigation identified a laboratory error among all but one case. Results of the investigation determined that there was a single case of *S.* Enteritidis PT 21b who acquired their infection whilst travelling through Malaysia.

In early 2003, OzFoodNet continued to investigate a multi-state cluster of *S.* Potsdam involving New South Wales, the Australian Capital Territory, Victoria, South Australia and Tasmania. OzFoodNet Site epidemiologists interviewed 50 cases of *S.* Potsdam using hypothesis-generating questionnaires, although a cause for the outbreak was not determined.

In 2003, there was an increase in cases of *Salmonella* Paratyphi B Java, particularly phage type 3b var 10. In response OzFoodNet commenced a case series investigation, in collaboration with the National Enteric Pathogen Surveillance Scheme. Between May 2003 and April 2004, state and territory health departments interviewed all notified cases of *S.* Paratyphi B Java using a standard questionnaire. Cases were excluded if they were unable to be contacted by telephone or had a history of overseas travel, which is quite common amongst patients infected with *S.* Paratyphi B Java. Of the 22 case patients interviewed as part of the national cluster investigation, the median age was three years old (range 0–48 years) and the male to female ratio was 1:1.2. Eighty-two per cent (18/22) of cases reported contact with tropical fish aquariums during their incubation period. This association between *Salmonella* Paratyphi B Java infection and contact with tropical fish has been reported previously.[14,15,16] Four cases had no fish exposure but one owned a snake. All isolates of *S.* Paratyphi B Java 3b var 10 (n=10) were resistant to ampicillin, streptomycin, tetracycline, chloromycin, sulfadiazine, spectinomycin (ASTCSuSp), which is similar to the profile for *S.* Typhimurium definitive type 104.[17] The exposure histories of cases are currently being analysed to

determine risk behaviours associated with illness. It is important to for people to wash their hands after feeding fish or cleaning aquariums to avoid infection from *Salmonella* and other pathogens, such as atypical mycobacteria.

State, Territory and OzFoodNet personnel also investigated clusters of pathogens other than *Salmonella*. In South Australia, for example there was an investigation of an outbreak of campylobacteriosis following a school camp at a dairy farm. Investigation of the cluster identified both food-based and environmental risk factors, including consumption of unpasteurised milk.

The true number of clusters investigated was difficult to determine, as the figures did not include all cluster investigations conducted in public health units or local government areas. States and Territories have different definitions and triggers for investigating clusters.

Risk factors for infection

During 2003, OzFoodNet identified several important risk factors and settings for foodborne illness as a result of outbreak investigations and from preliminary results of case control studies.

Fish and Seafood

There were more outbreaks of foodborne illness related to fish and seafood in 2003 than in previous years. Sixty four per cent (14/22) of these outbreaks were from Queensland, of which 71 per cent (10/14) were outbreaks of ciguatera poisoning. All of these outbreaks of ciguatera occurred in the home, except for one that occurred in a restaurant. Preventing ciguatera intoxications relies on increased awareness to prevent people catching and eating large reef fish from reefs affected by ciguatera.

Four outbreaks of histamine poisoning were more than has been reported in previous years. New South Wales reported a small outbreak of two cases of hepatitis A associated with prawns at a restaurant meal. There were three outbreaks due to Escolar fish, one of which was histamine poisoning and possibly kerriorrhoea. All three outbreaks occurred at restaurants showing that this fish is still being sold to the food industry.[16]

The three outbreaks of gastroenteritis due to consumption of individually quick frozen oysters led to considerable concern over the safety of these products for the Australian market. Food Standards Australia New Zealand (FSANZ) has assessed that these oysters from polluted growing areas presented a high risk for outbreaks, even where the products may be cooked.[18] During the outbreaks there was considerable debate about epidemiological evidence as a basis for food recalls when complementary microbiological evidence was absent. Food safety and communicable disease agencies are still considering these issues.

Chicken and poultry

There were eight outbreaks caused or possibly caused by poultry, which was the most common vehicle following fish and seafood. *Salmonella* was the aetiological agent in five of these outbreaks, *Campylobacter* in one and two were of unknown aetiology. The largest of these outbreaks was due to *S.* Typhimurium 99 in contaminated pigeon meat served at a birthday party, which affected 61 people. Poultry may have had a role in some of the cluster investigations of salmonellosis during 2003, although the association was unable to be confirmed. Concurrent isolation of the same serotype from routine samples of raw poultry meat at the same time as a cluster investigation were common. It is important to recognise that poultry consumption is very common, with approximately 80% of people having eaten it in the previous 7 days. This makes epidemiological comparison of ill and well people's food histories very difficult. FSANZ are currently preparing a primary production standard for poultry meat in cooperation with industry and other stakeholders.

Asian foods

There were seven outbreaks of foodborne illness associated with Asian style foods in 2003 that affected a total of 355 people. Five of these outbreaks were due to *S.* Typhimurium infection, including one outbreak of *S.* Typhimurium 135 associated with Vietnamese pork rolls affecting 213 people and associated with one fatality. Vietnamese pork rolls are a particularly high-risk food, which continue to cause outbreaks despite attempts by regulatory agencies to improve the safety of these foods.[19] The other food vehicles included 'fried tofu dish', 'pigs ear salad and ducks gizzards', 'pigeon meat', 'prawns', 'rice beef and black bean sauce' and 'fried rice'. Asian foods often use imported ingredients and have shorter cooking periods than are required to kill pathogens.[20] Food safety in this sector of the restaurant industry needs to continue to be a high priority to prevent foodborne infections.

Imported foods

The four outbreaks associated with imported foods during 2003 were essentially continuation of outbreaks due to these products in 2002. This illustrated how long shelf life products can remain in the market and continue to cause disease. The outbreak of *S.* Montevideo associated with tahini in Victoria and

the follow-on from the outbreak in the Hunter from 2002 highlighted the potential for sesame-based products as a vehicle for *Salmonella*.[26] The outbreaks of illness due to the oysters highlights how oysters grown in contaminated waters may result in foodborne illness. Oysters grown in contaminated water have cause large outbreaks, including previous outbreaks associated with oysters grown in Australia and New Zealand.[21,22] Contaminated oysters have commonly caused outbreaks of gastroenteritis in Japan and approximately nine per cent are positive for norovirus using reverse transcriptase polymerase chain reaction tests.[23,24,25]

Aged care

People resident in aged care settings may be at higher risk for foodborne disease. In 2003, there were nine outbreaks in this setting compared to five in 2002. The food vehicle could not be determined in any of these outbreaks, although raw egg drink was suspected as the cause in one and gravy added into vitamised meals in another. The main reason for difficulty in determining specific foods in these outbreaks was the often poor recall of foods consumed by elderly residents and the lack of accurate menus and dietary histories maintained by the facility. Four outbreaks in aged care settings were due to *C. perfringens* and three due to *Salmonella*. The *C. perfringens* outbreaks clearly indicate the need for better control of preparation and handling of foods in this sector.

Restaurants and catered events

Outbreaks due to this sector constituted 48 per cent (48/99) of outbreaks, compared to 54 per cent in 2002. The two most common aetiological agents *Salmonella* and norovirus were responsible for 21 per cent (10/48) and 17 per cent (8/48) of outbreaks respectively, although outbreaks of unknown aetiology were most common (42 per cent). It is likely that many outbreaks of unknown aetiology are actually due to norovirus. Staff members who handle foods should not work when they are ill, as they can cause large outbreaks of gastroenteritis when food becomes a fomite for enteric pathogens. Many outbreaks in restaurant and catering settings result from breaches in food safety that could be prevented by proper application of food safety programs. Clearly there is a need to continue to monitor the causes of outbreaks in this sector.

Camps and excursions

There were six outbreaks of foodborne illness associated with camps or excursions during 2003. There were a range of pathogens responsible for these outbreaks, two of which were either toxin related or suspected toxins. These outbreaks point to the potential for poor temperature control when large quantities of food are prepared.

There was one outbreak of campylobacteriosis due to drinking unpasteurised milk in Victoria. There was also another outbreak of campylobacteriosis in South Australia associated with either drinking unpasteurised milk or swimming. Unpasteurised milk is a high risk food for contamination with organisms spread from animal-to-person, such as *E. coli*, *Cryptosporidium*, *Campylobacter* and *Salmonella*.[27] While the sale of unpasteurised milk is now prohibited in all states and territories, this does not prevent people drinking this product in settings such as school camps. Camps and excursions can present a higher risk for waterborne illness where water supplies are inadequately treated.

Surveillance evaluation and enhancement

Continuous monitoring and improvement of surveillance systems is important to ensure that outbreaks of foodborne illness are investigated rapidly and effectively. To facilitate improvements in surveillance and investigative procedures, outcome indicators have been compared at OzFoodNet sentinel sites.

National information sharing

In 2003, all jurisdictions contributed to a fortnightly national cluster report to identify foodborne illness that was occurring across state and territory boundaries. The cluster report was useful for identifying common events affecting different parts of Australia. The cluster report is useful for tracking the investigation of multi-state clusters, such as hepatitis A and norovirus infections associated with oysters. The cluster report supplemented information sharing on a closed list server, teleconferences and at quarterly face-to-face meetings.

Outbreak reporting and investigation

During 2003, the Northern Territory Site recorded the highest reporting rate of outbreaks of foodborne disease (35.3 per 100,000 population) and foodborne salmonellosis (5.0 per 100,000 population). The rates of other Sites reporting foodborne *Salmonella* outbreaks ranged between 0–3.1 outbreaks per million population. Queensland investigated the largest number of foodborne disease outbreaks (30 outbreaks; 7.9 per million population).

Figure 14. Proportion of *Salmonella* notifications on State and Territory databases with appropriate information, by year, 2000 to 2003

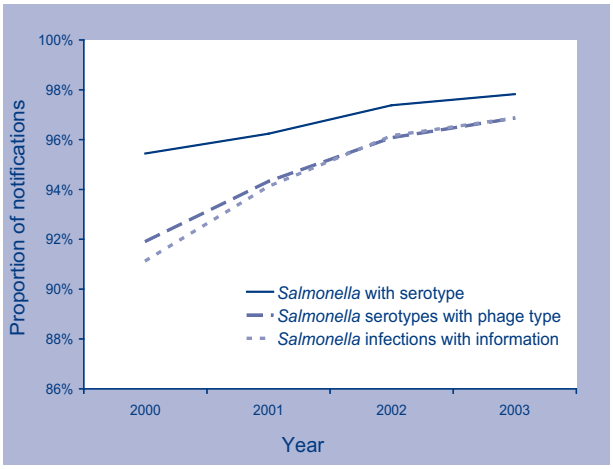
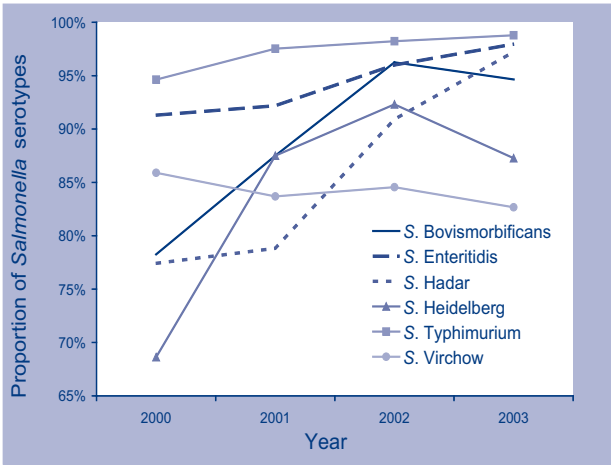


Figure 15. Proportion of *Salmonella* serotype notifications on State and Territory databases with phage type information, by serotype and year, 2000 to 2003



States and territories conducted 51 analytical studies (cohort or case control studies) to investigate foodborne disease outbreaks or clusters of suspected foodborne illness. Investigators used analytical studies for 31% (31/99) of foodborne disease outbreaks, which was slightly lower than 2002 (40%). The Northern Territory had the highest rate for investigations of foodborne disease or potentially foodborne clusters using analytical studies, followed by the Australian Capital Territory.

Completeness of Salmonella *serotype and phage type reports*

There was considerable improvement in the completeness of *Salmonella* data available on state and territory surveillance databases between the years 2000 to 2003 (Figure 14). Overall 97 per cent (6,740/6,952) of *Salmonella* notifications on databases contained either serotype or phage type, which was an increase of 2.4 per cent from 2001 and 0.7 per cent from 2002 (Table 11).

Queensland had the highest proportion of complete *Salmonella* notification (99.7%), while four sites reported 98 per cent or higher. New South Wales reported the lowest rate of completeness, but recorded an 11.8 per cent improvement when compared to the figures for 2000. The majority of missing records on the New South Wales database related to phage typing information, particularly for the Virchow serotype. Phage type recording for serotypes Virchow and Heidelberg, with only 82.7 per cent (291/352) and 87.3 per cent (48/55) of reports on databases recording this information respectively (Figure 15). From 2002 to 2003, recording phage types of serotype Bovismorbificans declined by 1.7 per cent. Phage type reports on state and territory databases were increased for all other serotypes.

It is important to recognise that this information on completeness does not reflect the practices of reference laboratories that serotype and phage type *Salmonella* in Australia. The information on completeness presented here mostly relates to the recording practices of state and territory health departments on surveillance databases.

Discussion

Approximately 5.4 million (credible interval 4.0–6.9 million) people experience foodborne illness each year in Australia.[6] The infections and outbreaks documented in this report represent only a small proportion of the total burden of these illnesses, as the majority of cases are never reported to health departments.[3] In 2003, both notifications of potentially foodborne infections and outbreaks of foodborne disease were higher than historical means. Reports of listeriosis and Shiga toxin producing *E. coli* were 17 per cent and 16 per cent higher than the mean reports for the previous five years respectively. These rates of infection are similar to many other developed countries.[28]

Rates of reported *Salmonella* and *Campylobacter* infections were considerably higher in Australia than

Table 11. Number of *Salmonella* infections notified and proportion of notifications with serotype and phage type information available on surveillance databases in Australia, 2000 to 2003, by OzFoodNet site

OzFoodNet Site	Year	*Salmonella* notifications	*Salmonella* with serotype	Serotype with phage type information	Salmonella infections with information
Australian Capital Territory	2000	102	96.1	98.5	95.1
	2001	76	98.7	100.0	98.7
	2002	96	97.9	93.8	92.7
	2003	80	98.8	98.3	96.3
Hunter	2000	86	94.2	87.8	87.2
	2001	117	97.4	92.7	92.3
	2002	170	95.9	96.6	94.1
	2003	112	98.2	93.9	94.6
New South Wales	2000	1334	92.2	78.1	78.9
	2001	1668	94.2	86.4	85.4
	2002	2078	94.8	88.9	88.0
	2003	1868	96.4	90.6	90.7
Northern Territory	2000	329	90.9	82.5	88.8
	2001	387	90.4	98.4	90.2
	2002	337	96.7	100.0	96.7
	2003	371	98.7	100.0	98.7
Queensland	2000	1818	97.3	94.8	95.0
	2001	2169	97.0	95.0	97.8
	2002	2722	97.5	98.5	99.4
	2003	2255	97.8	99.4	99.7
South Australia	2000	452	99.6	100.0	99.6
	2001	613	99.8	100.0	99.8
	2002	520	100.0	100.0	100.0
	2003	441	99.3	100.0	99.3
Tasmania	2000	127	96.9	97.9	96.1
	2001	159	98.7	97.1	98.1
	2002	165	99.4	100.0	99.4
	2003	138	97.8	100.0	97.8
Victoria	2000	1005	98.0	99.3	97.5
	2001	1090	97.7	100.0	97.7
	2002	1208	99.3	100.0	99.3
	2003	1267	99.3	100.0	99.3
Western Australia	2000	936	93.2	93.6	89.9
	2001	850	95.9	95.2	93.8
	2002	729	99.3	99.2	98.9
	2003	612	97.6	99.2	97.2
Australia	2000	6103	95.4	91.9	91.1
	2001	7012	96.2	94.3	94.1
	2002	7855	97.4	96.1	96.2
	2003	7032	97.8	96.9	96.9

in the United States of America (USA). This was despite the use of active surveillance to ascertain cases in the USA. The ratio of reported *Salmonella* and *Campylobacter* rates for OzFoodNet Sites and FoodNet Sites in 2003 were 2.4 (35.4/14.5) and 8.9 (112.0/12.6) times higher respectively.[29] Even after adjusting for the likelihood of people attending a doctor or having a stool specimen tested, these differences still persist.[30] The likely reasons for these international variations include differences in laboratory testing procedures, and different levels of exposures to these organisms in the general community.

Outbreaks of STEC are rarely identified in Australia even where screening for this organism is intense.[6] South Australia identified two clusters of toxigenic *E. coli* in 2003, although a specific food vehicle was not identified in either outbreak. There is considerable variation in rates of STEC infection in Australian states and territories, which primarily relates to the rate of screening stools for this organism. The proportion of diarrhoeal stool tested that is subsequently reported as positive in reference laboratories is relatively consistent around Australia, and ranges from 1.2–6.9 per cent.[12]

Salmonella caused the most foodborne outbreaks of any agent during 2003 similar to previous years. Foodborne *Salmonella* infections are a serious concern for Australia along with many other countries. In 2003, there were at least an equal number of investigations of *Salmonella* where no food vehicle was identified. For every case of salmonellosis reported to Australian surveillance systems there may be between 5–25 cases in the community.[31] Improved diagnostic tests have also shown the importance of norovirus as a cause of foodborne illness. In this report, norovirus was also identified as a significant cause of outbreaks that were spread from person-to-person. These outbreaks result in considerable costs to the aged care and healthcare sectors, as they cause significant illnesses in staff and patients and can result in ward or facility closures.

There were six deaths associated with foodborne outbreaks in Australia in 2003. This was higher than previous years, although small numbers make comparison with other years unreliable. Four deaths occurred in aged care or hospital settings, while two occurred in outbreaks in community settings. Four deaths occurred in outbreaks of *Salmonella* Typhimurium 135, while another occurred in an outbreak of *Salmonella* Typhimurium 170. The remaining outbreak where a death occurred in a nursing home was due to *Clostridium perfringens* and was believed to be associated with blended food. Approximately 42 per cent of outbreaks were associated with restaurants and caterers. The hospitalisation rate was highest in outbreaks in hospital and aged care settings.

Seafood and fish were the cause of several outbreaks during 2003. Seafood is a major cause of foodborne illness globally, although the total amount in Australia is difficult to estimate.[29] There have been six different norovirus or suspected viral outbreaks associated with imported Japanese Individually Quick Frozen (IQF) oyster meat in Australia in the last two years. New Zealand investigators have also identified Korean IQF oyster meat to be contaminated with norovirus (Pers Comm. G Simmons, July 2004). This highlights increasing concerns about seafood safety and the global distribution of foods that may cause widespread illness.[29]

In 2003, the outbreak of *Salmonella* Montevideo linked to sesame seed products continued from 2002. Numerous associated products were positive for *S.* Montevideo, which resulted in international alerts. These sesame seed products had very low concentrations of *Salmonella*, which might not have been detected using conventional microbiology. Following the outbreak in 2002, the Australian Quarantine and Inspection Service added tahini to the risk list where all imported products are tested. New Zealand elevated the risk category of sesame-based products following an outbreak in 2003 that was detected through the international alert about the Australian outbreak.

It is important to recognise some of the many limitations of the data that OzFoodNet report. Surveillance data are inherently biased and require careful interpretation. These biases include: the higher likelihood that certain population groups will be tested, and different testing regimes in different states and territories, resulting in different rates of disease. Some of the numbers of notifications are small, as are populations in some jurisdictions. This can make rates of notification unstable and meaningful interpretation difficult. Importantly, some of the most common enteric pathogens are not notifiable, particularly norovirus and enteropathogenic *E. coli.* There are many causes of illness that do not result in outbreaks, particularly for organisms such as *Campylobacter*. There can also be considerable variation in assigning causes to outbreaks depending on investigators and circumstances.

Health agencies conducting surveillance for foodborne disease need to constantly improve their practices and evaluate their efforts. The large number of analytical studies used in investigations of outbreaks is evidence of robust inquiry into the causes of these diseases. During 2003, OzFoodNet coordinated or participated in the investigation

of several multi-state outbreaks. The success of OzFoodNet is a testimony to the value of regular and informed communication.

The data arising from OzFoodNet's assessment of foodborne disease risks need to feed into the development of food safety policy for Australia. While many risk factors occur commonly from year-to-year, they require constant vigilance. The occurrence of repeated outbreaks with similar food vehicles or settings of preparation may indicate the need for enforcement of controls. National surveillance of foodbourne diseases is vitally important to provide data to evaluate these efforts. OzfoodNet needs to prioritise work for coming years to identify potential gaps in food safety and measure the impact on food safety of interventions in the national food safety work program.

Acknowledgements

This report is based on the work of epidemiologists in each of the eight OzFoodNet sites during 2003: Rosie Ashbolt, Karen Dempsey, Joy Gregory, Karin Lalor, Geetha Isaac-Toua, Geoff Millard, Jennie Musto, Leonie Neville, Jane Raupach, Mohinder Sarna, Russell Stafford, Marshall Tuck and Leanne Unicomb. It also represents the work of Gillian Hall from the National Centre for Epidemiology and Population Health, Martyn Kirk, Christopher Kenna, Janet Li and Rhonda Owen from the Australian Government Department of Health and Ageing. Epidemiologists, project officers, interviewers and research assistants at each of the sites contributed to this report, including: Robert Bell, Christine Carson, Barry Combs, Dot Little, Tony Merritt, Lillian Mwanri, and Cameron Sault.

We would like to thank the many people who assisted OzFoodNet in our work during 2003, particularly our international colleagues from the United States of America, Canada, the United Kingdom, Ireland, New Zealand and the World Health Organization. We would also like to thank members of the Communicable Disease Network Australia, the Public Health Laboratory Network, the Masters of Applied Epidemiology Program, and the Australian *Campylobacter* subtyping network.

We also acknowledge the hard work of various public health professionals and laboratory staff around Australia who interviewed patients, tested specimens and investigated outbreaks. The high quality of their work is the foundation of this report.

The OzFoodNet initiative is funded by the Australian Government Department of Health and Ageing.

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Appendices

Appendix 1. Summary of gastrointestinal infections notified to OzFoodNet sites potentially due to food, 2003

		ACT	Hunter	NSW	NT	Qld	SA	Tas	WA	Vic	Total
Campylobacter	Cases	413	NN	NN	274	3,886	2,661	618	1,959	5,653	15,464
	Rate	127.9	NN	NN	138.1	102.4	174.2	129.5	100.3	115.0	112.0
Salmonella	Cases	80	112	1,868	371	2,255	441	138	612	1,267	7,032
	Rate	24.8	20.4	27.9	187.1	59.4	28.9	28.9	31.3	25.8	35.4
Yersinia	Cases	2	NN	NN	1	94	18	0	2	0	117
	Rate	0.6	NN	NN	0.5	2.5	1.2	0.0	0.1	0.0	1.7
STEC	Cases	0	1	4	0	6	38	0	2	3	53
	Rate	0.0	0.2	0.1	0.0	0.2	2.5	0.0	0.1	0.1	0.3
HUS	Cases	0	3	5	1	1	3	0	1	4	15
	Rate	0.0	0.3	0.1	0.2	0.0	0.1	0.0	0.0	0.1	0.1
Typhoid	Cases	0	0	17	0	5	2	1	10	19	54
	Rate	0.0	0.0	0.3	0.0	0.1	0.1	0.2	0.5	0.4	0.3
Shigella	Cases	3	NR	58	132	55	32	4	109	50	443
	Rate	0.9	NR	0.9	66.6	1.4	2.1	0.8	5.6	1.0	2.2
Listeria	Cases	1	3	28	0	11	1	2	8	21	72
	Rate	0.3	0.5	0.4	0.0	0.3	0.1	0.4	0.4	0.4	0.4

NN Not notifiable.

NR Not reported.

Appendix 2. Summary of foodborne disease outbreaks reported by OzFoodNet sites, 2003

State	Month of outbreak	Setting Category	Agent category	Number affected	Hospitalised	Evidence *	Epidemiolgical study†	Responsible vehicles
Australian Capital Territory	Apr	Home	Unknown	3	0	D	D	Fish
	Apr	Hospital	*S.* Typhimurium	19	7	D	D	Unknown
	Nov	Childcare	Unknown	13	0	D	D	Vegetable pasta salad
New South Wales	Jan	Restaurant	*S.* Other	4	1	D	D	Pork dish
	Jan	Restaurant	*S.* Typhimurium	3	0	D	D	Chicken
	Jan	Restaurant	*Campylobacter*	2	1	D	D	Caesar salad
	Jan	Restaurant	Unknown	3	1	D	D	Unknown
	Feb	Camp/Excursion	*Campylobacter*	19	0	D	D	Chicken
	Feb	Restaurant	*S.* Typhimurium	11	1	M	D	Rice salad
	Feb	Restaurant	Hepatitis A	2	0	D	D	Prawns
	Mar	Home	Histamine poisoning	2	2	D	D	Sardines
	Mar	Take away	*S.* Typhimurium	12	0	M	D	Chicken
	Mar	Restaurant	Unknown	3	0	D	D	Fried rice
	May	Restaurant	Norovirus	67	0	A	CCS	Apple strudel
	May	Restaurant	Unknown	24	0	D	D	Suspected salad
	May	Restaurant	*S.* Typhimurium	61	5	M	C	Pigeon meat
	Jul	Home	Unknown	1	0	D	N	Soccerball ham
	Aug	Take away	*S.* Typhimurium	20	0	M	N	Pigs ear salad, ducks gizzards
	Aug	Restaurant	*S.* Typhimurium	20	3	D	CCS	Tofu dish
	Aug	Restaurant	Unknown	4	0	D	D	Unknown
	Sep	Community	*S.* Typhimurium	20	2	D	D	Suspected chicken/eggs
	Sep	Restaurant	Unknown	4	0	D	D	Unknown
	Sep	Institution	S. Typhimurium	20	1	D	N	Unknown
	Oct	Caterer	Unknown	23	2	D	D	Unknown

State	Month of outbreak	Setting Category	Agent category	Number affected	Hospitalised	Evidence *	Epidemiolgical study†	Responsible vehicles
New South Wales *continued*	Oct	Caterer	Unknown	19	0	D	N	Unknown
	Oct	Caterer	Unknown	78	1	D	CCS	Unknown
	Oct	Restaurant	Unknown	6	0	D	N	Unknown
	Nov	School	*S.* Typhimurium	19	0	AM	CCS	Cordial like drink
	Nov	Hospital	Unknown	3	3	D	N	Chicken Schnitzel
	Nov	Restaurant	*S.* Typhimurium	33	4	AM	CCS	Fried rice
	Dec	Home	Unknown	13	2	D	D	Unknown
	Dec	Restaurant	Unknown	25	0	D	CCS	Unknown
Northern Territory	Feb	Take away	*S.* Typhimurium	7	nil	D	D	Suspected roast turkey
	Feb	Caterer	Unknown	11	nil	D	C	Unknown
	Aug	Home	Unknown	18	0	D	C	Pizza
	Aug	Camp/Excursion	*S. aureus*	5	4	D	D	Rice, beef and black been sauce
	Oct	Caterer	Norovirus	11	0	D	C	Curried egg sandwich
	Nov	Caterer	Unknown	10	0	D	C	Suspected quail
	Dec	Restaurant	Norovirus	48	0	AM	C	Japanese IQF oysters
Queensland	Jan	Home	Ciguatera	2	0	D	D	Coral Trout
	Jan	Restaurant	Histamine poisoning	3	0	M	D	Dolphin Fish
	Jan	Home	Ciguatera	3	0	D	D	Mackerel Steaks
	Jan	Aged care facility	S. Other	2	0	D	D	Unknown
	Jan	Restaurant	S. Typhimurium	5	0	D	D	Unknown
	Jan	Restaurant	Unknown	6	0	D	D	Unknown
	Feb	Aged care facility	S. Other	2	1	D	D	Unknown
	Feb	Restaurant	Unknown	7	0	D	D	Beef Burgundy
	Feb	Caterer	S. aureus	16	0	M	D	Pasta Salad
	Feb	Home	Ciguatera	7	0	D	D	Coral Trout

State	Month of outbreak	Setting Category	Agent category	Number affected	Hospitalised	Evidence *	Epidemiolgical study†	Responsible vehicles
Queensland *continued*	Mar	Home	Histamine poisoning	2	0	D	D	Tuna Patties
	Mar	Home	Ciguatera	3	0	D	D	Fish (Mooloolaba Bay)
	May	Restaurant	*S.* Other	21	2	D	CCS	Roast Pork
	May	Home	*C. perfringens*	19	0	AM	C	Curried Prawns Dish
	May	Childcare	Sorbic acid	23	0	M	N	Cheese
	May	Home	Ciguatera	2	0	D	D	Cod Fish Heads
	May	Home	Ciguatera	3	0	D	D	Giant Trevally Fish
	Jul	Restaurant	Norovirus	31	0	A	C	Trifle
	Aug	Home	Ciguatera	5	5	D	N	Barracuda (Sphyraena spp.)
	Sep	Camp/Excursion	Norovirus	11	0	D	D	Unknown
	Sep	Home	*S.* Typhimurium	7	0	D	D	Unknown
	Sep	Caterer	Norovirus	13	0	D	C	Unknown
	Oct	Restaurant	Ciguatera	15	0	D	D	Spanish mackerel
	Oct	Restaurant	Unknown	5	0	D	D	Unknown
	Nov	Home	Ciguatera	3	0	D	D	Fish head soup - Red Emperor
	Dec	Restaurant	Escolar	20	0	D	D	Escolar Fish
	Dec	Home	Ciguatera	4	0	D	D	Fish species unknown
	Dec	Aged care facility	*S.* Typhimurium	47	16	D	D	Suspect raw egg
	Dec	Restaurant	S. Typhimurium	18	3	D	CCS	Suspect sauces based on raw eggs
	Dec	Home	S. Typhimurium	6	1	D	D	Unknown
South Australia	Aug	Community	S. Typhimurium	6	1	A	CCS	Cold set Cheesecake
Tasmania	Jun	Camp/Excursion	Hepatitis A	22	2	A	C	Coleslaw
Victoria	Jan	Bakery	S. Typhimurium	213	22	M	D	Pork Rolls
	Feb	Restaurant	Escolar	3	0	D	D	Escolar Fish
	Feb	Camp/Excursion	Unknown	10	0	D	D	Unknown

State	Month of outbreak	Setting Category	Agent category	Number affected	Hospitalised	Evidence *	Epidemiolgical study†	Responsible vehicles
Victoria *continued*	Feb	Home	*S.* Typhimurium	4	1	D	C	Unknown
	Feb	Caterer	*S.* Typhimurium	20	0	AM	C	Roast Pork
	Feb	Caterer	*S.* Typhimurium	12	0	AM	C	Roast Pork
	Apr	Aged care facility	Unknown	14	0	D	D	Unknown
	Jun	Aged care facility	*C. perfringens*	12	0	D	D	Unknown
	Jul	Community	S. Other	6	0	D	D	Suspect cucumbers
	Jul	Aged care facility	Unknown	5	0	D	D	Unknown
	Jul	Caterer	Unknown	7	0	D	C	Unknown
	Aug	Community	*S.* Other	3	0	D	D	Tahini
	Sep	Aged care facility	*C. perfringens*	28	0	D	D	Unknown
	Sep	Aged care facility	*C. perfringens*	15	0	D	D	Unknown
	Sep	Restaurant	Unknown	14	0	D	D	Unknown
	Oct	Take away	Unknown	28	0	A	C	Vegetables and Chilli dish
	Nov	Camp/Excursion	*Campylobacter*	13	0	D	C	Unpasteurised milk/animal contact
	Dec	Restaurant	Norovirus	18	0	D	C	Unknown
	Dec	Restaurant	*S.* Typhimurium	52	4	A	C	Raw egg dishes
	Dec	Restaurant	Histamine poisoning	22	0	AM	C	Escolar Fish
Western Australia	Jan	Home	*S.* Typhimurium	8	1	D	D	Unknown
	Feb	Caterer	Unknown	17	0	A	C	Japanese IQF oysters
	Mar	Institution	*S.* Typhimurium	29	6	M	C	Mixed foods
	Jun	Caterer	Unknown	10	0	D	C	Sandwiches
	Sep	Aged care facility	C. perfringens	42	DK	A	C	Suspected gravy
	Nov	Caterer	Unknown	17	0	D	C	Club sandwiches
	Nov	Restaurant	Norovirus	35	0	A	C	Japanese IQF oysters
	Dec	Restaurant	Norovirus	24	0	D	C	Unknown

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

† C=cohort study; CCS=case control study; D=descriptive study; N=individual patient data not collected.

Laboratory surveillance of Shiga toxin producing *Escherichia coli* in South Australia and the Hunter Health Area, New South Wales, Australia

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Abstract

To estimate the prevalence of Shiga toxin producing *Escherichia coli* in Australia, bloody stool samples from two Australian locations were screened for the presence of Shiga toxin genes, *stx1* and *stx2*. Four of 126 (3.2%) and 139 of 5,829 (2.4%) patients from the two locations had a positive polymerase chain reaction for Shiga toxin genes. *Commun Dis Intell* 2004;28:390–391.

Keywords: Shiga toxin, Escherichia coli*, Australia*

Shiga toxin producing *Escherichia coli* (STEC) is the commonest cause of post-diarrhoeal hemolytic uraemic syndrome (HUS) in industrialised countries including Australia.[1] The majority of STEC infections are acquired by humans via the food chain, particularly from contaminated meat sources.[2] In order to develop strategies to prevent acquisition of STEC, it is important to understand the prevalence of the organism in humans with diarrhoea.

This summary reports data on screening of stool samples for STEC toxin genes, *stx1* and *stx2,* from two Australian locations, South Australia and the Hunter Health Area of New South Wales, between January 1999 and December 2002. The Institute of Medical and Veterinary Science (IMVS) in South Australia screened all stools, including those referred from diagnostic laboratories, with macroscopic evidence of blood, or from patients with a clinical history of bloody diarrhoea, or when a sample was accompanied by a request from a physician. This included screening stools from HUS cases. In the Hunter, screening was undertaken on stool samples submitted to the Hunter Area Pathology Service (HAPS), with profuse red blood cells, from which *E. coli* but no other bacterial diarrhoeal pathogen was detected. Stool samples from HUS cases were also screened.

All samples were screened for STEC toxin genes using multiplex real-time TaqMan™ polymerase chain reaction (PCR) (Applied Biosystems). Controls used for each assay run included *stx1* and *stx2* positive *E. coli* O111, and O157 and an *stx* negative *E. coli* ATCC 25922 at HAPS, and *stx1* and *stx2* positive *E. coli* O157 and *stx* negative *E. coli* ATCC 25922 at IMVS.

During the study period, four of 126 patients from the Hunter Health Area were positive for STEC toxin genes (3.2%) of which two were positive for *stx2*, one for *stx1* and one for both genes. Positives were detected in 1999 and 2002 only. In South Australia, 139 of 5,829 patients were positive for STEC toxin genes (2.4%), 42 were positive for *stx1*, 37 for *stx2* and 60 were positive for both genes (Table). STEC positives were detected in each year of the study period.

The two cases of HUS positive for STEC reported in the Hunter Health Area included a 26-month-old male and a 62-year-old male, who was also diagnosed with *Campylobacter*. In South Australia, the three HUS patients positive for STEC comprised a 10-month-old male, a 2-year-old female, and a

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Table. Summary of Shiga toxin producing *Escherichia coli* screening of stool samples from South Australia and from the Hunter Health Area of New South Wales, Australia, between 1999 and 2002

Location	Patients tested*	Number positive for *stx1* and/or *stx2* genes	Percentage positive	Number with HUS†
South Australia	5,829	139‡	2.4	3§
Hunter	126	4	3.2	2

* Excludes repeat samples collected within 14 days.

† HUS: hemolytic uraemic syndrome.

‡ A further 2 Shiga toxin producing *Escherichia coli* cases were detected in 1999 by culture only.

§ There were three additional HUS cases in this period that were not positive for STEC.

21-year-old male. The HUS patients appeared to be sporadic cases as there was no observed link between them.

During the study period, South Australia and the Hunter using the same PCR method, found 2.4 to 3.2 per cent of bloody stools positive for STEC toxin genes, respectively, suggesting that other regions in Australia may have similar levels of STEC infection. These rates are in line with those detected in the United States of America, with studies reporting prevalence of 4.2 per cent (14/335)[3] and 2.1 per cent (39/1,851)[4] using ELISA toxin testing and PCR, respectively. In Thailand one per cent (2/211) of samples tested were positive for STEC by culture followed by PCR.[5]

Acknowledgements

This work in the Hunter Health Area was funded by OzFoodNet, enhanced surveillance program of the Australian Government Department of Health and Ageing.

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Enhanced surveillance of acute hepatitis B in south-eastern Sydney

Roslyn G Poulos,[1,2] Mark J Ferson[1,2]

Abstract

Hepatitis B is a notifiable condition in all Australian states and territories. Medical practitioners and health facilities are required to report episodes of acute disease, while laboratories must notify on positive serological results. In New South Wales laboratories are required to report only the presence of hepatitis B surface antigen (HBsAg). Without clinical information, laboratory reporting of HBsAg fails to distinguish between acute infection and chronic carriage. Since practitioner under reporting is well recognised, surveillance data are likely to underestimate the true incidence of acute clinical infection. Two retrospective reviews of an enhanced surveillance system to improve the identification of acute hepatitis B in south-eastern Sydney are presented. Over a 6-month period, the enhanced surveillance system increased the identification of acute cases by at least threefold. Over a 5-year period, medical practitioners or hospitals reported only 25 per cent of acute disease, the remainder being initially notified by laboratories. Approximately half of the laboratory notifications contained only HBsAg results. The availability of clinical notes, liver enzyme or IgM to core antigen results assisted the public health unit in the identification of possible acute disease. This system of enhanced surveillance has proven to be sustainable, with minimal resources required. We suggest that sentinel enhanced surveillance systems in a sample of New South Wales public health units would be an effective and efficient method to improve the surveillance of acute hepatitis B, and that laboratories be required to report IgM to core antigen, if available, when notifying a positive HBsAg result. *Commun Dis Intell* 2004;28:392–395.

Keywords: acute hepatitis B; surveillance; hepatitis serology

Introduction

Hepatitis B is a notifiable condition in all Australian states and territories. Medical practitioners and health facilities are required to report episodes of acute disease, while laboratories must notify on positive serological results. Under established practice in New South Wales, the only serological result required from laboratories is that of hepatitis B surface antigen (HBsAg).

Surveillance definitions in New South Wales classify cases of hepatitis B as acute (presumptive or confirmed), chronic or unspecified.[1]

A presumptive acute case has a positive HBsAg and clinical symptoms and signs of acute viral hepatitis where other causes of acute hepatitis have been excluded. A confirmed acute case is a presumptive case where IgM to core antigen is also positive.

Chronic carriers are cases with documented HBsAg in two blood samples collected at least six months apart. Cases for whom no further information, other than a single positive hepatitis B surface antigen is reported, are considered as unspecified. Under New South Wales guidelines, a public health response is required for cases of acute infection, but follow-up of chronic and unspecified cases is at the discretion of the public health unit director.

In the absence of clinical information, laboratory reporting of HBsAg fails to distinguish between acute infection and chronic carriage. Practitioner under reporting, however, is well recognised both in New South Wales[2] and overseas.[3] Therefore, surveillance data are likely to underestimate the true incidence of acute clinical infection. To improve the surveillance of acute hepatitis B in south-eastern Sydney, an enhanced surveillance system has been in place since 1992.[2]

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Methods

All laboratory reports of positive HBsAg in south-eastern Sydney residents are triaged by the author (MJF) on the basis of clinical notes, the detection of IgM to core antigen or liver enzyme results to determine if the case can be categorised as acute or chronic. Reports of chronic disease receive no further follow-up by the public health unit. Reports of possible acute disease (based on one or more of the following: positive IgM to core antigen; clinical notes on laboratory request form which indicate acute disease; raised alanine aminotransferase, aspartate aminotransferase or both) are followed-up by a telephone call from a public health nurse, to the referring doctor, to confirm the diagnosis and onset of acute infection and collect risk factor information. For reports which cannot be categorised, a form letter with a reply paid return address is sent to the referring doctor asking whether the case was thought to be acute or chronic, based on the occurrence of jaundice, dark urine or markedly elevated liver enzyme levels. If the diagnosis was considered acute, the date of onset is sought. Where the diagnosis is returned as acute, the referring doctor is then contacted by telephone, and risk factor information is collected (Figure 1).

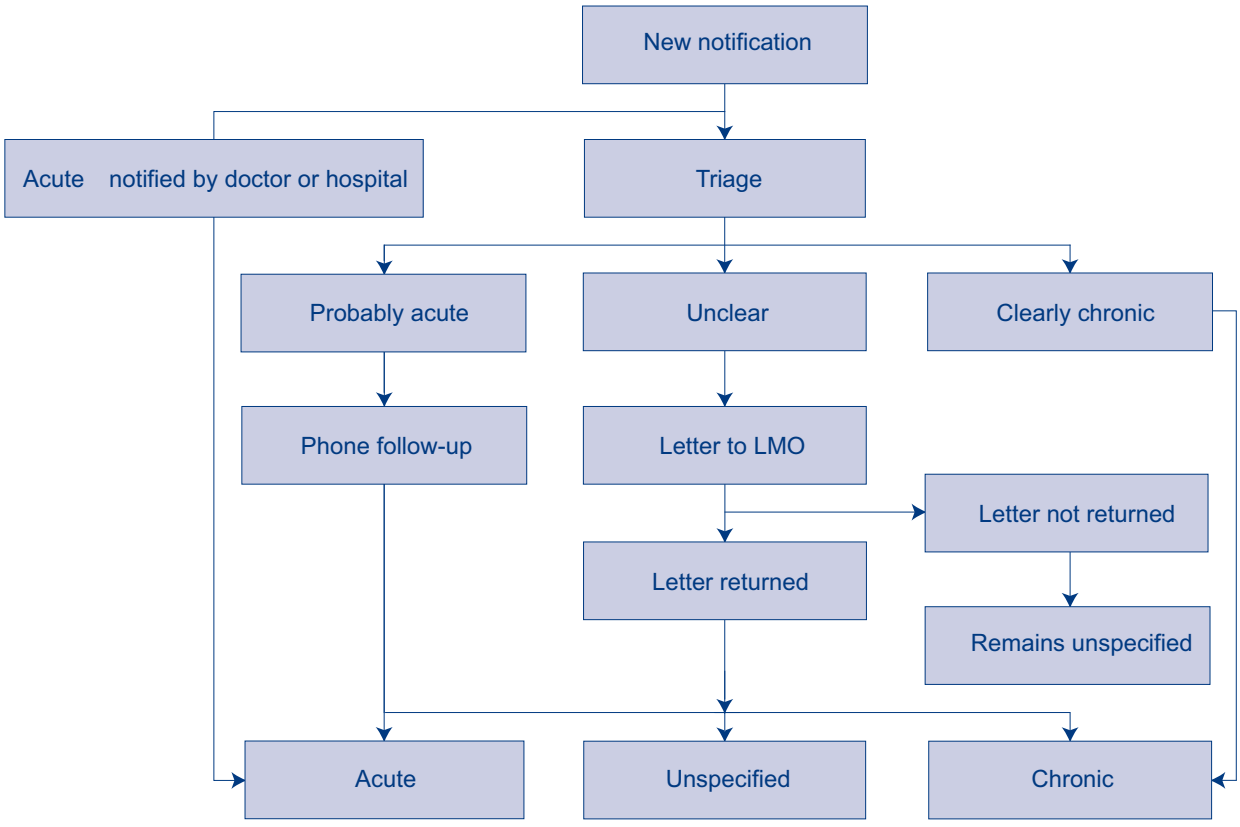
This paper presents two reviews. The first is a retrospective review of the enhanced system undertaken on data from the first six months of 2002 to assess the ability of the system to capture cases of acute hepatitis B not notified by doctors. Secondly, a review of five years of data on acute hepatitis B notifications was undertaken to explore the epidemiology of the disease in south-eastern Sydney, and to examine the method of reporting.

Results

Enhanced surveillance, January to June 2002

The South Eastern Sydney Public Health Unit received a total of 270 new notifications during the first six months of 2002. Files for five of these (all unspecified cases) could not be located, so a total of 265 notifications were reviewed in this study. Three notifications only were directly received as acute cases (one from a doctor and 2 referred from other public health units). Of the total of 262 reports, five were triaged as possibly acute cases, 115 as chronic cases and the remaining 142 cases could not be categorised. Possibly acute cases were followed up with a telephone call to the referring doctor; uncategorised cases were followed up by letter sent to the referring doctor. One hundred and five letters were returned, giving a response rate of 74 per cent. Telephone confirmation or letter contact

Figure 1. The enhanced surveillance system for acute hepatitis B



with referring doctors identified seven acute cases among triaged laboratory notifications (all reportedly had clinical symptoms or signs of acute hepatitis at the time of medical practitioner attendance), giving a total of 10 acute cases of hepatitis B in south-eastern Sydney residents for this period. The enhanced surveillance system increased the number of cases of acute disease at least threefold, from three to 10 (Figure 2).

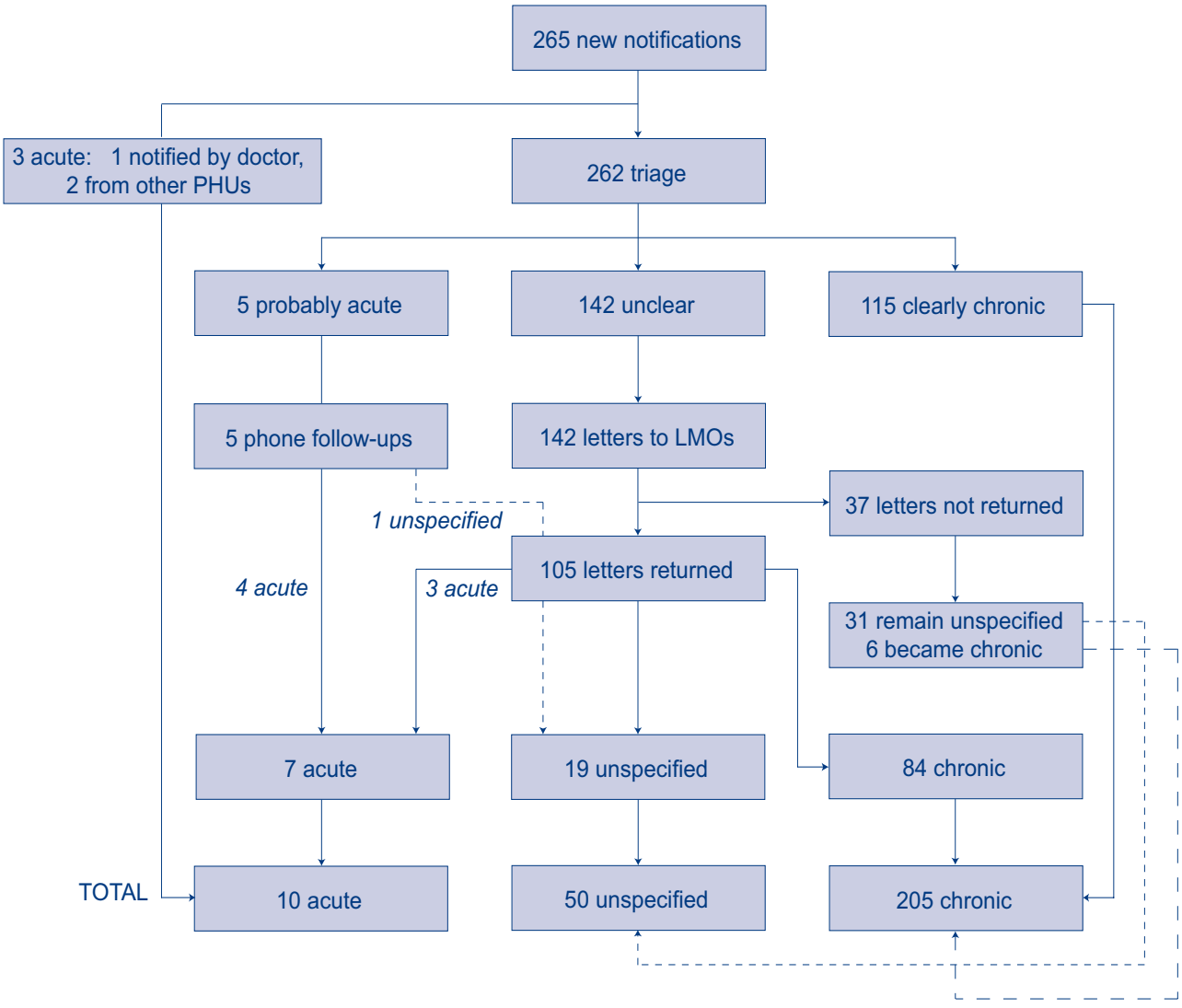
Epidemiology of acute hepatitis B, 1998 to 2002

The incidence of acute hepatitis B remained fairly steady between 1998 to 2002, ranging from 20 to 24 cases per annum, and giving a total of 108 cases over this 5-year period (2.8 cases per 100,000 per annum). The incidence peaked in the 20–24 year age group, followed by the 25–29 year age group. No cases were reported in children less than 15 years. The incidence ratio of females to males was 1:2.8. Risk factors for acute infection (n=69) based only on the report of the referring doctor included intravenous drug use (n=20), unprotected sexual activity (n=28: 13 heterosexual; 7 homosexual; 8 unspecified), unspecified high-risk behaviour (n=4), percutaneous exposure (n=6), occupational exposure (n=2), and other (n=7). More than one risk factor was identified in 10 cases. No risk factors were identified in 12 cases.

Three-quarters of the cases of acute disease were initially notified by a laboratory (n=82); the remainder by doctor (n=24) or hospital (n=2). The median number of days from disease onset to notification was 12.5 days (range 2–102 days, 2 cases had missing data) for doctor-notified cases, compared with 19 days (range 0–112 days, 9 cases had missing data) for laboratory-notified cases.

Amongst laboratory notifications, 32 contained HBsAg results only, while 39 contained additional hepatitis serology results (11 laboratory results were missing at the time of review). Acute cases were more likely to be suspected if serological results in addition to HBsAg were included with the laboratory notification, and therefore more likely to be triaged initially to telephone follow-up, rather than letter follow-up.

Figure 2. Outcome of new notifications reported to south-eastern Sydney in Epi Weeks 1 to 26, 2002



Conclusions

Hepatitis B is one of the most frequently reported notifiable conditions in New South Wales. Most reports are considered chronic infections, with only a small proportion known to be acute. In 2001, the notification rate for acute infection in New South Wales was 1.3 cases per 100,000 population.[4] However, this study indicates that this figure is likely to be a considerable underestimate, since notification by medical practitioners is not complete, and laboratory notification of positive HBsAg does not identify acute disease. In 2001, south-eastern Sydney had a rate of 2.6 cases per 100,000 population, twice that of New South Wales, and more than the surrounding health service areas of Central, Western and South Western Sydney (2/100,000, 0.4/100,000 and 1.6/100,000 respectively).[4] The ratio of notifications of acute disease to other cases (chronic and unspecified disease combined) varied considerably between south-eastern Sydney and New South Wales overall. One acute case was notified for every 29 notifications of chronic or unspecified disease in south-eastern Sydney compared with the New South Wales figure of one acute case for every 53 notifications of chronic or unspecified disease. The reasons for this discrepancy are unclear, and may be a reflection of variations in the incidence and prevalence of disease in the community, the extent of screening by medical practitioners, or, indeed, the effect of increased case identification due to enhanced versus routine surveillance.

Accurate identification of new infections is important if variations in the patterns of disease or in the prevalence of risk factors are to be detected, and for the evaluation of the effectiveness of hepatitis B vaccination programs. In the absence of complete reporting by medical practitioners, case ascertainment is problematic without some form of enhanced surveillance system. Surveillance of acute cases could be assisted if laboratories were required to report the results of all antibody and antigen tests undertaken, rather than only the HBsAg result. Recent changes to the Medicare Benefits Schedule which have simplified requests for hepatitis serology to three items (1 November 2002)[5] may still not provide public health units with the necessary information (such as IgM to core antigen) to identify acute infections in the absence of clinical data. For example, Item 69481 which provides three tests for the investigation of infectious causes of acute or chronic hepatitis will not provide IgM to core antigen if tests for antibodies to hepatitis A and C, and hepatitis B surface antigen are performed.

By comparison, the more centralised structure of the Victorian notification system means that notifications are received from a limited number of laboratories, and serological data (in particular IgM to core antigen) in addition to HBsAg are usually reported, aiding identification of acute disease (M Moloney, Department of Human Services, Victoria, personal communication, 25 November 2003). This may explain why the Victorian notification rate in 2000 was higher than the New South Wales rate (2.4 cases per 100,000 population[6] versus 1.5 cases per 100,000 population[7]).

The system of enhanced surveillance of acute hepatitis B operating in the south-eastern Sydney area improves acute case ascertainment compared with routine surveillance. It has proven to be a sustainable system, with minimal additional staff time required (estimated to be 5–10 minutes per week for triage, 5–10 minutes per week for telephone follow-up and 30 minutes per week in sending follow-up letters). Even so, such a system comes with an opportunity cost in that limited staff resources are diverted from other tasks. Sentinel enhanced surveillance systems in a sample of New South Wales public health units may be an effective and efficient method of improving the surveillance of acute hepatitis B. We would also recommend that laboratories in New South Wales (and elsewhere) be required to report IgM to core antigen, if available, when notifying a positive HBsAg result, to assist in the identification of acute disease. As liver enzyme results also assisted the triage process, we believe that inclusion of liver enzyme results with the notification would be of value.

Acknowledgement

We wish to acknowledge the key role played by the staff of the South Eastern Sydney Public Health Unit and the clinicians in this surveillance system.

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Antiviral prophylaxis in the management of an influenza outbreak in an aged care facility

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Southern New South Wales Public Health Unit

Abstract

Influenza in persons aged ≥ 65 years is associated with an increased risk of severe complications. Residents in aged care facilities have a higher proportion of chronic illnesses and within closed settings there is an increased risk of transmission. In July 2002, a 50 bed aged care facility reported an influenza-like illness (ILI) among residents and staff despite over 90 per cent influenza vaccine coverage among residents. A total of 17 of 49 residents and 9 of 43 staff met the case definition for ILI with onset on or after 26 June 2002. Seven people required hospitalisation and two died. Nasopharyngeal swabs were collected from symptomatic residents and staff, and influenza A was detected in six residents and two staff. Five unimmunised residents and 33 unimmunised staff were offered influenza vaccine and all residents and staff were offered oseltamivir prophylaxis of 75mg daily for 10 days. Subsequently, of 41 residents tested, seven demonstrated a fourfold or greater rise in antibody titres specific to H3N2 yet reported no symptoms. All seven had been immunised at least eight weeks previously, and had taken oseltamivir prophylaxis. This outbreak was characterised by a high attack rate of ILI in a well-immunised community. The ability to access rapid diagnostic testing enabled the prompt initiation of antiviral prophylaxis, which may have a role in controlling influenza in this setting. *Commun Dis Intell* 2004;28:396–400.

Keywords: influenza A, outbreak, aged care facility, oseltamivir prophylaxis

Introduction

Influenza A and B outbreaks are a major cause of serious illness, hospitalisation and death in elderly persons. Outbreaks in aged care facilities have resulted in attack rates of 10 per cent to 70 per cent, with up to 55 per cent of ill residents requiring hospitalisation or as many as 30 per cent dying from complications.[1–5]

In early July 2002 the director of a 50 bed aged care facility reported an influenza-like illness (ILI) among residents and staff. A coordinated response was undertaken by the Southern New South Wales Public Health Unit (SNSWPHU) initially to determine the cause of the illness and to design control measures.

The facility has an influenza immunisation program and 90 per cent of residents had been immunised in late March/early April 2002 with vaccine from three different manufacturers. Only 28 per cent of staff had been immunised. The composition of the influenza vaccine used in Australia in the 2002 season was A/New Caledonia/20/99 (H1N1)-like virus, A/Moscow/10/99 (H3N2)-like virus and B/Sichuan/379/99-like virus.

Aims

We conducted a study into this outbreak to determine its cause and extent, and the feasibility and effects of providing staff and residents of the institution with preventive antiviral therapy.

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Methods

ILI was defined as the onset of fever or cough or rhinitis and at least one secondary symptom such as sore throat, myalgia, headache, malaise, poor appetite and chills with onset on or after 26 June 2002. Data were collected from a review of the notes of the residents.

Combined nose and throat swabs were collected in a viral transport medium from symptomatic residents and staff, and couriered to the reference laboratory, where they were examined by direct immunofluorescence (DIF) and viral culture. Isolates were further subtyped by the WHO Collaborating Centre for Influenza Reference and Research in Melbourne.

Consent was obtained from all residents to have serum collected for acute and convalescent (4 weeks later) influenza serology in order to assess the effectiveness of the prophylaxis. Acute and convalescent sera were tested in parallel for antibodies to influenza A and B using complement fixation (Virology Department, Institute of Clinical Pathology and Medical Research) and haemagglutination inhibition against A/Brisbane/6/2002 and B/Brisbane/32/2002 (WHO Collaborating Centre for Influenza Reference and Research). Individuals with a fourfold or greater rise in influenza-specific antibody titre were considered as being recently infected.

The attack rate was calculated by dividing the number of confirmed cases by the total number of residents and staff.

A questionnaire was developed and administered to all staff members to provide information on compliance and side effects of antiviral prophylaxis. Patient notes were reviewed for reported side effects in residents.

Results

Epidemiological investigation

At the time the facility had 49 residents including 22 males and 27 females who resided in single rooms between three single storey wings (A, B and C). All wings were connected by corridors and opened onto a common dining area. Most residents requiring minimal care were located in A and B wings, whereas C wing was for residents with dementia who required closer attention to their daily living needs. There were 42 staff working at the facility during the outbreak including one volunteer and the local pathology collector.

Medical records for all 49 residents were reviewed and questionnaires were completed by 32 (76%) of the 42 staff. The first two cases in the outbreak became sick on the same day: one was a resident and the other a staff member. The average age of the residents and staff was 81 years (SD ± 10) and 48 years (SD ± 10) respectively. The most common symptoms are outlined in Table 1.

Residents

Seventeen (35%) of 49 residents met the case definition for an ILI. The male to female ratio was 1:1.1. Three hospitalised case-residents (6%) were diagnosed with pneumonia by their general practitioners, and confirmed by chest x-ray. Five case-residents were hospitalised (10%) and one of these case-residents died 14 days after the onset of illness. Onset of illness occurred between 26 June and 18 July 2002 with a peak of 13 case-residents in the week 29 June to 5 July (Figure).The attack rates in case-residents were highest in A and C wings, and lowest in B wing (45%, 38% and 25% respectively).

Forty-three (88%) residents had two or more chronic medical conditions. Forty-four residents (90%) were immunised prior to this outbreak with the trivalent vaccine between March and April 2002. One resident was immunised at the beginning of the outbreak and the remaining four residents declined immunisation. All cases of pneumonia, hospitalisation and death were among residents who were immunised. None of the unimmunised residents became sick.

Table 1. Symptoms experienced by residents and staff

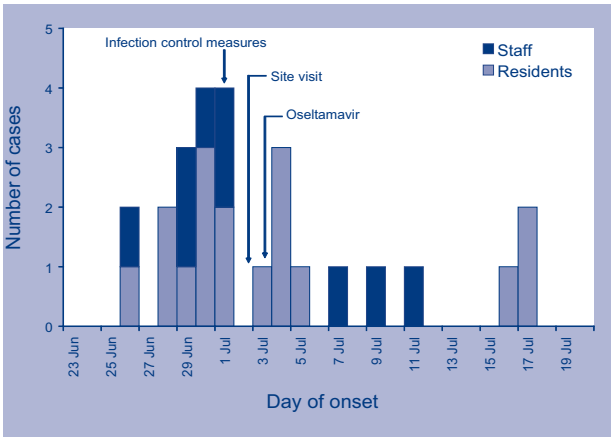
Cases	Fever (%)	Myalgia (%)	Head-ache (%)	Severe malaise (%)	Non productive cough (%)	Sore throat (%)	Rhinitis (%)	Chills (%)	Productive cough (%)	Poor appetite (%)
Residents (n=17)	100	23	18	23	29	12	29	12	53	35
Staff (n=9)	44	33	66	22	44	66	77	22	55	22

Staff

Nine (21%) of 43 staff met the case definition for an ILI. This included a staff member who died after hospitalisation on 29 June 2002 following a 3-day history of influenza-like illness, with the cause of death at autopsy given as bilateral pneumonia. One staff case was diagnosed with bronchitis. Onset of illness occurred between 26 June and 11 July 2002 with a peak of six staff cases in the week 26 June to 2 July (Figure). Six (67%) of the staff cases were in roles that required direct contact with the residents. The remaining three staff cases were not in direct contact with residents.

Prior to this outbreak, 13 of 46 (28%) staff had been immunised, with a further 11 (24%) immunised after the onset of the outbreak.

Figure. Cases of ILI by day of onset, June to July 2002



Laboratory investigations

Influenza A H3N2 was detected in eight combined nose and throat swabs taken from either case-residents or staff cases. Seven were detected by DIF and eight were culture positive. One influenza isolate was later further subtyped as A/Moscow/10/99 (H3N2)-like, and thus covered by the 2002 influenza vaccine.

Overall, 41 paired sera from residents who had oseltamivir chemoprophylaxis and seven acute sera were tested. A fourfold rise or persistently elevated antibody titres for influenza were seen in 14 of 41 paired sera including 10 influenza A and four influenza B; three of seven single sera had elevated influenza A antibody titres. There were 16 individuals who had both DIF and antibody testing—eight were negative by both assays, four had influenza A infection detected by both methods, three had serological evidence of influenza A but negative DIF, and one had influenza A on DIF and negative serology on a single acute serum sample. Laboratory test results by immunisation status are outlined in Table 2.

Public health interventions

The SNSWPHU initially ensured that infection control measures were implemented to minimise transmission and that combined nose and throat swabs were collected immediately from sick residents and staff. Information was given to all staff, residents and their consulting general practitioners detailing the illness.

Although it may have been too late to avert illness in this outbreak, those residents and staff who were not immunised were offered influenza vaccine. In addition, all residents and staff who were not confirmed cases of influenza, were offered antiviral prophylaxis with oseltamivir. A media release was also distributed locally to raise community awareness of an increase in influenza activity.

Infection control measures

Droplet precautions and environmental cleaning measures were advised.[8] As all residents had single rooms in this facility, those who became sick were asked to remain in their rooms and refrain from using the communal dining room until they had recovered or for at least five days after the onset of symptoms. This practice was challenging for staff when residents from the dementia wing became sick. Any sick staff or volunteers were excluded from the facility for five days from the onset of symptoms or until the symptoms resolved. Unimmunised staff were also advised not to work at other health care

Table 2. Immunisation status and confirmed influenza A

Influenza-like illness	With influenza-like illness and immunised		Tested		Tested with confirmed influenza A	
	n	%	n	%	n	%
Residents (n=17)	16	94	17	100	6	35
Staff (n=9)	3	33	4	44	2	50

facilities until after four days had elapsed from their last shift at this facility. The manager of the facility was advised to restrict visitors to residents and to defer new admissions or transfers of residents for the duration of the outbreak. All non-urgent medical appointments were rescheduled and if a resident required hospitalisation the health service manager was to be made aware of the illness at the facility.

Antiviral prophylaxis

Oseltamivir was offered as prophylaxis to all staff and residents at a dose of 75mg daily for 10 days from 4 July 2002 at a cost of $3.48 per dose. The total direct cost of providing oseltamivir prophylaxis was $2,679. All residents (43/49) who did not have confirmed influenza received prophylaxis, as did all staff members (34/42) who did not have either confirmed influenza or a medical contraindication.

One resident did not complete the 10-day course as the general practitioner discontinued treatment because of vomiting. No other adverse effects were reported among residents. Staff questionnaires were distributed to ascertain adverse effects in this group, with a response rate of 76 per cent. Nausea (27%) and headache (19%) were the most common adverse effects followed by individual reports of vomiting, abdominal pain, rash, indigestion and a feeling of being thirsty. Only one staff member discontinued prophylaxis owing to adverse effects.

Four residents (8%) reported ILI consistent with the case definition after the introduction of prophylaxis but did not develop secondary complications nor require hospitalisation. Nose and throat swabs were negative for influenza A and B, and no antibody response to influenza A or B was detected.

Discussion

This influenza outbreak occurred despite over 90 per cent of the residents having received vaccine in recent months. Prompt recognition of the outbreak, its notification to the SNSWPHU and the institution of infection control measures may have limited its progress. Rapid diagnosis of influenza from nose and throat swabs gave the opportunity to administer antiviral prophylaxis to prevent the secondary cases of influenza in this high-risk setting. Oseltamivir is an oral neuraminidase inhibitor whose use leads to viral aggregation at the host cell surface and a reduction in the amount of virus that is released to infect other cells. It has activity against both influenza A and B viruses, and has shown to be effective in both the treatment and prophylaxis of influenza infections.[9]

Whilst the strain identified in a patient in this outbreak was included in the Australian 2002 vaccine, there may have been some patients in this outbreak with influenza caused by different strains not covered by the vaccine. The outbreak occurred at the time that influenza activity, both locally and throughout the State, had begun to increase, and when the majority of influenza strains isolated across Australia were B/Hong Kong/330/2001-like viruses, against which the B/Sichuan-like component of the 2002 vaccine was expected to have reduced effectiveness.[6,7] In addition, it is possible that another respiratory virus was circulating through the aged care facility as four residents on oseltamivir prophylaxis developed an ILI consistent with the case definition but were negative for influenza by antigen, culture and antibody testing. There was increased respiratory syncytial virus activity at this time in 2002 reported to NSW Health Department.[11]

Oseltamivir may have prevented influenza illness in this outbreak, although a randomised control trial would be required to give definitive evidence. Oseltamivir was used as prophylaxis as it was easy to administer, it was readily accessible and has few adverse effects (especially when compared to amantadine, an antiviral sometimes used as prophylaxis or treatment in influenza A outbreaks) or contraindications. Neuraminidase inhibitors are effective against both influenza A and B, unlike amantadine. In Australia, oseltamivir is approved for use as treatment and prophylaxis of both influenza A and B, and should be considered in influenza outbreaks in aged care facilities. The value of antiviral drugs depends on rapid laboratory confirmation of influenza.

Whilst the cost of oseltamivir was not insubstantial, it may have prevented clinical illness in at least seven residents and may have prevented further transmission, hospitalisations and even deaths. Interestingly, four residents had an antibody rise to influenza A and three had antibody rise to influenza B. Sequential outbreaks of influenza A and B have been reported in nursing homes in the United States of America.[10]

Immunisation remains the single most important tool against influenza and is 50–60 per cent effective in preventing hospitalisation or pneumonia and 80 per cent effective in preventing death in aged care facilities. It is widely accepted that when influenza vaccine and epidemic strains are well-matched, achieving increased immunisation rates among staff can reduce the risk for outbreaks by inducing herd immunity.[1,12,13]

The role of health care workers in the introduction and transmission of influenza in these settings, led the National Health and Medical Research Council to recommend annual immunisation of staff employed in health care facilities. Immunisation of health-care workers has been associated with a substantial

decrease in mortality among residents of aged care facilities, however, virological surveillance show no associated decrease in non-fatal influenza infection in residents.[14]

The Canadian National Advisory Committee on Immunization recommends policies to exclude health care workers from direct patient care who develop confirmed or presumed influenza and unimmunised health care workers who are not on antiviral prophylaxis in order to protect vulnerable patients in outbreak situations.[15]

Following the introduction of oseltamivir prophylaxis on 4 July 2002, there were no further laboratory-confirmed cases of influenza A or B, hospitalisations or deaths among residents in this facility despite exposure to influenza A and B viruses. The total cost of providing oseltamivir prophylaxis in comparison to the cost of treating one person in hospital for moderate to severe respiratory infection ($4,040) suggested that prophylaxis may have been cost effective.[16, 17]

Whilst the SNSWPHU has made staff influenza immunisation recommendations to all health care facilities in Southern New South Wales, clinics are commonly poorly attended by staff in direct patient care roles.[18] It is pleasing to note that following this outbreak 40 of 42 (95%) staff at this facility accepted the offer of influenza immunisation in 2003 (C Puckett, personal communication, 18 June 2003).

Acknowledgements

We gratefully acknowledge the general practitioners; staff and residents of the aged care facility. Glenis Catlin, Gabrielle Couch, Jenny Heuston and Peter and Geraldine Doyle for their assistance with specimen collection and antiviral medication distribution. Pamela Edwards and Penny Symons for their assistance with the manuscript. Sau-wan Chan at the Institute of Clinical Pathology and Medical Research, Westmead Hospital for serological testing, and Alan Hampson and staff at the WHO Collaborating Centre for Influenza Reference and Research, Melbourne, for influenza virus subtyping and haemagglutination inhibition serology.

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OzFoodNet: enhancing foodborne disease surveillance across Australia: quarterly report, April to June 2004

The OzFoodNet Working Group

Introduction

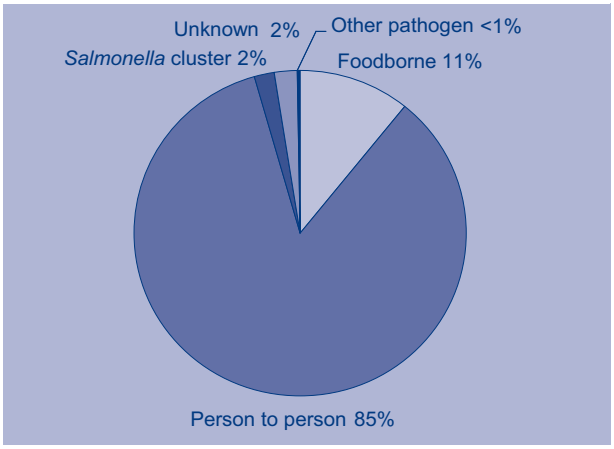
The Australian Government Department of Health and Ageing established the OzFoodNet network in 2000 to collaborate nationally to investigate foodborne disease. OzFoodNet conducts studies on the burden of illness and coordinates national investigations into outbreaks of foodborne disease. This quarterly report documents investigations of gastroenteritis outbreaks and clusters of disease potentially related to food occurring around Australia. For information on sporadic cases of foodborne illness, see Communicable Disease Surveillance, Highlights for 2nd quarter 2004 in this issue.

This report summarises the occurrence of foodborne disease outbreaks and cluster investigations between April and June 2004. Data were reported from all Australian state and territory jurisdictions and a sentinel site in the Hunter region of New South Wales. The data in this report are provisional and subject to change, as results of outbreak investigations can take months to finalise. We would like to thank State, Territory and public health unit investigators, public health laboratories, and local government environmental health officers who contributed data to this report.

Foodborne disease outbreaks

During the second quarter of 2004, OzFoodNet sites reported 342 outbreaks of gastrointestinal infections (Figure). Eighty-seven per cent (298) of these outbreaks were spread from person-to-person or were of unknown transmission affecting 8,668 people, hospitalising 199 and causing 14 fatalities. The majority of these outbreaks occurred in aged care facilities (71%), hospitals (14%) and childcare centres (7%). Outbreaks of gastroenteritis not transmitted by food are often not reported to health agencies or the reports are delayed, meaning that these figures significantly under-represent the true burden of these infections.

Figure. Mode of transmission for gastrointestinal outbreaks reported by OzFoodNet sites, April to June 2004



Thirty-seven outbreaks were due to foodborne transmission compared to 24 in the first quarter of 2004 (Table). The outbreaks affected 839 people and 43 people were hospitalised. There were no fatalities relating to these outbreaks. Thirteen outbreaks were due to *Salmonella* infection, four outbreaks of *Clostridium perfringens* (2 confirmed, 2 suspected), three outbreaks of ciguatera poisoning, two outbreaks of norovirus infection, one outbreak of rotavirus, one outbreak of a suspected toxin and one outbreak of *Bacillus cereus* poisoning. The remaining 12 outbreaks

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

Table 1. Outbreaks of foodborne disease reported by OzFoodNet sites, April to June 2004

State	Month	Setting	Agent responsible	Number exposed	Number affected	Evidence*	Responsible vehicles
ACT	April	Restaurant	Unknown	40	15	A	Calamari
	April	Restaurant	*S.* Typhimurium 197	Unknown	12	AM	Ling fish
	May	Bakery	Unknown	7	10	D	Chocolate cake
	May	Caterer	Norovirus	1140	247	A	Salmon and egg sandwiches
NSW	April	Restaurant	*S.* Typhimurium 170	13	13	D	Chicken
	April	Grocery store	*S.* Typhimurium U290	Unknown	5	D	Suspected fish cakes
	May	Fast food	Unknown	5	5	D	Takeaway chicken
	May	Fast food	Unknown	8	5	D	BBQ meat pizza
	May	Community	*S.* Typhimurium RDNC and 170	60	27	AM	Roast pork
	May	Grocery store	Unknown	27	18	D	Sandwiches mixed
	June	Restaurant	Unknown	20	6	D	Unknown
	June	Restaurant	Rotavirus	52	14	D	Dips (salsa, bean and guacamole)
	June	Restaurant	Suspected toxin	15	6	D	Unknown
	June	Restaurant	Unknown	Unknown	3	D	Mixed Asian foods
	June	Hostel	*S.* Typhimurium 135	57	43	M	Custard
	May	Restaurant	Unknown	10	3	D	Unknown
NT	May	Home	Unknown	5	5	D	Japanese imported oyster meat
Qld	April	Community	*S.* Singapore	Unknown	13	A	Sushi Rolls
	April	Restaurant	*Bacillus cereus*	190	16	M	Japanese lunch box
	April	Fast food	*S.* Typhimurium 12a	Unknown	41	D	Unknown
	April	Home	Ciguatera	Unknown	5	D	Spanish mackerel / golden trevally
	June	Restaurant	Unknown	35	25	D	Buffet meal
	June	Home	Ciguatera	5	3	D	Trevally
	June	Home	Ciguatera	4	4	D	Grey mackerel
SA	April	Home	*S.* Typhimurium 108	24	8	A	unknown
	April	Restaurant	*S.* Typhimurium 108	Unknown	9	D	unknown
	June	Restaurant	Unknown	12	9	D	unknown
	June	Fast food	*S.* Typhimurium 35	Unknown	3	D	unknown

State	Month	Setting	Agent Responsible	Number exposed	Number affected	Evidence*	Responsible vehicles
Tas	May	Restaurant	Norovirus	Unknown	57	D	Bakery products
	June	Caterer	Unknown	Unknown	13	D	Unknown
Vic	April	Community	*S.* Typhimurium 9	Unknown	9	D	Unknown
	May	Caterer	*S.* Typhimurium 12a	61	28	AM	Gourmet rolls/Red onion
	June	Hostel	Suspected toxin	30	8	D	Unknown
	May	Restaurant	*S.* Typhimurium 9	Unknown	8	D	Suspect Hollandaise sauce
	May	Hospital	Suspected toxin	Unknown	21	D	Unknown
	June	Hostel	*Clostridium perfringens*	47	22	D	Unknown
WA	April	Caterer	*Clostridium perfringens*	700	100	M	Pasta meat sauce

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

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

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

were of unknown aetiology, affecting a total of 117 people. Fourteen of the outbreaks occurred in association with meals at restaurants or cafes, five with private residences, four in association with meals prepared by commercial caterers and four with fast food outlets. Twelve outbreaks occurred in April, 12 in May and 13 in June 2004.

Sites conducted 10 retrospective cohort studies and three case control studies to investigate these foodborne outbreaks. Forty-nine per cent of outbreak investigations relied on descriptive epidemiology alone. Three outbreak investigations obtained both epidemiological evidence of an association with a food vehicle and microbiological evidence of the agent in the food. In four outbreaks investigators obtained analytical epidemiological evidence only, and a further three found evidence of a microbiological agent in the food.

During the quarter there were seven outbreaks of foodborne illness in Queensland. Queensland reported an outbreak of *Salmonella* Singapore, in association with the consumption of sushi rolls purchased from at least one sushi outlet. Thirteen people were affected. It is likely that this pathogen was introduced from a contaminated raw product, used directly as an ingredient. An investigation of an outbreak of *S.* Typhimurium 12a showed a significant association with the consumption of foods from a nationally franchised fast food chain. Cases were associated with at least three stores. No specific food vehicle or source of infection was identified and extensive traceback investigations of various produce did not identify a potential common source. Three outbreaks were due to ciguatera following consumption of Spanish mackerel, trevally and grey mackerel. All three outbreaks occurred at home. In an outbreak involving a Japanese restaurant, *Bacillus cereus* was detected in a composite food sample of rice, chicken and egg. *Salmonella aureus* enterotoxin was also detected. Lunch boxes had been prepared on the previous evening and left at room temp overnight until lunch the next day. There was also an outbreak at a restaurant where the food vehicle was not identified.

The Victorian Department of Human Services reported three outbreaks of *Salmonella* infection, and three of *Clostridium perfringens* (1 confirmed, 2 suspected). An outbreak of *Salmonella* Typhimurium 12a occurred at a conference facility and affected 28 people. Gourmet filled rolls served at the conference were suspected. Sliced red onions, which were a component of the gourmet rolls, sandwiches and salads served at one of the lunches, tested positive for *S.* Typhimurium 12a. However, it could not be determined if these were contaminated from another source, since a whole red onion tested negative for *Salmonella.* All other food samples were negative for bacterial pathogens. In one outbreak of *S.* Typhimurium 9 all eight cases ate a breakfast meal of poached eggs with hollandaise sauce from the same cafe in a rural Victorian town. Samples of eggs, hollandaise sauce and bacon all tested negative for bacterial pathogens and all egg and

environmental samples from the egg supplier tested negative for *Salmonella.* The second outbreak of *S.* Typhimurium 9 affected nine people of the same ethnic background, who bought goods at local ethnic shops. All cases ate Kosher foods at or around Passover and despite many foods being in common with cases and extensive sampling at manufacturing and retail level, no source for the outbreak could be determined. Of the three outbreaks involving or suspected to involve *Clostridium perfringens*, two occurred in aged care settings and one in a hospital. No food vehicles were identified.

New South Wales reported 12 outbreaks during the quarter, four of which were due to *Salmonella* Typhimurium. One outbreak of *S.* Typhimurium 170 involved a chicken wrap shop. Samples of chicken kebabs tested positive for *Salmonella.* Another outbreak of *S.* Typhimurium 170 at a community dinner dance affected 27 people and implicated roast pork as the food vehicle. Investigations into an outbreak at a drug and alcohol rehabilitation centre affecting at least 18 people, produced positive tests for *S.* Typhimurium 135 in 10 samples and *S.* Waycross in one sample. The food vehicle implicated in this outbreak was custard. The suspected food vehicles in the remaining outbreaks included fish-cakes, takeaway chicken, pizza, roast pork, dips, spring rolls, prawn chop suey, and sweet and sour pork.

The Northern Territory reported a single outbreak of gastroenteritis involving a private dinner party. Investigations implicated imported Japanese oyster meat. The product was clearly labelled 'cook before consumption'. The five guests who consumed raw oysters became ill while the three guests who ate either steamed oysters or no oysters did not. Faecal specimens were negative for pathogens as were a bag of raw, frozen oysters collected from the retailer. No pathogen was identified, although the illness was consistent with norovirus infection. The importer agreed to voluntarily withdraw the implicated product from the market place. This was the sixth outbreak implicating this product in the previous 18 months.

South Australia investigated four outbreaks of foodborne illness during the quarter. Following a party at a private residence in rural South Australia, eight of 24 people became ill after consumption of a BBQ lunch. A cohort study demonstrated a strong association between illness and consumption of a home made lemon meringue pie (RR= 14.0 CI 2.06-95.09) and potato bake (RR=undefined). There was also an outbreak of *S.* Typhimurium 108 associated with dining at a cafe in Metro Adelaide where six of eight cases reported eating the warm chicken salad. A case control study produced an elevated odds ratio of 5.5 but this was not significant at the 95 per cent confidence level. The study did not identify significant association with other food items.

An outbreak in a vegetarian restaurant involving four groups of people did not identify a confirmed pathogen. However, rice from the fridge tested positive for *Bacillus cereus.* In June, an outbreak of *Salmonella* Typhimurium 35 was investigated. All three cases had consumed 'yiros' from the same outlet. Routine microbiological testing of food samples did not identify any pathogens.

Western Australia reported an outbreak where 100 of 700 people working at a mine site became ill with *Clostridium perfringens*. Illness was associated with pasta meat sauce. The pathogen was isolated from both cases and food samples.

Tasmania reported two outbreaks for the quarter. One involved norovirus infection and affected 57 people. Bakery products were found to be the food vehicle. Investigations found that a food handler had been ill with vomiting and diarrhoea while serving at the bakery. The second outbreak involved a commercial caterer and no food vehicle or pathogen was identified.

The Australian Capital Territory reported four foodborne outbreaks. One outbreak of *S.* Typhimurium 197 involved a restaurant where samples of ling fish tested positive for *S.* Typhimurium 197 even though there was no epidemiological evidence to implicate the fish. An outbreak of norovirus affected 247 people in numerous work places and conferences throughout Canberra. All groups affected used a common caterer and there was a significant association between smoked salmon and egg sandwiches, which may have related to staff illness at the catering company. Two other outbreaks of unknown aetiology occurred, involving the consumption of calamari at a restaurant and chocolate cake purchased from a cake shop.

Cluster investigations

During the second quarter of 2004, Australian states and territories conducted 12 investigations into clusters of various *Salmonella* serovar infections, including: *S.* Typhimurium 135a, and *S.* Subs 3b in Queensland; *S.* Typhimurium 197, *S.* Typhimurium 170, *S.* Typhimurium 135, and *S.* Infantis in Victoria; *S.* Typhimurium 108 (2) in South Australia; *S.* Saintpaul in the Northern Territory; and *S.* Typhimurium 170, *S.* Typhimurium U290 and *S.* Typhimurium 12 in New South Wales.

The Hunter OzFoodNet site continued to coordinate an investigation into the large increase of *Salmonella* Typhimurium 12 across New South Wales. There were in excess of 130 cases of this infection for the first six months of 2003. A case control study of *S.*

Typhimurium 12 was conducted to explore hypotheses for the increase, which showed that cases were more likely than controls to have consumed chicken.

Victoria investigated a cluster of 20 cases of *S.* Typhimurium 197, 16 of which were geographically clustered. Seventeen of 18 cases with onset of illness in May were infected with organisms that were resistant to ampicillin. This cluster occurred at the same time as the outbreak of *S.* Typhimurium 197 in a restaurant in the Australian Capital Territory that had a positive isolation from ling fish. However, no connection between this cluster and the outbreak was identified.

The Australian Capital Territory reported a case of locally acquired *S.* Typhimurium 104L, which was the third case reported in the previous six months. All three cases lived in the same locality and had no history of travel. No source was identified for this cluster of infections due to this antibiotic resistant-ant organism, despite intensive efforts to generate hypotheses.

There were also several cluster investigations into pathogens other than *Salmonella,* including *Campylobacter* infections in a military camp in Victoria, and a community-wide increase in *Shigella flexneri* 2a in the Northern Territory.

Summary

Salmonella incidence was increased during the quarter, similar to the first quarter of 2004. There were several outbreaks of different phage types of *S.* Typhimurium occurring in multiple Australian states. OzFoodNet held several discussions during the quarter to try to identify links between these increases. In total, *Salmonella* infections were responsible for 32 per cent of foodborne outbreaks. Large norovirus outbreaks were reported in association with food service industries where people had worked while ill. It is vital that people responsible for preparing and handling food do not work while they have symptoms of gastroenteritis, as the results can be devastating for food businesses. Imported Japanese oysters were again implicated in an outbreak of suspected viral illness, highlighting the need for improved control measures for these products.

A report from the Communicable Diseases Network Australia, April to June 2004

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

Biannual meeting

CDNA convened for its biannual meeting in Melbourne on 13 and 14 April 2004. Issues discussed at this meeting included: proposed changes to the CDNA *Guidelines for the Early Clinical and Public Health Management of Meningococcal Disease in Australia*; development of national pertussis guidelines; recommendations flowing from the evaluation of the National Notifiable Diseases Surveillance System; and the national frozen imported oyster meat investigation. The meeting also provided an opportunity for CDNA to participate in a consultation regarding Australia's national capacity to investigate foodborne disease outbreaks, and to establish a relationship with the Health Care Associated Infections Advisory Committee of the Australian Council for Safety and Quality in Health Care.

Gastroenteritis associated with imported quick frozen oyster meat imported from Japan

During this quarter, the sixth outbreak of norovirus illness associated with consumption of contaminated imported frozen oysters in the last 18 months occurred. The six outbreaks have occurred in three different Australian states and territories and were traced back to a single growing region in Japan. The oysters in these outbreaks were consumed both raw and cooked and over 70 people reported illness. OzFoodNet and Food Standards Australia New Zealand, both members of CDNA undertook or were involved in the outbreak investigations and related food recalls and are now working with the Australian Quarantine and Inspection Service towards measures to prevent outbreaks relating to these products. To improve Australia's response to outbreaks of national significance, CDNA plans to meet with the Implementation Sub-Committee of the Food Regulation Standing Committee later this year to agree on a proposed approach and revisions to food recall protocols for foodborne diseases.

Severe acute respiratory syndrome and highly pathogenic avian influenza

To assist hospital staff during epidemics of severe acute respiratory syndrome (SARS) and highly pathogenic avian influenza, CDNA developed *SARS and influenza A (H5N1) – Interim Guidance for Recognition, Investigation and Infection Control, May 2004*. The algorithm briefly explains the steps hospital staff would follow in screening, assessing and reassessing patients. It also recommends appropriate infection control and reporting measures. The algorithm is available from http://www.health.gov.au/avian_influenza/

Improving reporting and control of trachoma

In recognition of the ongoing high prevalence of trachoma in some regions of Australia, and the lack of consistent surveillance data and control activities, CDNA recently established the Trachoma Steering Group. The Group will provide recommendations for surveillance and reporting of trachoma and a mechanism to develop a nationally consistent approach to the public health management of this condition in Australia. It is anticipated that consultation with draft national trachoma guidelines will occur over the next few months, with endorsed guidelines published by the end of 2004. Should you wish to provide input to this project, please contact the CDNA Secretariat. (Contact details see following page).

Australian bat lyssavirus and companion animals

During this reporting period, two incidents were brought to the attention of CDNA in which humans were potentially at risk of infection with Australian bat lyssavirus (ABL). Both involved dogs who had become aggressive following exposure to bats. In the first incident, the bat tested positive for ABL,

and two people bitten by the dog were offered post-exposure prophylaxis. The dog was quarantined and placed under observation for 90 days. In the second incident, the dog killed the bat, the dog was euthanased, and neither animal was tested for ABL. In light of these incidences, CDNA wishes to highlight the precautions necessary to prevent ABL transmission to people. In 2001, CDNA in consultation with the Australian Veterinary Association, and the Australian Government Departments of Agriculture, Fisheries and Forestry, and Health and Ageing, endorsed three documents that provide information relevant to the needs of medical practitioners, veterinarians, and the general public. These documents are all available on the internet at http://www.cda.gov.au/pubs/other/bat_lyssa.htm

Subcommittees and working groups of CDNA

At 30 June 2004, CDNA subcommittees and working groups included:

- Avian Influenza Protocols Working Group
- Intergovernmental Committee on AIDS/HIV, Hepatitis C and Related Diseases
- Invasive Pneumococcal Disease Steering Committee/Pneumococcal Working Party
- Jurisdictional Executive Group
- Meningococcal Disease Committee
- National Arbovirus and Malaria Advisory Committee
- National Enteric Pathogens Surveillance System Steering Committee
- National Immunisation Committee
- National Surveillance Committee
- National Tuberculosis Advisory Committee
- Public Health Laboratory Network
- Trachoma Steering Group

How to contact CDNA

Key activities of CDNA will be reported quarterly in *Communicable Diseases Intelligence*. For further information, please contact the CDNA Secretariat at: email: CDNA@health.gov.au, or telephone +61 2 6289 7983 or refer to the CDNA webpages at http://www.cda.gov.au/cdna/index.htm and http://www.nphp.gov.au/workprog/cdna/index.htm

Communicable diseases surveillance

Highlights for 2nd quarter, 2004

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

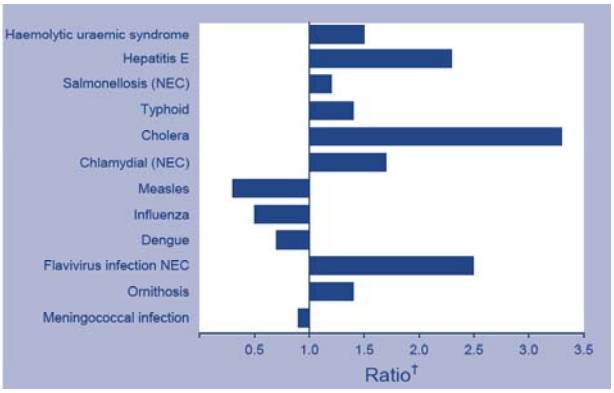
The NNDSS is conducted under the auspices of the Communicable Diseases Network Australia. NNDSS collates data on notifiable communicable diseases from State or Territory health departments. The Virology and Serology Laboratory Reporting Scheme (LabVISE) is a sentinel surveillance scheme which collates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

Please note: Figure 3 published in *Commun Dis Intell* 2004;28:409 was incorrect. The figure has been corrected in this version.

Figure 1 shows the changes in disease notifications with an onset in the second quarter 2004 compared with a 5-year mean for the same period. Diseases notifications outside the 5-year mean plus or minus two standard deviations are marked.

During the second quarter of 2004, there were increases in flavivirus NEC, cholera, chlamydial infections, haemolytic uraemic syndrome and ornithosis. There were declines in measles, dengue and influenza.

Figure 1. Selected* diseases from the National Notifiable Diseases Surveillance System, comparison of provisional totals for the period 1 April to 30 June 2004 with historical data



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

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

Gastrointestinal disease

Haemolytic uraemic syndrome

There were three cases of haemolytic uraemic syndrome (HUS) in the second quarter, two from New South Wales and one from Western Australia. The cases were unrelated and no enterohaemorrhagic *Eschericia coli* were isolated.

Hepatitis E

There were nine cases of hepatitis E notified in the second quarter, which takes the year to date to 20 cases, well above the 5-year mean. There was a history of overseas travel to endemic areas (South Asia, South America and China) in all cases.

Typhoid

There were 15 cases of typhoid notified to the National Notifiable Diseases Surveillance System (NNDSS) in the second quarter. This is also above the 5-year mean for the quarter. Eight cases were reported from New South Wales, three from Western Australia, two from Victoria and one each in Queensland and South Australia. Of the 15 cases, overseas travel was confirmed in eight cases and three cases were born overseas. Of the overseas born, one case, born in China and who had no recent overseas travel was thought to be a carrier. Of the remaining three cases, two occurred in spouses, possibly co-primary and probably locally acquired, since no travel or link to an overseas traveller could be established. There was no information on travel in the remaining two cases.

Cholera

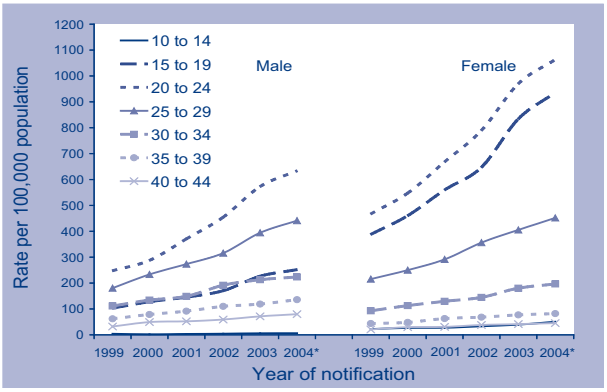
There were two cases of cholera reported in second quarter, one in Queensland and one in Victoria. The Queensland case occurred in a 50-year-old traveller returning from Thailand and was *a Vibrio cholerae* 01. The case reported in Victoria was in a 33-year-old traveller diagnosed with *V. cholerae* 01 Ogawa.

Sexually transmitted infections

Chlamydial infections

Notifications of chlamydial infections continued to increase in the second quarter. There were 8,904 notifications and a rate of 179.3 notifications per 100,000 population. Increases in *Chlamydia* notifications since 1999 have been greatest in women aged 15–24 years and men aged 20–24 years (Figure 2). The annualised rates for the first half of 2004 reached 1,063 per 100,000 in 20–24 year-old women, 935 per 100,000 in 15–19 year-old women and 633 per 100,000 in 20–24 year men.

Figure 2. Trends in notification rates of chlamydial infection aged 10 to 44 years, Australia, 1999 to 2004 (YTD), by sex



Vaccine preventable diseases

Measles

There were seven cases of measles notified in the second quarter of 2004 all in young adults aged between 18 and 31 years. Four cases were reported from Victoria and one each in New South Wales, South Australia and Western Australia. Two of the cases in Victoria were in an unvaccinated traveller returning from Asia and a contact. The cases in South Australia and Western Australia were also overseas-acquired. In the latter, there was concern that travel during the infectious period may have caused secondary cases, but despite extensive follow-up none were reported. The New South Wales case occurred in a unvaccinated 31-year-old without a history of overseas travel, but with possible workplace exposure to infected travellers.

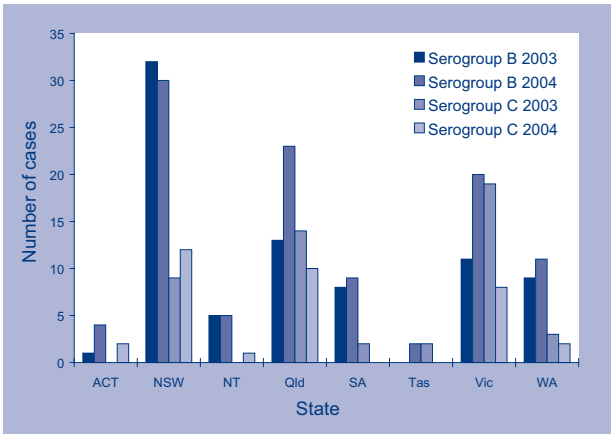
Flavivirus NEC

There were 29 cases of flavivirus NEC in second quarter. Twenty-five of these were reported from Queensland. Craig Davis from Queensland Health reported that these were a mixture of imported cases from China, the Philippines, East Timor and Papua New Guinea and serological detection as a result of extra screening as part of control efforts in local dengue outbreaks.

Meningococcal infection

There were 120 notifications of meningococcal infection in the second quarter of 2003. Provisional meningococcal serogroup data up to the end of June show that while the overall numbers had not declined there had been a significant change in the proportion of serogroup B and C. Serogroup C notifications have fallen from 26 per cent in the first half of 2003 to 17 per cent of notifications in 2004 (YTD). Conversely, serogroup B notifications increased as a proportion of all notification from 41 per cent in 2003 to 52 per cent (Figure 3). While there is a natural variation in the frequency of meningococcal serogroups, the fall in serogroup C is likely to be the result of the meningococcal serogroup C vaccination (MenCCV) program that commenced in January 2003.

Figure 3. Notifications of serogroup B and C meningococcal infection, January to June 2003 and January to June 2004, by jurisdiction

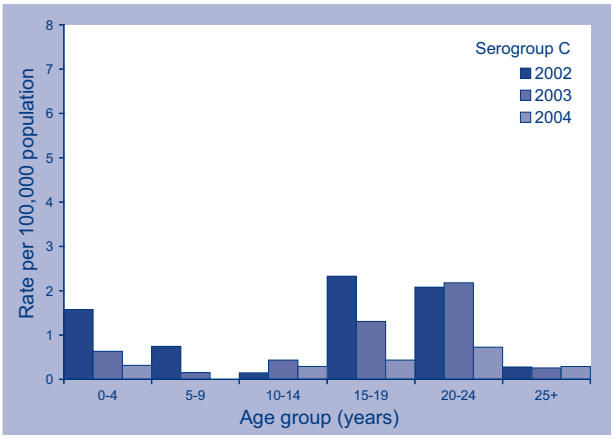


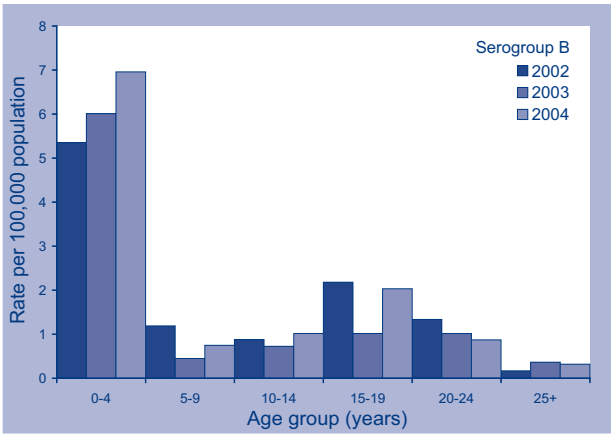
Further analysis of three years data which examined age specific rates of serogroup B and C disease was performed. Figure 4 shows rates of serogroup B and serogroup C meningococcal disease in the first six months of 2002, 2003 and 2004. While a decline in serogroup C disease was evident in all

age groups under 25 years between 2003 and 2004, there were declines in the rates of serogroup C disease between 2002 and 2003 in the under-5s, 5–9 and 15–19 year age groups. An increase in rates of serogroup B disease was seen in all age groups under 20 years in 2003 and 2004. However rates of serogroup B disease also increased in 2002 and 2003 in the under 5 years.

This analysis of rates by age group suggests that some of the change in rates of serogroup B and C disease preceded the implementation of the MenCCV program. Changes in rates since vaccination commenced in 2003 should be interpreted in the light of natural variation in different age groups and jurisdictions.

Figure 4. Notification rates of meningococcal infection, January to June 2002 to 2004, by age group: Panel A, Serogroup C, Panel B, Serogroup B





LabVISE

Laboratory reports from the Virology and Serology Laboratory Reporting Scheme (LabVISE) are shown in Table 4. In the second quarter there were fewer reports of influenza A and B viruses and an increase in reports of respiratory syncytial virus compared with the same period in 2003. The lower rate of influenza reports confirms the low rate of influenza notifications in NNDSS (Table 2) compared with the last three years average (Figure 1).

There were 81 reports of Norovirus to LabVISE in the quarter compared to 13 in the same period in 2003. This increase is likely to reflect increased Norovirus activity in Australia during the quarter. An outbreak of Norovirus affecting 140 passengers on a cruise ship was reported in May[1]. An outbreak in the Royal Hobart Hospital caused the postponement of elective surgery and isolation of gastroenteritis cases in June[2]. In New South Wales, widespread outbreaks of Norovirus were reported in the central-west of the State, that affected hospitals and aged care facilities. OzFoodNet continued investigations of food-borne outbreaks of Norovirus associated with Japanese oysters (see OzFoodNet report).

The increase in norovirus outbreaks worldwide since 2002 has been associated with the emergence of a new predominant variant of norovirus genogroup II4. This variant was detected in nine of 10 European countries[3] and in the United States of America.[4] The Public Health Laboratory Network reported that a local variant of the norovirus II4 genogroup has been identified in recent outbreaks, distinct from the European variant (Greg Smith, Queensland Health Scientific Services, personal communication).

With thanks to: Mark Bartlett (NSW Health), Craig Davis (Queensland Health) and Minda Sarna (Health Department of Western Australia).

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4. Widdowson M-A, Cramer E, Hadley L, Bresee JS, Beard S, Bulen SN et al. Outbreaks of acute gastroenteritis on cruise ships and on land: identification of a predominant circulating strain of norovirus – United States, 2002. *Infect Dis* 2004;190:27–36.

Tables

A summary of diseases currently being reported by each jurisdiction is provided in Table 1. There were 27,163 notifications to the National Notifiable Diseases Surveillance System (NNDSS) with a notification date between 1 April and 30 June 2004 (Table 2). The notification rate of diseases per 100,000 population for each State or Territory is presented in Table 3.

There were 4,737 reports received by the Virology and Serology Laboratory Reporting Scheme (LabVISE) in the reporting period, 1 April and 30 June 2004 (Tables 4 and 5).

Please note: The wrong data was published for tables 2 and 3 in *Commun Dis Intell* 2004;28:412–416. The data has been corrected in this version.

Table 1. Reporting of notifiable diseases by jurisdiction

Disease	Data received from:
Bloodborne diseases	
Hepatitis B (incident)	All jurisdictions
Hepatitis B (unspecified)	All jurisdictions except NT
Hepatitis C (incident)	All jurisdictions except Qld
Hepatitis C (unspecified)	All jurisdictions
Hepatitis D	All jurisdictions
Hepatitis (NEC)	All jurisdictions except WA
Gastrointestinal diseases	
Botulism	All jurisdictions
Campylobacteriosis	All jurisdictions except NSW
Cryptosporidiosis	All jurisdictions
Haemolytic uraemic syndrome	All jurisdictions
Hepatitis A	All jurisdictions
Hepatitis E	All jurisdictions
Listeriosis	All jurisdictions
Salmonellosis	All jurisdictions
Shigellosis	All jurisdictions
SLTEC, VTEC	All jurisdictions
Typhoid	All jurisdictions
Quarantinable diseases	
Cholera	All jurisdictions
Plague	All jurisdictions
Rabies	All jurisdictions
SARS	All jurisdictions except ACT
Smallpox	All jurisdictions except ACT, NSW, Qld, SA
Tularemia	All jurisdictions except ACT, NSW, NT, Qld, SA
Viral haemorrhagic fever (NEC)	All jurisdictions
Yellow fever	All jurisdictions
Sexually transmissible infections	
Chlamydial (NEC)	All jurisdictions
Donovanosis	All jurisdictions
Gonococcal infection	All jurisdictions
Syphilis (unspecified)*	All jurisdictions
Syphilis < 2 years duration	All jurisdictions except NSW, Qld, SA, Tas, Vic
Syphilis > 2 years duration	All jurisdictions except ACT, NT
Syphilis - congenital	All jurisdictions except ACT, NT

Disease	Data received from:
Vaccine preventable diseases	
Congenital Rubella	All jurisdictions
Diphtheria	All jurisdictions
Haemophilus influenzae type b	All jurisdictions
Influenza†	All jurisdictions
Measles	All jurisdictions
Mumps	All jurisdictions
Pertussis	All jurisdictions
Pneumococcal disease (invasive)	All jurisdictions
Poliomyelitis	All jurisdictions
Rubella	All jurisdictions
Tetanus	All jurisdictions
Vectorborne diseases	
Barmah Forest virus infection	All jurisdictions
Flavivirus infection (NEC)‡	All jurisdictions
Dengue	All jurisdictions
Japanese encephalitis	All jurisdictions
Kunjin virus§	All jurisdictions except ACT
Malaria	All jurisdictions
Murray Valley encephalitis§	All jurisdictions except ACT
Ross River virus infection	All jurisdictions
Zoonoses	
Anthrax	All jurisdictions
Australian bat lyssavirus	All jurisdictions
Brucellosis	All jurisdictions
Leptospirosis	All jurisdictions
Lyssaviruses (NEC)	All jurisdictions
Ornithosis	All jurisdictions
Q fever	All jurisdictions
Other bacterial infections	
Creutzfeldt-Jakob disease	All jurisdictions except, ACT, NSW, NT, Qld, SA
Legionellosis	All jurisdictions
Leprosy	All jurisdictions
Meningococcal infection	All jurisdictions
Tuberculosis	All jurisdictions

* Syphilis data from South Australia, Tasmania and Western Australia cannot yet be classified into less than 2 years and more than 2 years duration, NSW classifies syphilis into < 1year and > 1 years or unknown duration.

† Laboratory confirmed influenza is not notifiable in the ACT or South Australia but reports are forwarded to NNDSS.

‡ Flavivirus (NEC) replaces Arbovirus (NEC) from 1 January 2004. Western Australian data cannot yet be classified.

§ In the Australian Capital Territory, Murray Valley encephalitis virus and Kunjin are combined under Murray Valley encephalitis.

Table 2. Notifications of diseases received by State and Territory health authorities for the period 1 April to 30 June 2004, by date of onset*

Disease	State or territory								Total 2nd quarter 2004[1]	Total 1st quarter 2004	Total 2nd quarter 2003	Last 5 years mean 2nd quarter	Year to date 2004	Last 5 years YTD mean	Ratio†
	ACT	NSW	NT	Qld	SA	Tas	Vic	WA							
Bloodborne diseases															
Hepatitis B (incident)	2	17	2	13	2	10	9	5	60	70	90	99.2	130	190.6	0.6
Hepatitis B (unspecified)	12	1,324	NN	216	66	30	386	87	2,121	1,479	1,470	1,817.2	3600	3,458.2	1.2
Hepatitis C (incident)	3	4	NN	NN	8	2	13	18	48	79	110	114.0	127	247.0	0.4
Hepatitis C (unspecified)	58	1,218	72	709	182	170	714	275	3,398	3,572	3,292	4,237.4	6970	8,699.0	0.8
Hepatitis D	0	2	0	5	0	0	1	0	8	4	5	5.4	12	10.8	1.5
Gastrointestinal diseases															
Botulism	0	0	0	0	0	0	0	0	0	0	0	0.0	0	0.0	0.0
Campylobacteriosis[2]	77	NN	43	767	401	208	1,211	389	3,096	4,437	3,515	3,295.8	7533	6,870.8	0.9
Cryptosporidiosis‡	0	60	27	215	17	6	82	31	438	515	310	452.0	953	1,493.7	1.0
Haemolytic uraemic syndrome	0	2	0	0	0	0	0	1	3	3	2	2.0	6	6.6	1.5
Hepatitis A	0	32	3	7	1	0	15	16	74	106	108	174.4	180	399.4	0.4
Hepatitis E	0	1	0	1	0	2	5	0	9	11	1	4.0	20	6.2	2.3
Listeriosis	0	11	0	3	1	0	3	1	19	17	19	15.8	36	35.2	1.2
Salmonellosis (NEC)	44	524	102	666	145	48	321	137	1,987	2,772	1,602	1,676.0	4759	4,281.0	1.2
Shigellosis	1	20	36	23	15	2	15	31	143	154	103	130.6	297	285.0	1.1
SLTEC,VTEC[3]	0	0	0	2	5	0	1	0	8	12	12	9.2	20	26.6	0.9
Typhoid	0	8	0	1	1	0	2	3	15	27	6	11.0	42	35.0	1.4
Quarantinable diseases															
Cholera	0	0	0	1	0	0	1	0	2	1	1	0.6	3	1.4	3.3
Plague	0	0	0	0	0	0	0	0	0	0	0	0.0	0	0.0	0.0
Rabies	0	0	0	0	0	0	0	0	0	0	0	0.0	0	0.0	0.0
Smallpox	0	0	0	0	0	0	0	0	0	0	0	0.0	0	0.0	0.0
Tularemia	0	0	0	0	0	0	0	0	0	0	0	0.0	0	0.0	0.0
Viral haemorrhagic fever	0	0	0	0	0	0	0	0	0	0	0	0.0	0	0.0	0.0
Yellow fever	0	0	0	0	0	0	0	0	0	0	0	0.0	0	0.0	0.0

Table 2. Notifications of diseases received by State and Territory health authorities for the period 1 April to 30 June 2004, by date of onset* *continued*

Disease	State or territory								Total 2nd quarter 2004[1]	Total 1st quarter 2004	Total 2nd quarter 2003	Last 5 years mean 2nd quarter	Year to date 2004	Last 5 years YTD mean	Ratio[†]
	ACT	NSW	NT	Qld	SA	Tas	Vic	WA							
Sexually transmissible diseases															
Chlamydia	157	2,329	443	2,136	666	258	1,866	1,049	8,904	9,215	7,432	5,236.8	18119	10,387.2	1.7
Donovanosis	0	0	0	1	0	0	0	0	1	2	3	4.6	3	10.4	0.2
Gonococcal infection[4]	6	319	443	318	123	16	257	400	1,882	1,751	1,684	1,599.0	3633	3,190.0	1.2
Syphilis (unspecified)	5	0	57	0	0	0	0	0	62	82	134	213.8	144	423.2	0.3
Syphilis < two years duration	0	47	1	24	8	0	23	8	111	142	104	96.5	253	202.5	1.2
Syphilis > two years or unknown duration	0	220	17	33	1	0	87	27	385	409	249	259.5	794	514.0	1.5
Syphilis - congenital	0	0	2	0	0	0	1	0	3	2	3	2.8	5	4.4	1.1
Vaccine preventable disease															
Diphtheria	0	0	0	0	0	0	0	0	0	0	0	0.0	0	0.2	0.0
Haemophilus influenzae type b	0	1	1	1	0	0	0	0	3	7	8	10.2	10	15.8	0.3
Influenza (laboratory confirmed)[‡]	1	106	4	26	13	2	8	1	161	107	130	306.3	268	395.7	0.5
Measles	0	1	0	0	1	0	4	1	7	11	27	26.8	18	71.6	0.3
Mumps	0	12	0	5	1	0	0	0	18	24	12	39.8	42	69.0	0.5
Pertussis	13	573	1	140	59	8	112	147	1,053	1,107	832	1,100.6	2160	2,273.0	1.0
Pneumococcal disease (invasive)[‡]	17	226	18	107	51	23	106	42	590	328	544	510.7	918	768.3	1.2
Poliomyelitis	0	0	0	0	0	0	0	0	0	0	0	0.0	0	0.0	0.0
Rubella	0	6	0	4	0	0	0	0	10	6	11	51.4	16	105.8	0.2
Rubella - congenital	0	0	0	0	0	0	0	0	0	1	0	0.0	1	0.2	0.0
Tetanus	0	0	0	1	0	0	0	0	1	2	0	0.4	3	2.4	2.5
Vectorborne diseases															
Barmah Forest virus infection	0	135	8	157	3	0	3	10	316	319	760	397.8	635	662.6	0.8
Dengue	2	6	5	47	0	0	3	1	64	236	264	88.6	300	227.6	0.7
Flavivirus infection NEC	0	4	0	24	0	0	1	0	29	74	15	11.4	103	32.0	2.5
Japanese encephalitis[‡]	0	0	0	0	0	0	0	0	0	1	0	0.0	1	0.0	0.0
Kunjin virus[‡]	0	0	0	0	0	0	0	0	0	6	5	2.3	6	7.3	0.0
Malaria	4	19	8	75	8	8	19	8	149	127	155	169.4	276	380.8	0.9
Murray Valley encephalitis[‡]	0	0	1	0	0	0	0	0	1	0	0	0.0	1	1.5	0.0
Ross River virus infection	3	324	17	866	7	10	21	138	1,386	2,444	2,350	1,386.2	3830	2,812.2	1.0

Table 2. Notifications of diseases received by State and Territory health authorities for the period 1 April to 30 June 2004, by date of onset* *continued*

Disease	State or territory								Total 2nd quarter 2004[1]	Total 1st quarter 2004	Total 2nd quarter 2003	Last 5 years mean 3rd quarter	Year to date 2004	Last 5 years YTD mean	Ratio†
	ACT	NSW	NT	Qld	SA	Tas	Vic	WA							
Zoonoses															
Anthrax‡	0	0	0	0	0	0	0	0	0	0	0	0.0	0	0.0	0.0
Australian bat lyssavirus‡	0	0	0	0	0	0	0	0	0	0	0	0.0	0	0.0	0.0
Brucellosis	0	0	0	3	0	0	1	0	4	6	7	5.0	10	12.0	0.8
Leptospirosis	0	5	1	46	0	0	0	0	52	65	28	67.4	117	143.6	0.8
Lyssavirus unspecified‡	0	0	0	0	0	0	0	0	0	0	0	0.0	0	0.0	0.0
Ornithosis	0	15	0	0	1	0	37	0	53	70	44	37.0	123	62.8	1.4
Q fever	0	52	1	34	6	0	11	2	106	110	121	150.2	216	320.4	0.7
Other bacterial infections															
Creutzfeldt-Jakob disease	0	0	0	0	0	0	0	0	0	0	0	0.0	0	0.0	0.0
Legionellosis	1	26	3	3	10	0	34	11	88	86	87	113.4	174	191.4	0.8
Leprosy	0	0	0	0	0	0	0	0	0	2	2	1.2	2	2.4	0.0
Meningococcal infection	1	46	4	22	5	6	27	9	120	96	104	136.6	216	241.8	0.9
Tuberculosis	0	52	3	19	18	3	64	16	175	227	192	225.8	402	474.4	0.8
Total	407	7,747	1,323	6,721	1,825	812	5,464	2,864	27,163	30,324	25,953	24,296.5	57,487	50,053.4	1.1

1. Totals comprise data from all states and territories. Cumulative figures are subject to retrospective revision so there may be discrepancies between the number of new notifications and the increment in the cumulative figure from the previous period.
2. Not reported from New South Wales where it is only notifiable as 'foodborne disease' or 'gastroenteritis in an institution'.
3. Infections with shiga-like toxin (verotoxin) producing *Escherichia coli* (SLTEC/VTEC).

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

† Ratio = ratio of current quarter total to the mean of last 5 years for the same quarter.

‡ Notifiable from January 2001. Ratio and mean calculations are based on the last three years.

NN Not notifiable.

NEC Not elsewhere classified.

Table 3. Notification rates of diseases by state or territory, 1 April to 30 June 2004. (Rate per 100,000 population)

Disease[1]	State or territory								
	ACT	NSW	NT	Qld	SA	Tas	Vic	WA	Australia
Bloodborne diseases									
Hepatitis B (incident)	2.5	1.0	4.0	1.4	0.5	8.4	0.7	1.0	1.2
Hepatitis B (unspecified)	14.9	79.2	NN	22.8	17.3	25.2	31.5	17.8	43.1
Hepatitis C (incident)	3.7	0.2	NN	NN	2.1	1.7	1.1	3.7	1.2
Hepatitis C (unspecified)	71.9	72.9	145.2	74.7	47.7	142.5	58.2	56.3	68.4
Hepatitis D	0.0	0.1	0.0	0.5	0.0	0.0	0.1	0.0	0.2
Gastrointestinal diseases									
Botulism	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
Campylobacteriosis[2]	95.4	NN	86.7	80.8	105.0	174.4	98.7	79.7	94.0
Cryptosporidiosis	0.0	3.6	54.4	22.7	4.5	5.0	6.7	6.4	8.8
Haemolytic uraemic syndrome	0.0	0.1	0.0	0.0	0.0	0.0	0.0	0.2	0.1
Hepatitis A	0.0	1.9	6.0	0.7	0.3	0.0	1.2	3.3	1.5
Hepatitis E	0.0	0.1	0.0	0.1	0.0	1.7	0.4	0.0	0.2
Listeriosis	0.0	0.7	0.0	0.3	0.3	0.0	0.2	0.2	0.4
Salmonellosis (NEC)	54.5	31.3	205.7	70.2	38.0	40.2	26.2	28.1	40.0
Shigellosis	1.2	1.2	72.6	2.4	3.9	1.7	1.2	6.4	2.9
SLTEC,VTEC[3]	0.0	0.0	0.0	0.2	1.3	0.0	0.1	0.0	0.2
Typhoid	0.0	0.5	0.0	0.1	0.3	0.0	0.2	0.6	0.3
Quarantinable diseases									
Cholera	0.0	0.0	0.0	0.1	0.0	0.0	0.1	0.0	0.0
Plague	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
Rabies	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
Smallpox	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
Tularemia	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
Viral haemorrhagic fever	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
Yellow fever	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
Sexually transmissible diseases									
Chlamydia	194.5	139.3	893.4	225.0	174.4	216.3	152.2	214.9	179.3
Donovanosis	0.0	0.0	0.0	0.1	0.0	0.0	0.0	0.0	0.0
Gonococcal infection	7.4	19.1	893.4	33.5	32.2	13.4	21.0	82.0	37.9
Syphilis (unspecified)	6.2	0.0	114.9	0.0	0.0	0.0	0.0	0.0	1.2
Syphilis < 2 years duration	0.0	2.8	2.0	2.5	2.1	0.0	1.9	1.6	2.2
Syphilis > 2 years or unknown duration	0.0	13.2	34.3	3.5	0.3	0.0	7.1	5.5	7.8
Syphilis - congenital	0.0	0.0	4.0	0.0	0.0	0.0	0.1	0.0	0.1

Table 3. Notification rates of diseases by state or territory, 1 April to 30 June 2004. (Rate per 100,000 population) , *continued*

Disease[1]	State or territory								Australia
	ACT	NSW	NT	Qld	SA	Tas	Vic	WA	
Vaccine preventable diseases									
Diphtheria	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
Haemophilus influenzae type b	0.0	0.1	2.0	0.1	0.0	0.0	0.0	0.0	0.1
Influenza (laboratory confirmed)	1.2	6.3	8.1	2.7	3.4	1.7	0.7	0.2	3.2
Measles	0.0	0.1	0.0	0.0	0.3	0.0	0.3	0.2	0.1
Mumps	0.0	0.7	0.0	0.5	0.3	0.0	0.0	0.0	0.4
Pertussis	16.1	34.3	2.0	14.7	15.5	6.7	9.1	30.1	21.2
Pneumococcal disease (invassive)	21.1	13.5	36.3	11.3	13.4	19.3	8.6	8.6	11.9
Poliomyelitis	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
Rubella	0.0	0.4	0.0	0.4	0.0	0.0	0.0	0.0	0.2
Rubella - congenital	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
Tetanus	0.0	0.0	0.0	0.1	0.0	0.0	0.0	0.0	0.0
Vectorborne diseases									
Barmah Forest virus infection	0.0	8.1	16.1	16.5	0.8	0.0	0.2	2.0	6.4
Dengue	2.5	0.4	10.1	5.0	0.0	0.0	0.2	0.2	1.3
Flavivirus infection (NEC)	0.0	0.2	0.0	2.5	0.0	0.0	0.1	0.0	0.6
Japanese encephalitis	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
Kunjin virus	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
Malaria	5.0	1.1	16.1	7.9	2.1	6.7	1.5	1.6	3.0
Murray Valley encephalitis	0.0	0.0	2.0	0.0	0.0	0.0	0.0	0.0	0.0
Ross River virus infection	3.7	19.4	34.3	91.2	1.8	8.4	1.7	28.3	27.9
Zoonoses									
Anthrax	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
Australian bat lyssavirus	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
Brucellosis	0.0	0.0	0.0	0.3	0.0	0.0	0.1	0.0	0.1
Leptospirosis	0.0	0.3	2.0	4.8	0.0	0.0	0.0	0.0	1.0
Lyssavirus unspecified	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
Ornithosis	0.0	0.9	0.0	0.0	0.3	0.0	3.0	0.0	1.1
Q fever	0.0	3.1	2.0	3.6	1.6	0.0	0.9	0.4	2.1
Other bacterial infections									
Legionellosis	1.2	1.6	6.0	0.3	2.6	0.0	2.8	2.3	1.8
Leprosy	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
Meningococcal infection	1.2	2.8	8.1	2.3	1.3	5.0	2.2	1.8	2.4
Tuberculosis	0.0	3.1	6.0	2.0	4.7	2.5	5.2	3.3	3.5

1. Rates are subject to retrospective revision.
2. Not reported from New South Wales where it is only notifiable as ‘foodborne disease’ or ‘gastroenteritis in an institution’.
3. Infections with Shiga-like toxin (verotoxin) producing *Escherichia coli* (SLTEC/VTEC).

NN Not Notifiable.

NEC Not Elsewhere Classified.

Table 4. Virology and serology laboratory reports by state or territory[1] for the reporting period 1 April to 30 June 2004, and total reports for the year[2]

	State or territory								This period 2004	This period 2003	Year to date 2004[3]	Year to date 2003
	ACT	NSW	NT	Qld	SA	Tas	Vic	WA				
Measles, mumps, rubella												
Measles virus	0	0	0	1	1	0	3	1	6	26	13	40
Mumps virus	0	0	0	0	1	0	0	0	1	2	3	7
Rubella virus	0	1	0	1	0	0	0	0	2	4	8	12
Hepatitis virus												
Hepatitis A virus	0	0	2	1	0	0	0	6	9	19	19	36
Hepatitis E virus	0	0	0	0	0	0	2	0	2	–	7	–
Arboviruses												
Ross River virus	0	8	3	211	9	0	6	12	249	900	680	1,112
Barmah Forest virus	1	1	1	33	4	0	0	1	41	264	126	341
Dengue not typed	0	0	0	0	0	0	0	1	1	7	1	22
Flavivirus (unspecified)	0	0	2	26	0	0	1	0	29	65	78	104
Adenoviruses												
Adenovirus type 40	0	0	0	0	0	0	0	5	5	9	5	20
Adenovirus not typed/ pending	1	60	0	22	71	1	26	16	197	242	367	455
Herpesviruses												
Herpes virus type 6	0	1	0	0	0	0	1	0	2	2	2	3
Cytomegalovirus	1	96	2	18	27	3	9	0	156	240	364	511
Varicella-zoster virus	0	38	5	171	58	0	12	61	345	375	794	794
Epstein-Barr virus	0	20	19	138	137	0	8	100	422	381	1,051	839
Other DNA viruses												
Molluscum contagiosum	0	0	0	0	0	0	0	1	1	2	1	10
Poxvirus group not typed	0	0	0	0	0	0	1	0	1	–	2	1
Parvovirus	0	5	0	6	1	0	7	28	47	56	108	106
Picornavirus												
Coxsackievirus A9	0	1	0	0	0	0	0	0	1	12	1	12
Coxsackievirus A16	1	2	0	0	0	0	0	0	3	2	5	4
Echovirus type 7	0	1	0	0	0	0	0	0	1	1	1	1
Echovirus type 9	0	2	0	0	0	0	0	0	2	5	2	9
Echovirus type 11	0	4	0	0	0	0	0	0	4	1	6	2
Echovirus type 22	0	1	0	0	0	0	0	0	1	–	2	–
Echovirus type 30	0	2	0	0	0	0	0	0	2	1	4	1
Poliovirus type 1 (uncharacterised)	0	4	0	0	0	0	0	0	4	17	6	26
Poliovirus type 2 (uncharacterised)	0	6	0	0	0	0	0	0	6	3	8	4
Poliovirus type 3 (uncharacterised)	0	1	0	0	0	0	0	0	1	1	1	1
Rhinovirus (all types)	1	48	0	0	7	0	3	32	91	127	172	251
Enterovirus not typed/ pending	0	20	1	3	1	0	7	12	44	45	86	89
Picornavirus not typed	0	0	0	0	0	1	1	0	2	3	4	5

Table 4. Virology and serology laboratory reports by state or territory[1] for the reporting period 1 April to 30 June 2004, and total reports for the year,[2] *continued*

	State or territory								This period 2004	This period 2003	Year to date 2004[3]	Year to date 2003
	ACT	NSW	NT	Qld	SA	Tas	Vic	WA				
Ortho/paramyxoviruses												
Influenza A virus	0	0	0	2	15	0	0	2	19	101	58	204
Influenza B virus	0	2	1	0	13	0	0	2	18	32	30	59
Parainfluenza virus type 1	0	12	0	4	5	0	4	4	29	10	69	26
Parainfluenza virus type 2	0	0	0	2	1	0	0	0	3	30	5	49
Parainfluenza virus type 3	0	4	1	1	37	0	2	36	81	86	169	197
Respiratory syncytial virus	1	553	3	85	66	7	66	78	859	537	1,053	679
Other RNA viruses												
HTLV-1	0	0	1	0	0	0	0	0	1	4	3	8
Rotavirus	0	25	0	0	1	7	16	16	65	84	142	143
Reovirus (unspecified)	0	2	0	0	0	0	0	0	2	–	2	1
Calicivirus	0	0	0	0	0	0	0	26	26	46	26	80
Norovirus	0	0	0	0	0	0	81	0	81	13	164	41
Other												
Chlamydia trachomatis not typed	9	177	2	388	319	6	8	140	1,049	1,087	2,226	2,318
Chlamydia pneumoniae	0	0	0	0	0	0	2	0	2	6	4	10
Chlamydia psittaci	0	0	0	0	2	0	43	0	45	25	104	42
Chlamydia spp typing pending	0	2	0	0	0	0	0	0	2	–	2	–
Chlamydia species	0	1	0	0	0	0	0	1	2	–	2	–
Mycoplasma pneumoniae	0	39	9	129	33	2	47	9	268	232	623	433
Coxiella burnetii (Q fever)	1	0	0	5	16	0	5	1	28	37	76	93
Rickettsia tsutsugamushi	0	0	0	0	0	0	0	1	1	1	1	1
Streptococcus group A	0	1	0	60	0	0	35	0	96	126	221	271
Yersinia enterocolitica	0	1	0	0	0	0	0	0	1	2	2	4
Brucella abortus	0	0	0	0	1	0	0	0	1	1	4	2
Brucella species	0	3	0	0	0	0	0	0	3	–	3	2
Bordetella pertussis	2	21	0	12	17	0	14	22	88	117	244	256
Bordetella parapertussis	0	0	0	0	0	0	1	0	1	–	1	–
Legionella pneumophila	0	5	0	0	2	0	19	0	26	10	46	53
Legionella longbeachae	0	1	0	0	6	0	6	4	17	18	33	28
Legionella species	0	3	0	0	0	0	2	0	6	1	10	4
Cryptococcus species	0	0	0	2	5	0	0	0	7	9	20	12
Leptospira species	0	0	0	3	0	0	0	0	3	5	16	11
Treponema pallidum	1	33	0	109	82	0	0	0	225	302	553	674
Entamoeba histolytica	0	0	0	0	0	0	0	1	1	3	5	6
Toxoplasma gondii	0	0	0	0	0	0	1	0	1	7	15	21
Echinococcus granulosus	0	0	0	0	2	0	0	0	2	6	6	11
Total	19	1,207	52	1,434	940	27	439	619	4,737	5,750	9,865	10,557

1. State or territory of postcode, if reported, otherwise state or territory of reporting laboratory.
2. From January 2000 data presented are for reports with report dates in the current period. Previously reports included all data received in that period.

3. Totals comprise data from all laboratories. Cumulative figures are subject to retrospective revision, so there may be discrepancies between the number of new notifications and the increment in the cumulative figure from the previous period.

– No data received this period.

Table 5. Virology and serology reports by laboratories for the reporting period 1 April to 30 June 2004*

State or territory	Laboratory	April 2004	May 2004	June 2004	Total this period
Australian Capital Territory	The Canberra Hospital	–	–	–	–
New South Wales	Institute of Clinical Pathology and Medical Research, Westmead	144	134	135	413
	New Children's Hospital, Westmead	89	93	105	287
	Repatriation General Hospital, Concord	–	–	–	–
	Royal Prince Alfred Hospital, Camperdown	17	31	28	76
	South West Area Pathology Service, Liverpool	87	132	190	409
Queensland	Queensland Medical Laboratory, West End	455	567	465	1,487
	Townsville General Hospital				
South Australia	Institute of Medical and Veterinary Science, Adelaide	436	502	–	938
Tasmania	Northern Tasmanian Pathology Service, Launceston	10	6	11	27
	Royal Hobart Hospital, Hobart	–	–	–	–
Victoria	Monash Medical Centre, Melbourne	15	27	31	73
	Royal Children's Hospital, Melbourne	62	65	10	137
	Victorian Infectious Diseases Reference Laboratory, Fairfield	79	79	73	231
Western Australia	PathCentre Virology, Perth	–	21	510	531
	Princess Margaret Hospital, Perth	–	–	–	–
	Western Diagnostic Pathology	11	57	60	128
Total		1,405	1,714	1,618	4,737

* The complete list of laboratories reporting for the 12 months, January to December 2004, will appear in every report regardless of whether reports were received in this reporting period. Reports are not always received from all laboratories.

– No data received this period.

Additional reports

Australian Sentinel Practice Research Network

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

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

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

In 2004, nine conditions are being monitored, four of which are related to communicable diseases. These include influenza, gastroenteritis, varicella and shingles. There are two definitions for influenza for 2004. A patient may be coded once or twice depending on their symptoms. The definition for influenza 1 will include more individuals. Definitions of these conditions were published in Commun Dis Intell *2004;28:99–100.*

Data from 1 April to 30 June 2004 *are shown as the rate per 1,000 consultations in Figures 5, 6, 7, and 8.*

Figure 5. Consultation rates for influenza-like illness, ASPREN, 1 April to 30 June 2004, by week of report

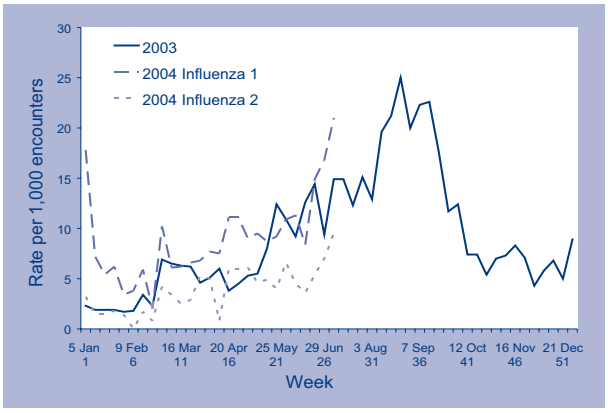


Figure 6. Consultation rates for gastroenteritis, ASPREN, 1 April to 30 June 2004, by week of report

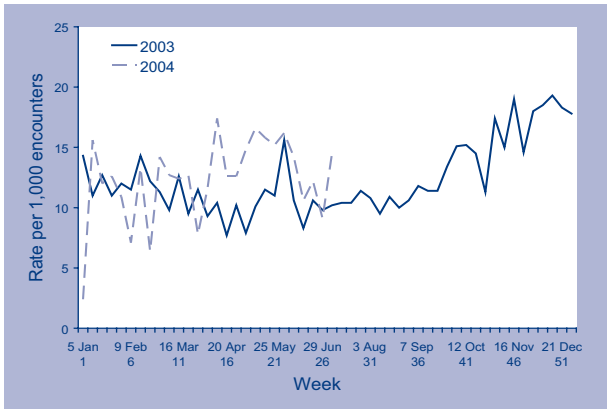


Figure 7. Consultation rates for chickenpox, ASPREN, 1 April to 30 June 2004, by week of report

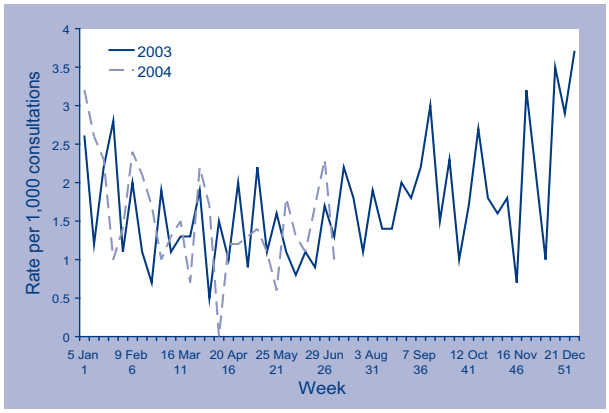
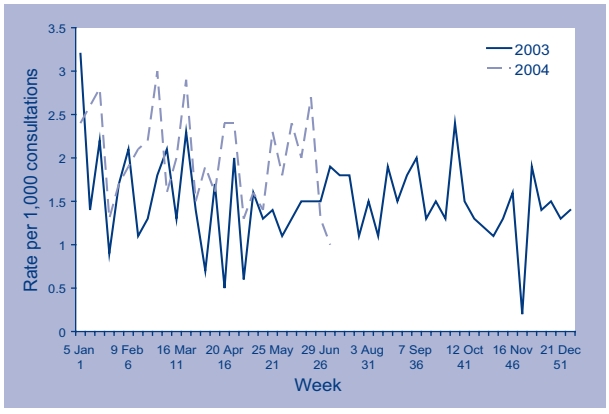


Figure 8. Consultation rates for shingles, ASPREN, 1 April to 30 June 2004, by week of report



Childhood immunisation coverage

Tables 6, 7 and 8 provide the latest quarterly report on childhood immunisation coverage from the Australian Childhood Immunisation Register (ACIR).

The data show the percentage of children fully immunised at 12 months of age for the cohort born between 1 January and 31 March 2003, at 24 months of age for the cohort born between 1 January and 31 March 2002, and at 6 years of age for the cohort born between 1 January and 31 March 1998 according to the Australian Standard Vaccination Schedule.

A full description of the methodology used can be found in Commun Dis Intell 1998;22:36-37.

Commentary on the trends in ACIR data is provided by the National Centre for Immunisation Research and Surveillance of Vaccine Preventable Diseases (NCIRS). For further information please contact the NCIRS at telephone: +61 2 9845 1256, Email: brynleyh@chw.edu.au.

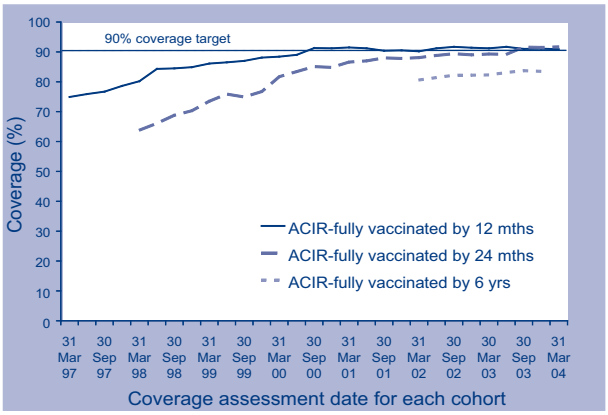
Immunisation coverage for children 'fully immunised' at 12 months of age for Australia decreased marginally from the last quarter by 0.2 percentage points to 90.9 per cent (Table 6). There were substantial decreases in 'fully immunised' coverage by State and Territory in two jurisdictions, the Northern Territory (-3.9%) and the Australian Capital Territory (-2.3%), whilst all other jurisdictions experienced very little change in coverage. Both jurisdictions also experienced decreases in coverage for diphtheria, tetanus, pertussis (DTP), poliomyelitis (OPV), *Haemophilus influenzae* type b (Hib) and hepatitis B (Hep B). Significant changes in coverage in jurisdictions like the Northern Territory and the Australian Capital Territory, who have relatively small populations, are likely to be the result of small numbers of unimmunised children having large impacts on the coverage percentages.

Coverage for children 'fully immunised' at 24 months of age for Australia increased marginally from the last quarter by 0.2 percentage points to 91.7 per cent (Table 7). Coverage for individual vaccines for Australia basically remained largely unchanged. DTP coverage remained high for this age group for all jurisdictions due to the removal of the 4th dose of DTP (due at 18 months) from the immunisation schedule from the December 2003 quarter onwards. The only other significant jurisdictional change in coverage for this age group was an increase in DTP coverage in Tasmania (+1.7%).

Table 8 shows immunisation coverage estimates for 'fully immunised' and for individual vaccines at six years of age for Australia and by state or territory. 'Fully immunised' coverage at six years of age for Australia remained the same, however there was a significant decrease in coverage in Tasmania (-3.7%) and an increase in coverage in Western Australia (+1.5%). Coverage for all individual vaccines at six years of age remained largely unchanged in most states and territories with the only significant changes occurring in Tasmania for DTP (-3.9%), OPV (-3.4%) and MMR (-3.8%). Coverage for vaccines assessed at six years is now over 85 per cent in the majority of jurisdictions, and close to 85 per cent in most jurisdictions, although coverage in Western Australia, Tasmania and the Northern Territory for this age group remains well below other jurisdictions.

Figure 9 shows the trends in vaccination coverage from the first ACIR-derived published coverage estimates in 1997 to the current estimates. There is a clear trend of increasing vaccination coverage over time for children aged 12 months, 24 months and six years, although the rate of increase has slowed over the past year for all age groups.

Figure 9. Trends in vaccination coverage, Australia, 1997 to 2004, by age cohorts



Acknowledgement: These figures were provided by the Health Insurance Commission (HIC), to specifications provided by the Australian Government Department of Health and Ageing. For further information on these figures or data on the Australian Childhood Immunisation Register please contact the Immunisation Section of the HIC: Telephone: +61 2 6124 6607.

Table 6. Percentage of children immunised at 1 year of age, preliminary results by disease and state or territory for the birth cohort 1 January to 31 March 2003; assessment date 30 June 2004

Vaccine	State or territory								
	ACT	NSW	NT	Qld	SA	Tas	Vic	WA	Australia
Number of children	974	20,842	945	12,335	4,188	1,372	14,814	5,991	61,461
Diphtheria, tetanus, pertussis (%)	92.7	92.1	87.4	92.6	92.4	94.7	93.1	90.9	92.3
Poliomyelitis (%)	92.7	92.0	86.9	92.5	92.2	94.6	93.0	90.8	92.2
Haemophilus influenzae type b (%)	93.7	94.0	92.3	95.0	94.9	96.0	95.2	93.4	94.5
Hepatitis B (%)	93.7	94.8	93.9	95.1	95.3	95.7	94.8	93.0	94.7
Fully immunised (%)	90.8	90.4	85.2	91.6	91.4	93.4	91.7	89.3	90.9
Change in fully immunised since last quarter (%)	-2.3	-0.6	-3.9	+0.1	-0.4	+1.6	+0.2	+0.3	-0.2

Table 7. Percentage of children immunised at 2 years of age, preliminary results by disease and state or territory for the birth cohort 1 January to 31 March 2002; assessment date 30 June 2004

Vaccine	State or territory								
	ACT	NSW	NT	Qld	SA	Tas	Vic	WA	Australia
Total number of children	980	21,468	915	12,595	4,566	1,466	15,295	6,402	63,687
Diphtheria, tetanus, pertussis (%)	95.0	95.5	97.3	95.2	95.7	97.0	95.9	94.6	95.5
Poliomyelitis (%)	94.6	94.7	97.2	94.6	95.3	96.8	95.3	94.0	94.9
Haemophilus influenzae type b (%)	92.0	92.8	95.4	93.6	94.1	95.2	93.9	92.4	93.4
Measles, mumps, rubella (%)	92.5	92.9	95.9	93.6	94.1	95.8	94.0	92.9	93.5
Hepatitis B(%)	95.1	95.4	98.1	95.2	95.9	97.5	96.2	95.1	95.7
Fully immunised (%)	90.0	91.0	94.5	91.9	92.7	94.9	92.3	90.6	91.7
Change in fully immunised since last quarter (%)	+1.6	+0.3	+0.8	-0.1	-0.0	+2.8	-0.1	+0.3	+0.2

Table 8. Percentage of children immunised at 6 years of age, preliminary results by disease and state or territory for the birth cohort 1 January to 31 March 1998; assessment date 30 June 2004

Vaccine	State or territory								
	ACT	NSW	NT	Qld	SA	Tas	Vic	WA	Australia
Total number of children	1,040	21,660	934	13,272	4,777	1,549	15,481	6,516	65,229
Diphtheria, tetanus, pertussis (%)	86.5	85.2	80.3	85.0	84.9	82.4	87.2	82.7	85.2
Poliomyelitis (%)	86.7	85.2	81.7	85.1	85.2	82.8	86.7	82.7	85.2
Measles, mumps, rubella (%)	85.6	84.3	82.4	84.8	84.3	81.4	87.0	82.6	84.8
Fully immunised (%)[1]	84.9	83.2	78.7	83.6	83.3	80.4	85.5	81.1	83.5
Change in fully immunised since last quarter (%)	+0.8	0.0	-1.3	+0.6	-0.0	-3.7	-0.3	+1.5	+0.1

Gonococcal surveillance

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

The Australian Gonococcal Surveillance Programme (AGSP) reference laboratories in the various States and Territories report data on sensitivity to an agreed 'core' group of antimicrobial agents quarterly. The antibiotics currently routinely surveyed are penicillin, ceftriaxone, ciprofloxacin and spectinomycin, all of which are administered as single dose regimens and currently used in Australia to treat gonorrhoea. When in vitro resistance to a recommended agent is demonstrated in 5 per cent or more of isolates from a general population, it is usual to remove that agent from the list of recommended treatment.[1] Additional data are also provided on other antibiotics from time to time. At present all laboratories also test isolates for the presence of high level (plasmid-mediated) resistance to the tetracyclines, known as TRNG. Tetracyclines are however, not a recommended therapy for gonorrhoea in Australia. Comparability of data is achieved by means of a standardised system of testing and a program-specific quality assurance process. Because of the substantial geographic differences in susceptibility patterns in Australia, regional as well as aggregated data are presented. For more information see Commun Dis Intell *2004;28:100.*

Reporting period 1 April to 30 June 2004

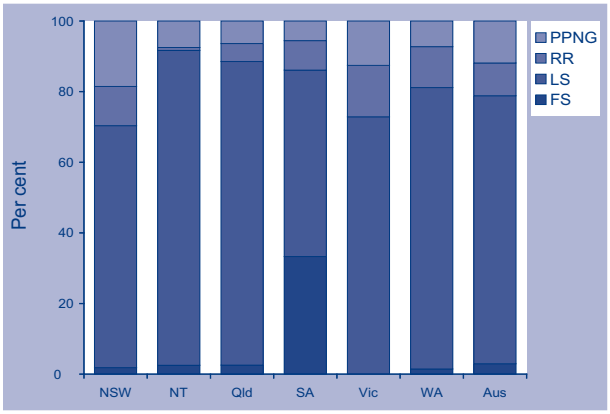
The AGSP laboratories received a total of 873 isolates in this quarter of which 851 underwent susceptibility testing. The total received was about 10 per cent less than the 980 isolated or referred in 2003. About 31 per cent of this total was from New South Wales, 23 per cent from Victoria, 18 per cent from Queensland, 14.2 per cent from the Northern Territory, 8.4 per cent from Western Australia and 4.4 per cent from South Australia. Isolates from other centres were few.

Penicillins

In this quarter 21.2 per cent of all isolates examined were penicillin resistant by one or more mechanisms—9.3 per cent penicillinase producing *neisseria gonorrhoeae* (PPNG) and 11.9 per cent by chromosomal mechanisms (CMRNG). The proportion of all penicillin resistant strains is little changed from the previous quarter, but is about five per cent more than in the corresponding period of 2003. The number of PPNG increased to 101 from the 72 seen in the same period in 2003, but the number of CMRNG decreased to 79 from 88. The proportion of all strains resistant to the penicillins by any mechanism ranged from 8.3 per cent in the Northern Territory to 30 per cent in New South Wales. In the Northern Territory, nine of 10 resistant strains were PPNG.

Figure 10 shows the proportions of gonococci fully sensitive (MIC ≤ 0.03 mg/L), less sensitive (MIC 0.06 – 0.5 mg/L), relatively resistant (MIC ≥ 1 mg/L) or else penicillinase producing (PPNG) aggregated for Australia and by State and Territory. A high proportion those strains classified as PPNG or else resistant by chromosomal mechanisms fail to respond to treatment with penicillins (penicillin, amoxycillin, ampicillin) and early generation cephalosporins.

Figure 10. Categorisation of gonococci isolated in Australia, 1 April to 30 June 2004, by penicillin susceptibility and region



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

The highest proportion of PPNG was found in New South Wales where the 50 PPNG were 18.5 per cent of all isolates. Twenty-five PPNG representing 12.5 per cent of all isolates were found in Victoria and 10 (6.4%) in Queensland. Five PPNG were found in Western Australia and two in South Australia. In addition to the increase in PPNG in the Northern Territory, PPNG numbers also rose markedly in New South Wales from the 14 detected in this period of 2003. Victoria maintained a high rate of PPNG, but numbers of PPNG were halved in other States. Numbers of isolates resistant to the penicillins by separate chromosomal mechanisms declined overall. This was mainly due to a decrease in numbers in New South Wales (from 58 to 30). In other jurisdictions, CMRNG numbers were unaltered or else increased from 2003 data.

Ceftriaxone

Three isolates with decreased susceptibility to ceftriaxone were detected in New South Wales. Small numbers of these strains have been seen for a number of years, mostly in New South Wales, but occasionally in other jurisdictions.

Spectinomycin

All isolates were susceptible to this injectable agent.

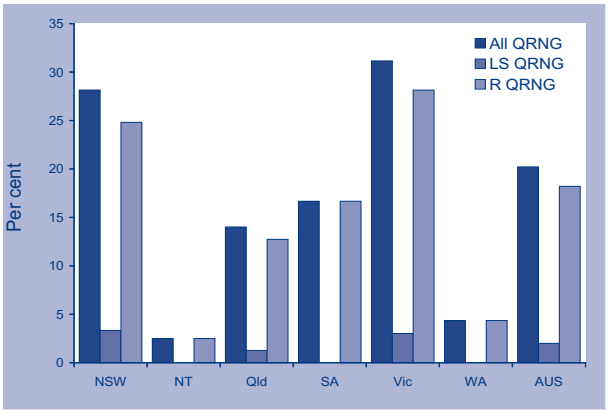
Quinolone antibiotics

The total number (172) and proportion (20.2%) of all quinolone resistant N. gonorrhoeae (QRNG) was slightly less than in the first quarter of 2004 (188, 20.5%), but substantially higher than the corresponding figures in the second quarter of 2003 (135 isolates, 14%). The majority of QRNG (155 of 172, 90%) exhibited higher-level resistance

QRNG are defined as those isolates with an MIC to ciprofloxacin equal to or greater than 0.06 mg/L. QRNG are further subdivided into less sensitive (ciprofloxacin MICs 0.06 – 0.5 mg/L) or resistant (MIC ≥ 1 mg/L) groups.

QRNG were again widely distributed. The highest number (76) was found in New South Wales (28% of isolates) while 62 QRNG were 31 per cent of gonococci in Victoria. In South Australia there were six (16%) QRNG, in Queensland 19 (14%), and three each in Western Australia (4.3%) and the Northern Territory (2.5%) (Figure 11).

Figure 11. The distribution of quinolone resistant isolates of *Neisseria gonorrhoeae* in Australia, 1 April to 30 June 2004, by jurisdiction



LS QRNG Ciprofloxacin MICs 0.06–0.5 mg/L.

R QRNG Ciprofloxacin MICs ≥1 mg/L.

High level tetracycline resistance

The number (121) and proportion (14.2%) of high level tetracycline resistance (TRNG) detected increased from the 2003 figures (92, 9.5%). TRNG are also PPNG when the different resistance determinants are both present on a single plasmid. The increase in PPNG noted above thus helps to explain the increase in TRNG. TRNG represented between 7.5 per cent (Northern Territory) and 20 per cent of all isolates (Western Australia) in the different jurisdictions.

Reference

1. Management of sexually transmitted diseases. World Health Organization 1997; Document WHO/GPA/TEM94.1 Rev.1 p 37.

Meningococcal surveillance

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

The reference laboratories of the Australian Meningococcal Surveillance Programme report data on the number of laboratory confirmed cases confirmed either by culture or by non-culture based techniques. Culture positive cases, where a Neisseria meningitidis is grown from a normally sterile site or skin, and non-culture based diagnoses, derived from results of nucleic acid amplification assays and serological techniques, are defined as invasive meningococcal disease (IMD) according to Public Health Laboratory Network definitions. Data contained in the quarterly reports are restricted to a description of the number of cases per jurisdiction, and serogroup, where known. A full analysis of laboratory confirmed cases of IMD is contained in the annual reports of the Programme, published in Communicable Diseases Intelligence.

Laboratory confirmed cases of invasive meningococcal disease for the period 1 April to 30 June 2004, are included in this issue of Communicable Diseases Intelligence *(Table 9).*

Table 9. Number of laboratory confirmed cases of invasive meningococcal disease, Australia, 1 April to 30 June 2004, by jurisdiction and serogroup

Jurisdiction	Year	Serogroup													
		A		B		C		Y		W135		ND		All	
		Q2	ytd	Q2	ytd	Q2	ytd	Q2	ytd	Q2	ytd	Q2	ytd	Q2	ytd
Australian Capital Territory	2004			1	6	1	1			1	1			3	8
	2003			(5)	(6)	(0)	(0)			(0)	(0)			(5)	(6)
New South Wales	2004	1	1	11	23	5	12	1	1	1	1	0	2	19	40
	2003	(0)	(0)	(9)	(17)	(8)	(15)	(0)	(0)	(0)	(0)	(6)	(8)	(23)	(40)
Northern Territory	2004			22	37	5	9	2	2	2	2	5	10	36	60
	2003			(26)	(37)	(6)	(13)	(1)	(3)	(1)	(1)	(8)	(12)	(42)	(66)
Queensland	2004			0	4	2	4							2	8
	2003			(0)	(1)	(0)	(0)							(0)	(1)
South Australia	2004			18	28	5	9	1	3	0	0	1	2	25	42
	2003			(8)	(13)	(9)	(22)	(0)	(0)	(1)	(1)	(2)	(5)	(20)	(41)
Tasmania	2004			5	9	0	0							5	9
	2003			(4)	(8)	(0)	(1)							(4)	(9)
Victoria	2004			6	10	1	2							7	12
	2003			(5)	(11)	(1)	(3)	(1)	(1)					(7)	(15)
Western Australia	2004			0	2	0	0			1	1	1	3	2	6
	2003						(1)								1
Australia	2004	1	1	63	119	19	37	4	6	5	5	7	17	99	185
	2003	(0)	(0)	(57)	(93)	(24)	(55)	(2)	(4)	(2)	(2)	(16)	(25)	(101)	(179)

Q2 = second quarter; *ytd* = year to 30/06/04; ND = not determined.

Numbers of laboratory confirmed diagnoses of IMD made in the same periods in 2003 are also shown in parenthesis.

Australian Paediatric Surveillance Unit

The Australian Paediatric Surveillance Unit (APSU) conducts nationally based active surveillance of rare diseases of childhood, including specified communicable diseases and complications of rare communicable diseases in children. The primary objectives of the APSU are to document the number of Australian children under 15 years newly diagnosed with specified conditions, their geographic distribution, clinical features, current management and outcome. Contributors to the APSU are clinicians known to be working in paediatrics and child health in Australia. In 2003, over 1,000 clinicians participated in the surveillance of 14 conditions through the APSU, with an overall response rate of 96 per cent. The APSU can be contacted by telephone: +61 2 9845 2200, email: apsu@chw.edu.au. For more information see Commun Dis Intell *2004;28:101.*

The results for 1 January and 31 March 2004 are shown in Table 10.

HIV and AIDS surveillance

National surveillance for HIV disease is coordinated by the National Centre in HIV Epidemiology and Clinical Research (NCHECR), in collaboration with State and Territory health authorities and the Commonwealth of Australia. Cases of HIV infection are notified to the National HIV Database on the first occasion of diagnosis in Australia, by either the diagnosing laboratory (Australian Capital Territory, New South Wales, Tasmania, Victoria) or by a combination of laboratory and doctor sources (Northern Territory, Queensland, South Australia, Western Australia). Cases of AIDS are notified through the State and Territory health authorities to the National AIDS Registry. Diagnoses of both HIV infection and AIDS are notified with the person's date of birth and name code, to minimise duplicate notifications while maintaining confidentiality.

Tabulations of diagnoses of HIV infection and AIDS are based on data available three months after the end of the reporting interval indicated, to allow for reporting delay and to incorporate newly available

Table 10. Confirmed cases of communicable diseases reported to the Australian Paediatric Surveillance Unit between 1 January to 31 March 2004*

Condition	Previous reporting period January–December 2003	Current reporting period January–March 2004*
Acute flaccid paralysis	30	5
Congenital cytomegalovirus confirmed (< 3 weeks of age) suspected (3–52 weeks of age)	10	3
Congenital rubella	3	1
Perinatal exposure to HIV HIV infection	14 1†	6
Neonatal herpes simplex virus infection	6	2
Hepatitis C virus infection	11	2

* Surveillance data are provisional and subject to revision

† HIV infection through heterosexual contact

information. More detailed information on diagnoses of HIV infection and AIDS is published in the quarterly Australian HIV Surveillance Report, and annually in 'HIV/AIDS, viral hepatitis and sexually transmissible infections in Australia, annual surveillance report'. The reports are available from the National Centre in HIV Epidemiology and Clinical Research, 376 Victoria Street, Darlinghurst NSW 2010. Internet: http://www.med.unsw.edu.au/nchecr. Telephone: +61 2 9332 4648. Facsimile: +61 2 9332 1837. For more information see Commun Dis Intell *2004;28:99.*

HIV and AIDS diagnoses and deaths following AIDS reported for January to March 2004, as reported to 30 June 2004, are included in this issue of Communicable Diseases Intelligence *(Tables 11 and 12).*

Table 11. New diagnoses of HIV infection, new diagnoses of AIDS, and deaths following AIDS occurring in the period 1 January to 31 March 2004, and reported by 30 June 2004, by sex and state or territory of diagnoses

	Sex	State or territory								Totals for Australia			
		ACT	NSW	NT	Qld	SA	Tas	Vic	WA	This period 2004	This period 2003	Year to date 2004	Year to date 2003
HIV diagnoses	Female	1	24	2	6	1	0	3	0	37	20	37	20
	Male	5	92	3	38	14	0	49	1	202	201	202	201
	Sex not reported	0	1	0	0	0	0	0	0	1	1	1	1
	Total[1]	6	117	5	44	15	0	53	1	241	221	241	221
AIDS diagnoses	Female	0	1	0	2	0	0	0	0	3	3	3	3
	Male	0	21	0	4	3	0	5	3	36	34	36	34
	Total[1]	0	22	0	6	4	0	5	3	40	38	40	38
AIDS deaths	Female	0	0	0	1	0	0	0	0	1	4	1	4
	Male	0	5	0	2	1	0	3	1	12	17	12	17
	Total	0	5	0	3	1	0	3	1	13	21	13	21

1. Totals include people whose sex was reported as transgender.

Table 12. Cumulative diagnoses of HIV infection, AIDS, and deaths following AIDS since the introduction of HIV antibody testing to 31 March 2004 and reported by 30 June 2004, by sex and state or territory

	Sex	State or territory								
		ACT	NSW	NT	Qld	SA	Tas	Vic	WA	Australia
HIV diagnoses	Female	30	747	17	215	81	7	292	159	1,548
	Male	247	12,413	121	2,347	800	85	4,624	1,049	21,686
	Not reported	0	237	0	0	0	0	22	0	259
	Total[1]	277	13,424	138	2,571	882	92	4,957	1,215	23,556
AIDS diagnoses	Female	9	217	1	58	30	4	89	34	442
	Male	93	5,077	41	952	383	47	1,828	407	8,828
	Total[1]	102	5,309	42	1,012	414	51	1,927	443	9,300
AIDS deaths	Female	6	126	0	40	20	2	58	22	274
	Male	72	3,433	26	622	257	32	1,350	277	6,069
	Total[1]	78	3,568	26	664	277	34	1,416	300	6,363

1. Totals include people whose sex was reported as transgender.

National Enteric Pathogens Surveillance System

The National Enteric Pathogens Surveillance System (NEPSS) collects, analyses and disseminates data on human enteric bacterial infections diagnosed in Australia. These pathogens include Salmonella, E. coli, Vibrio, Yersinia, Plesiomonas, Aeromonas *and* Campylobacter.

Communicable Diseases Intelligence *NEPSS quarterly reports include only* Salmonella. *Data are based on reports to NEPSS from Australian laboratories of laboratory-confirmed human infection with* Salmonella. Salmonella *are identified to the level of serovar and, if applicable, phage-type. Infections apparently acquired overseas are included. Multiple isolations of a single* Salmonella *serovar/phage-type from one or more body sites during the same episode of illness are counted once only. The date of the case is the date the primary diagnostic laboratory isolated a* Salmonella *from the clinical sample.*

Interpret historical quarterly mean counts cautiously – these may be affected by outbreaks and surveillance artefacts such as newly recognised and incompletely typed Salmonella.

Reported by Joan Powling (NEPSS Co-ordinator) and Mark Veitch (Public Health Physician), Microbiological Diagnostic Unit — Public Health Laboratory, Department of Microbiology and Immunology, University of Melbourne. NEPSS can be contacted at the above address or by telephone: +61 3 8344 5701, facsimile: +61 3 9625 2689.

Reports to the National Enteric Pathogens Surveillance System of Salmonella *infection for the period 1 April to 30 June 2004 are included in Tables 13 and 14. Data include cases reported and entered by 14 July 2004. Counts are preliminary, and subject to adjustment after completion of typing and reporting of further cases to NEPSS. For more information see* Commun Dis Intell *2004;28:101–102.*

1 April to 30 June 2004

The total number of reports to the National Enteric Pathogens Surveillance System (NEPPS) of human *Salmonella* infection declined to 1,980 in the second quarter of 2004, 27 per cent fewer than in first quarter of 2004, and nine per cent fewer than in the comparable first quarter of 2003. Case counts to 14 July 2004 are approximately 97 per cent of the expected final counts for the quarter.

During the second quarter of 2004, the 25 most common *Salmonella* types in Australia accounted for 1,363 cases, 69 per cent of all reported human *Salmonella* infections.

Twenty-two of the 25 most common *Salmonella* infections in the second quarter of 2004 were among the 25 most commonly reported in the previous quarter.

S. Typhimurium phage type 170 (including the similar *S.* Typhimurium phage type 108) has been one of the most common serovars since 2002. This quarter it was, for the first time, the most common *Salmonella* in Australia. It was also the most common in New South Wales, the Australian Capital Territory and South Australia, and among the most common in Victoria.

Reports of other common salmonellae with counts well above historical averages include *S.* Typhimurium phage type 12a (particularly in Queensland and Victoria), *S.* Typhimurium phage type 197 (in the eastern mainland states), *S.* Typhimurium phage type 12 (in New South Wales) and S. Virchow phage type 8, S. Hvittingfoss and S. Weltevreden (in Queensland)

Acknowledgement

We thank scientists, diagnostic and reference laboratories, State and Territory health departments, and the Australian Government Department of Health and Ageing for their contributions to NEPSS.

Table 13. Reports to the National Enteric Pathogens Surveillance System of *Salmonella* isolated from humans during the period 1 April to 30 June 2004, as reported to 14 July 2004

	State or territory								
	ACT	NSW	NT	Qld	SA	Tas	Vic	WA	Australia
Total all *Salmonella* for quarter	48	543	97	670	142	26	328	126	1980
Total contributing *Salmonella* types	19	105	43	117	45	14	78	63	220

Table 14. Top 25 *Salmonella* types identified in Australia, 1 April to 30 June 2004, by state or territory

National rank	*Salmonella* type	State or territory								Total 2nd quarter 2004	Last 10 years mean 2nd quarter	Year to date 2004	Year to date 2003
		ACT	NSW	NT	Qld	SA	Tas	Vic	WA				
1	*S* Typhimurium 170	21	105	0	14	0	0	42	2	184	32	382	298
2	*S* Typhimurium 135	0	62	1	38	4	1	43	8	157	126	360	507
3	*S* Saintpaul	1	13	13	59	3	3	5	11	108	84	241	188
4	*S* Typhimurium 9	1	35	0	4	8	0	47	5	100	115	249	294
5	*S* Virchow 8	2	13	0	70	3	0	3	0	91	41	236	110
6	*S* Typhimurium 12a	2	7	1	47	8	1	16	6	88	13	99	25
7	*S* Birkenhead	0	22	0	42	0	0	3	1	68	54	175	124
8	*S* Typhimurium 197	5	17	0	13	2	0	20	0	57	6	141	110
9	*S* Typhimurium 12	2	37	0	5	3	0	4	1	52	13	195	62
10	*S* Chester	1	9	3	24	2	0	3	8	50	39	130	155
11	*S* Hvittingfoss	0	1	3	35	0	0	3	0	42	20	108	58
12	*S* Aberdeen	0	1	2	37	0	0	0	0	40	27	76	55
13	*S* Typhimurium 108	0	4	0	0	35	0	0	0	39	4	82	27
14	*S* Infantis	2	11	1	2	8	1	11	1	37	29	90	135
15	*S* Typhimurium U290	1	14	0	4	0	0	16	0	35	7	89	77
16	*S* Waycross	0	10	0	21	0	0	0	0	31	27	92	48
17	*S* Muenchen	0	3	6	14	2	0	0	3	28	33	72	88
18	*S* Typhimurium RDNC	1	6	2	6	6	0	5	2	28	17	55	29
19	*S* Weltevreden	1	1	1	14	0	0	3	1	21	7	39	24
20	*S* Singapore	0	7	0	6	4	1	1	1	20	16	60	48
21	*S* Typhimurium 4	0	16	0	1	0	0	3	0	20	11	49	44
22	*S* Anatum	0	1	6	8	0	0	1	3	19	27	65	76
23	*S* Agona	1	7	0	1	2	0	7	1	19	16	53	42
24	*S* Potsdam	0	3	0	8	2	1	0	1	15	14	41	28
25	*S* Ball	0	0	12	0	1	0	1	0	14	9	31	29

Overseas briefs

ProMED-mail

This material has been summarised from information provided by ProMED-mail (http://www.promedmail.org). A link to this site can be found under 'Other Australian and international communicable diseases sites' on the Communicable Diseases Australia homepage.

Poliomyelitis — Nigeria

Source: Yahoo news, 29 June 2004 [edited]

Nigeria's fast-growing polio outbreak now accounts for more than three-quarters of the world's fresh cases of the crippling disease and threatens children across West Africa.

The World Health Organization (WHO) said that, by 23 June 2004, it had confirmed 257 new victims of polio in Nigeria and that represents 77 per cent of known cases worldwide. In March 2004, Nigeria saw the highest ever-recorded monthly incidence of wild poliovirus, with 85 confirmed cases across the country.

As of April 2004, only six of the 36 states in Africa's most populous country were polio-free. Intense transmission of the polio disease in Nigeria continues despite the low transmission season. High transmission season of polio occurs during the rainy season when children have contact with contaminated water and food. Nigeria's rains begin in March and build up in August and September.

In March 2004, Kano's state government refused to take part in a United Nations-led campaign to vaccinate West African children against polio, following a campaign by Islamic clerics against the vaccine in use. Some local preachers alleged that the vaccine had been laced with fertility hormones by western agents as part of a United States of America led bid to sterilise African girls. International health experts dismissed the claims. Kano suspended vaccination, saying that it preferred to import 'safe' vaccines produced in an Islamic country. On 27 May 2004, Kano said that it was testing polio vaccines made in Indonesia, and was waiting for the results.

As of week of 22 June 2004, 333 cases of polio were reported worldwide, with 257 cases in Nigeria (77.2% of the global reports), 18 cases in Niger (5.4%), 15 cases in Pakistan (4.5%), 13 cases in India (3.9%), three cases in Afghanistan (1%), and one case in Egypt (0.3%). These six countries are those countries where polio is considered to be endemic (wild poliovirus transmission has not been interrupted) and account for 307 of the 333 reported cases in 2004 (92.2% of the reported cases globally). Of the remaining 26 reported cases, all are from countries in Africa and are related to the ongoing outbreak in Nigeria (Central African Republic 1 case, Burkina Faso 4, Benin 4, Cote d'Ivoire 8, Chad 7, Botswana 1, Sudan 1). Data are available from: http://www.polioeradication.org/content/fixed/casecount.shtml

Wild poliovirus importations — West and Central Africa, January 2003 to March 2004

Source: MMWR Morb Mortal Wkly Rep *28 May 2004;53:433–435 [edited]*

Since the 1988 World Health Assembly resolution to eradicate poliomyelitis,[1] three World Health Organization (WHO) regions (Americas, European, and Western Pacific) have been certified polio-free. The number of countries with endemic polio has decreased from 125 in 1988 to six in 2003 (Afghanistan, Egypt, India, Niger, Nigeria, and Pakistan).

Between January 2003 and March 2004, importations of wild poliovirus (WPV) occurred in eight countries that were previously polio-free: five in West Africa (Benin, Burkina Faso, Cote d'Ivoire, Ghana, and Togo) and three in Central Africa (Cameroon, Central African Republic, and Chad), resulting in 63 polio cases.[2,3]

During 1999–2000, West and Central African countries began intensifying and synchronising National Immunisation Days, leading to a decrease in the number of countries with endemic WPV from 13 in 1999 to one in 2001.[4] During January 2003–March 2004, eight previously polio-free countries reported WPV importations from endemic poliovirus reservoirs shared by northern Nigeria and southern Niger, which were largely a result of suspension of immunisation campaigns in certain northern states of Nigeria in August 2003.[3] Many of these countries had continued transmission after importation because of low routine vaccination coverage, increased intervals between supplementary immunisation activities (SIAs), and possibly declining quality of SIAs. The importations and spread highlight the increased vulnerability of countries with low routine vaccination coverage that are no longer conducting SIAs.

Ongoing transmission in Nigeria and Niger has set back the goal to interrupt poliovirus transmission in Africa by the end of 2004.[3] To restore gains made in polio eradication in West and Central Africa, WPV transmission must be interrupted in Nigeria and Niger. Until that time, neighbouring countries must create a population immunity barrier by implementing high routine vaccination coverage and high-quality SIAs. In 2002, these steps proved successful in preventing importation of WPV into Bangladesh and Nepal during a resurgence of polio in India. Surveillance standards also must be maintained to ensure rapid detection of any WPV importation, allowing for timely response and containment.

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Nipah virus — Bangladesh, 2004

Source: Centers for Disease Control and Prevention, National Center for Infectious Diseases, Travelers' Health 26 May 2004 [edited]

The outbreak of Nipah virus encephalitis that began in mid-March 2004 has ended. The outbreak, which occurred in the Faridpur District of Bangladesh, was responsible for 34 cases, including 26 deaths. No new cases have been reported since mid-April 2004. The Centers for Disease Control and Prevention (CDC) in the United States of America confirmed Nipah virus infection in 16 of the cases. Health authorities in Bangladesh, the International Center for Diarrheal Disease Research in Bangladesh, CDC, and other international partners worked together to assess and control this outbreak. Outbreaks of Nipah virus encephalitis have previously occurred in Bangladesh in May 2001, January/February 2003, and January/February 2004.

Nipah virus, which was discovered in 1999, is a zoonotic virus (infectious disease that can be transmitted from certain animals to humans). Fruit bats of the genus *Pteropus* are thought to be the reservoir for this virus. The initial outbreak in Malaysia and Singapore affected humans and commercial swine-herds, although other species, including cats, dogs, and horses, were also infected. The virus mainly caused respiratory illness among the pigs and an encephalitis syndrome among humans. Nipah virus infection can cause fever, muscle pains (myalgia), drowsiness, and encephalitis characterised by serious central nervous system illness, coma, seizures, and inability to maintain breathing.

As a general precaution, CDC advises travellers to avoid contact with wild or domestic animals in the region. As with other infectious illnesses, one of the most important and appropriate preventive practices is careful and frequent hand washing, which helps remove potentially infectious materials from the skin and prevents disease transmission.

Hepatitis A — Europe

Source: Euro Surveill Wkly, 2004;8. May 2004 [edited]

Denmark

A review of notifications to the Department of Epidemiology in the Statens Serum Institut (http://www.ssi.dk/sw379.asp) has revealed a cluster of cases of hepatitis A virus infection acquired in Denmark among men aged 18 years or older.[1] Twenty-eight cases in men have been notified so far in 2004. Of the 20 patients from the greater Copenhagen area, at least 16 are men who have sex with men (MSM). At least five Swedish men have also been infected with hepatitis A virus in Copenhagen. In the past five years, the median number of notified cases of hepatitis A acquired in Denmark each year among men aged 18 years or over was eight (range 6–11). Because of missing or delayed notifications, a full overview of the current outbreak has not yet been achieved. An increased incidence of syphilis has also been observed among MSM in Copenhagen,[2] but a possible association between these two outbreaks has not yet been established.

Outbreaks of hepatitis A among MSM have previously been reported both in Copenhagen and abroad, acquired in places such as saunas.[3,4] The most recently described outbreak in Denmark was in 1991. Studies have established risk factors for infection with hepatitis A among MSM. Examples of these risk factors are recent anonymous sexual partners, oral-anal sex or digital-anal sex, as well as visiting certain bars or saunas. Social contact of a non-sexual nature and secondarily contaminated foodstuffs may also contribute to infection. In the current outbreak, no particular risk factors have so far been found. Danish HIV/AIDS organisations are currently launching a nationwide information campaign about sexually transmitted infections, which includes hepatitis A virus infection.

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The Netherlands

A recent unusual increase in the number of notifications of hepatitis A virus infection has been detected through the Dutch data collection system for notifiable diseases.

Men who have sex with men (MSM) appear to be particularly affected. In 2004, there have so far been 99 notifications of hepatitis A virus infection acquired by men aged 18 years or older, compared with 37 during the same period in 2003. Among the notifications in 2004, 31 reported homosexual sex as a risk factor for hepatitis A. Information about patients' sexual behaviour is not yet a standard requirement of notification of hepatitis A across the country, and therefore hepatitis A cases acquired by this route of infection could be underestimated at present. In 2003, there was just one notification with sex between men as a risk factor over the same period of time. However, the current outbreak is not unusual; a similar increase in hepatitis A infections in MSM was seen in 2001. The recent outbreak of lymphogranuloma venereum in MSM[1] has increased awareness of sexually transmitted infections in the MSM community.

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Food and waterborne disease — Asia, Pacific rim

Source: News Medical.net [edited] 25 May 2004

Foodborne diseases pose a serious threat to densely populated areas of Asia and the Pacific, two United Nations agencies said today. 'So far, food contamination incidents and foodborne disease outbreaks in the region have been relatively isolated, but the potential danger is just round the corner. Already an estimated one in three people worldwide suffer annually from a foodborne disease, and 1.8 million die from severe food and waterborne diarrhoea.'

'The danger of food-related outbreaks is particularly acute in Asia and the Pacific, because of the instances in which animals and people live in proximity and the way in which some food is produced and distributed,' says Dr Kerstin Leitner, World Health Organization (WHO) Assistant Director-General responsible for Food Safety. The avian influenza epidemic, as the most recent example of a disease linking food, animals and human health, has been historically unprecedented and of great concern for human health as well as for agriculture, with 23 fatal human cases and about 100 million birds died or culled. However, in the region, more than 700,000 people die and many more are debilitated every year from single cases of food and waterborne disease—single cases that most often do not hit press headlines.

On the trade side, disruptions due to shortcomings in food quality have also been on the increase. 'Since 2001, unacceptable pesticide residue levels in fruits and vegetables, chloramphenicol and other antibiotic residues in seafood and poultry, pathogens in seafood and mycotoxins in crops and peanuts have been the cause of rejection of food export from the Asian region,' according to Hartwig de Haen, Food and Agriculture Organization (FAO) Assistant Director-General, Economic and Social Department. A ban on fish imports into the European Community (EU) cost one Asian country $335 million of lost export opportunities.

The Food Safety Regional Conference is the response to the urgent need for countries in the region to work together to develop harmonised and coordinated food safety systems, resulting in uniform emergency responses to such threats, the UN agencies say.

The Conference is part of a series of regional meetings that FAO and WHO are jointly organising to meet the needs of member countries for policy guidance and capacity-building in food safety. A practical action plan is expected to emerge from this meeting to help the region overcome the difficulties

and problems it faces in improving food safety, including surveillance and response systems. Particular attention is devoted to covering the full food production chain, with a special focus on the segments that are best suited for interventions to significantly lower the foodborne disease risk.

Severe Acute Respiratory Syndrome — China, 2004

Source: World Health Organization, CSR 18 May 2004 [edited]

China's latest Severe Acute Respiratory Syndrome (SARS) outbreak has been contained, but concerns remain. It has been more than three weeks since the last case was placed in isolation in China's latest SARS outbreak, prompting the World Health Organization (WHO) to declare that the chain of human-to-human transmission appears to have been broken. However, WHO experts and the Chinese authorities are still trying to determine the exact cause of the outbreak. The investigation has centred primarily on the National Institute of Virology in Beijing, where experiments using live and inactivated SARS coronavirus have been carried out. Two researchers at the institute developed SARS in late March and mid-April 2004. The outbreak was reported on 22 April 2004 and the institute was closed a day later.

Preliminary findings in the investigation have yet to identify a single infectious source or single procedural error at the institute—and it is conceivable that an exact answer may never be determined. Neither of the researchers is known to have directly conducted experiments using live SARS coronavirus, but investigators have serious concerns about safety procedures at the institute—including how and where procedures using SARS coronavirus were carried out, and how and where SARS coronavirus samples were stored.

WHO and Chinese authorities view with concern the occurrence of laboratory-associated SARS cases. WHO urges all member states to view this latest outbreak as an opportunity to review the safety practices of institutions and laboratories working with SARS coronavirus.

During and after the SARS outbreak of 2003, a large number of specimens were collected from possible human cases, animals and the environment. These specimens, which may contain live SARS coronavirus, are still kept in various laboratories around the world. Some of them are stored in laboratories at an inappropriate containment level. SARS coronavirus has also been propagated in reference and research laboratories, and distributed to other laboratories for research purposes. Research using live and inactivated SARS coronavirus—and other pathogens capable of causing serious illness—is being conducted in many laboratories.

WHO has issued laboratory safety guidelines and recommendations.

Creutzfeldt-Jakob disease (new variant) carrier frequency study — United Kingdom

Source: BBC News online, 21 May 2004 [edited]

Researchers at Plymouth's Derriford Hospital and the Creutzfeldt-Jakob Disease Surveillance Unit have tested 12,674 appendix and tonsil samples. Three samples showed signs of variant Creutzfeldt-Jakob disease (vCJD). Extrapolating their findings to the whole population, they estimated that 3,800 Britons might harbour the disease.

A total of 141 people have died from vCJD in the United Kingdom since the disease emerged in 1995. Scientists have been suggesting that the number of deaths from the disease had peaked. A recent study by researchers at the Imperial College London predicted the disease would claim fewer than 540 lives.

The scientists who carried out this latest study said their findings need to be interpreted with caution. There is still much to learn about vCJD and presence of the protein in these tissue samples does not necessarily mean that those affected will go on to develop vCJD.

Meanwhile, the Health Protection Agency is in the process of collecting 100,000 tonsil samples which will be tested for signs of vCJD. A long-awaited trial to test potential treatments for vCJD could start within weeks. It will examine whether an antipsychotic called quinacrine or an unlicensed drug called pentosan polysulphate can help people with the disease.

World Organisation for Animal Health (OIE): New animal disease notification system

Source: OIE internet site, Editorial. Accessed 15 May 2004 [edited]

Resolutions passed by the International Committee and recommendations issued by the Regional Commissions have instructed the World Organisation for Animal Health (OIE) Central Bureau to establish a single OIE list of notifiable terrestrial animal diseases to replace the current Lists A and B. The

aim in drawing up a single list is to be in line with the terminology of the Sanitary and Phytosanitary Agreement of the World Trade Organization, by classifying diseases as specific hazards and giving all listed diseases the same degree of importance in international trade.

An Ad Hoc Group on Terrestrial Animal Disease/ Pathogenic Agent Notification, comprised of internationally renowned experts, was convened to support the OIE Animal Health Information Department in defining criteria to determine whether a given disease should be included in the OIE list.

The proposed criteria for a disease to be included in the OIE single list were kept to a minimum and consist of easily definable factors applicable worldwide. The overriding criterion for a disease to be listed is its potential for international spread. Other criteria include a capacity for significant spread within naive populations and the zoonotic potential. Each criterion is linked to measurable parameters: if a disease fulfils at least one of these parameters, then it becomes notifiable.

Under the future OIE notification system, not only the disease but other related events will require urgent notification. The events of epidemiological significance that should be notified immediately are as follows:

- the first occurrence of a listed disease or infection in a country or compartment ('Compartment': autonomous epidemiological entity defined on the basis of either geography (zone) or management (enterprise) for the purpose of international trade);
- the re-occurrence of a listed disease or infection in a country or compartment following a report by the Delegate of the Member Country declaring the outbreak closed;
- the first occurrence of a new strain of a pathogen of a listed disease in a country or compartment;
- a sudden and unexpected increase in morbidity or mortality caused by an existing listed disease;
- emerging diseases with significant morbidity/ mortality or zoonotic potential; or
- evidence of a change in the epidemiology of a listed disease (including host range, pathogenicity, strain of causative pathogen), in particular if there is a zoonotic impact.

Rabies: human organ transplantation — USA

Source: WWMR Morb Mortal Wkly Rep*, Dispatch Vol 53 9 July 2004 [edited]*

On 1 July 2004, the Centers for Disease Control and Prevention (CDC) reported laboratory confirmation of rabies as the cause of encephalitis in an organ donor and three organ recipients at Baylor University Medical Center (BUMC) in Dallas, Texas.[1] Hospital and public health officials in Alabama, Arkansas, Oklahoma, and Texas initiated public health investigations to identify donor and recipient contacts, assess exposure risks, and provide rabies post-exposure prophylaxis (PEP). As of 9 July 2004, PEP had been initiated in approximately 174 (19%) of 916 persons who had been assessed for exposures to the organ recipients or the donor.

As a result of its public health investigation, the Arkansas Department of Health determined that the donor had reported being bitten by a bat.

On 7 July 2004, the CDC was notified of an additional organ transplant patient at BUMC who had died of encephalopathy of unknown origin in early June 2004. This case was detected as part of an ongoing review of transplant-patient autopsies. The patient, who had end-stage liver disease, had received a liver transplant at BUMC in early May 2004. The patient remained hospitalised, with transplant-related complications, and began having neurologic abnormalities in early June 2004, progressing to seizure, coma, and death. On 7 July 2004, pathologists at BUMC identified intracytoplasmic inclusions, suggestive of rabies, in neurons in multiple areas of the brain.

Specimens from the recipient were sent to the CDC on 7 July 2004, and direct fluorescent antibody and immunohistochemical staining procedures confirmed the presence of rabies viral antigens in multiple areas of the brain, including the hippocampus, midbrain, pons, medulla, and cerebellum. Similar to the findings with the three previously known rabies-infected transplant recipients, preliminary antigenic characterisation of the agent was consistent with a rabies virus variant associated with insectivorous bats. On 8 July 2004, CDC laboratory testing of tissues and serum from the donor who provided the liver yielded no evidence of infection with rabies virus.

Review of surgical procedures at BUMC determined that a segment of iliac artery, recovered from the donor subsequently determined to have rabies, had been stored at the facility for future use in liver transplants. This artery segment subsequently was used in the transplantation of the liver in the most recently

identified rabies-infected recipient. Investigation of rabies transmission sources is ongoing, although current evidence suggests that the artery segment originating from the rabies-infected donor likely is the source of the latest rabies infection. Identification of contacts of this liver recipient is under way, and initiation of PEP when indicated, or, as appropriate, is in progress.

Reference

1. Centers for Disease Control and Prevention. Investigation of rabies infections in organ donor and transplant recipients—Alabama, Arkansas, Oklahoma, and Texas, 2004. *MMWR Morb Mortal Wkly Rep* 2004;53:586–589.

Avian influenza: genesis of H5N1 epidemic

Source: World Health Organization, CSR, Disease Outbreaks News, 8 July 2004 [edited]

In the last two weeks, avian influenza appears to have re-emerged in poultry in several countries in Asia. These outbreaks could either be new outbreaks of highly pathogenic avian influenza A(H5N1) virus, or, a continuation of the outbreaks first reported earlier in 2004. These events, in addition to two new research reports—about the virus becoming increasingly pathogenic and becoming more widespread in birds in the region—fuel WHO's concern about the threat the virus poses to human health.

WHO has been concerned about the influenza A(H5N1) virus, because of its threat to humans, both in farm settings in Asia, and its greater, potentially global risk. Several countries in Asia have witnessed this virus crossing the species barrier—moving from infected chickens or ducks directly into humans—in three documented outbreaks since 1997. These direct human infections have produced severe, and sometimes fatal, outcomes. Moreover, the virus has the potential to acquire the ability to spread easily from human to human, thus triggering a global influenza pandemic.

Two research reports have now been added to our understanding of this virus. Firstly, members of China's Ministry of Agriculture, and colleagues reported in a paper published in July 2004 in the Proceedings of the National Academy of Sciences, that the virus appears to be widespread in domestic ducks in southern China. Further, the scientists found that the virus is causing increasingly severe disease. However, these trials were done in mice and may not have a direct implication for humans.

In July 2004, the journal *Nature* published a report which indicates domestic and wild birds in the region may have contributed to the increasing spread of the virus, and suggests that the virus is gaining a stronger foothold in the region. These observations suggest that control of the virus may be even more difficult than thought in the spring of 2004.

Effective risk management tools exist to control outbreaks of influenza A(H5N1) virus infection, when they are detected in poultry operations. China, for example, was quick to employ these tools during July 2004, when an outbreak was discovered in Anhui province. These risk-management measures include the culling of infected and exposed birds, stringent biosecurity measures, and vaccination. While this approach can still take months or even years to contain the virus completely, these methods have been effective in the past.

However, tools to assess the risk to human health are less well developed. While recent reports indicate the virus has been present consistently in the environment for the last several years, it has still not acquired the ability to infect humans easily. Why? Is there something about this virus which resists this development? Given the recent reports, WHO urges and offers assistance, that such risk-assessment activities—including surveillance in animals and humans, and strain analysis—be undertaken as soon as possible.

More knowledge of the virus could be acquired if WHO had full access to all virus isolates and clinical specimens from recent outbreaks. All H5N1 viruses are not the same and how they differ could provide important insights. For example, the work reported in *Nature* suggests that the Indonesian avian influenza virus, while belonging to the genotype of viruses seen in Viet Nam and Thailand, is also distinct. What, if any, impact does this difference have? With this information, public health planners would know that they are confronting the same virus in all of the recent outbreaks in Asia. This is another set of the many questions that need to be answered imperatively.

Pandemic preparedness activities started by WHO in the wake of the outbreaks reported earlier in 2004 continue. At the end of June 2004, WHO hosted a meeting in Kuala Lumpur with experts from 13 countries and areas of the Asia-Pacific region. Among other activities, the meeting participants were provided with a WHO preparedness self-assessment tool. WHO is collaborating with scientists and the pharmaceutical community on a global surveillance system to monitor changes in the virus' susceptibility to known antivirals. Finally, pandemic vaccine development continues. Two vaccine manufactur-

ers, both based in the United States of America, have produced a supply of trial vaccine which will be tested for safety and efficacy in humans.

In summary, recent developments suggest that:

- the virus is more widespread than previously thought and found in wild birds, and therefore, it may be more difficult to eliminate;
- virus isolates and specimens from all recent outbreaks need to be shared with the WHO laboratory network to monitor the circulating viruses and to assess the adequacy of the current pandemic vaccine strain;
- as control measures are strengthened, national governments are encouraged to provide human influenza vaccinations to culling workers;
- all people, especially culling workers, exposed to infected birds need to be provided with antivirals;
- human trials of experimental influenza pandemic vaccines should be accelerated; and
- while early identification of avian influenza cases in humans is difficult, stepped up surveillance for the early detection of the disease in humans is essential.

Bovine spongiform encephalopathy (BSE) update 2004

Source: BSE in Europe, updated 14 May 2004 [edited]. Available from: http://home.hetnet.nl/~mad.cow/

These are not official data. The data from some countries may include exceptional imported cases or exclude exported cases which were found positive in the countries of destination. During 2003, decreased incidence of recorded Bovine spongiform encephalopathy cases, compared to 2002, was seen in most countries. The exceptions were Portugal, Spain, Japan, Poland, and the Czech Republic.

For additional information, the reader is referred to the source and to the Office International des Epizooties (OIE) table. Available from: http://www.oie.int/eng/info/en_esbmonde.htm

Bovine spongiform encephalopathy update 2004

Country	2001	2002	2003	2004	Total since 1987
United Kingdom	1,175	1,104	611	76	182,547
Austria	1	0	0	0	1
Belgium	46	38	15	7	124
Canada	0	0	1	0	1
Czech Republic	2	2	4	2	10
Denmark	6	3	2	0	13
Finland	1	0	0	0	1
France	274	239	137	27	919
Germany	125	106	54	21	319
Greece	1	0	0	0	1
Ireland	246	333	182	59	1,417
Israel	0	1	0	0	1
Italy*	50	36	31	1	120
Japan	3	2	4	2(1)	11
Liechtenstein	0	0	0	0	2
Luxembourg	0	1	0	0	2
Netherlands	20	24	19	5	76
Portugal	110	86	133	17	879
Poland*	0	4	5	5	14
Slovakia*	5	6	2	2	15
Slovenia	1	1	1	1	4
Spain	82	127	167	33	428
Switzerland	42	24	21	0	453
United States	0	0	1*	0	1

* An imported case (from Canada).

Source: BSE in Europe, updated 14 May 2004.Available from:http://home.hetnet.nl/~mad.cow/