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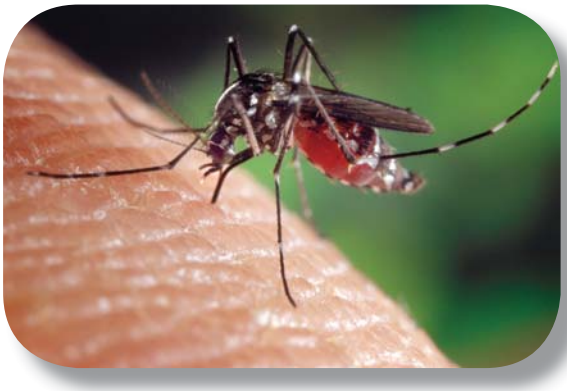
Communicable Diseases Intelligence



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Clockwise from top left: spring rolls are a common source of foodborne disease, M Hilpertshauser; Female *Aedes albopictus* mosquito, J Gathany; Laboratory examination of agar-filled Petri dish, J Gathany.

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An evaluation of the Australian Sentinel Practice Research Network (ASPREN) surveillance for influenza-like illness

Hazel J Clothier,[1] James E Fielding,[2] Heath A Kelly[3]

Abstract

The Australian Sentinel Practice Research Network (ASPREN) is a national network of general practitioners (GPs) who collect and report data on selected conditions, including influenza-like illness (ILI). The Australian Government Department of Health and Ageing initiated an evaluation of ASPREN, aiming to assess its potential to contribute to surveillance of emerging infectious diseases including pandemic influenza. System attributes and utility for decision-making were elucidated from stakeholder surveys. ASPREN ILI data for 2002 to 2004 were compared with ILI data from South Australia and New South Wales. In 2004, 50 GPs participated in the ASPREN surveillance, with proportionately more in New South Wales (30%) and South Australia (30%) than in other states. The majority (78%) of GPs were in metropolitan practices. Compliance with the manual data collection system was not optimal, nor consistent by state. ASPREN ILI data compared favourably with that of other surveillance systems. No formal structures were in place by which to assess data trends, provide alerts or initiate public health action. To maximise the contribution to biosecurity surveillance, ASPREN would require targeted GP recruitment to achieve geographic representativeness; exploration of alternative technologies for data collection and reporting; provision of committed resources adequate for system operation; and negotiation with state-based public health reference laboratories to provide laboratory support. The main potential of ASPREN is to permit rapid dissemination of a syndromic case definition and acquisition of nationwide community level clinical presentation data. These evaluation findings will be used to inform redevelopment of ASPREN as part of the Biosecurity Surveillance System project. ***Commun Dis Intell*** **2005;29:231–247.**

Keywords: evaluation, influenza, disease surveillance, biosecurity

Introduction

Influenza, a communicable disease that spreads rapidly, is an important global public health problem. While seasonal activity poses an ongoing burden on medical resources through increased numbers of general practitioner (GP) consultations and hospital admissions, and on the community through lost days of work, the ever-present threat of a pandemic has heightened awareness of the need for influenza surveillance.

The implications of an influenza pandemic are extreme, with the global attack rate for the 1918–1919 pandemic estimated to be 25 per cent.[1] In Australia, the most recent pandemic of 1968 had a similar attack rate of 25–30 per cent, predominantly affecting those aged over 65 years.[1] In order to lessen the impact of pandemics and enable planning measures to be rapidly implemented, much effort has been spent on early or rapid detection of influenza epidemics and characterisation of circulating virus strains. The need for pandemic planning and an effective national surveillance system has been highlighted recently by infection of humans in Viet Nam and Thailand with highly pathogenic avian influenza that has shown evidence of limited person-to-person transmission.[2,3]

The World Health Organization (WHO) established a global influenza surveillance network in 1952 that now comprises 112 institutions in 83 countries.[4] Australia participates in the WHO global network through the WHO Collaborating Centre for Influenza Reference and Research in Melbourne and three

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designated national influenza centres in Melbourne, Perth and Sydney. There are also several influenza surveillance systems operating in Australia that inform national and jurisdictional public health authorities about influenza epidemiology.[1,5]

Laboratory-confirmed influenza was listed as a nationally notifiable disease in 2001.[6] De-identified data from each state and territory are collated and reported to the National Notifiable Diseases Surveillance System.[7] The Laboratory Virology and Serology Reporting Scheme (LabVISE) also collects data on laboratory-confirmed diagnoses from participating laboratories.[8]

Sentinel practice surveillance systems aim to monitor influenza activity in the community. Cases are ascertained by diagnosis of clinical influenza-like-illness (ILI), defined since 2004 by the nationally adopted ILI case definition of fever, cough and fatigue.[9] State-specific sentinel practice surveillance systems are also operated in New South Wales, the Northern Territory, Queensland, Victoria and Western Australia. Laboratory confirmation of influenza in a sample of ILI diagnoses reported is an additional component of the Victorian and Western Australia systems.[10] The Australian Sentinel Practice Research Network (ASPREN) aims to conduct surveillance across all states and territories.

Evaluation framework

This evaluation commissioned by the Australian Government Department of Health and Ageing, aimed to assess the utility of ILI surveillance conducted by ASPREN, in the context of the Biosecurity Surveillance System requirements.

Aim and objectives

The evaluation was conducted between December 2004 and March 2005 with objectives to:

1. provide a comprehensive summary of how the surveillance system operates through information provided by ASPREN representatives;
2. assess the simplicity, flexibility, acceptability, timeliness and stability of ASPREN ILI surveillance from information provided by ASPREN representatives, GPs who participate or have participated in ASPREN, and users of ASPREN data;
3. assess the data quality of the system by examination of ASPREN data from 2002 to 2004;
4. assess the representativeness of the system by comparison of ASPREN data from 2002 to 2004 with other influenza-like illness surveillance systems in New South Wales and South Australia, and
5. make recommendations to improve the system consistent with existing uses.

Methods

This evaluation of ASPREN, with particular reference to ILI surveillance, was conducted using the principles from the Centers for Diseases Control and Prevention *Updated Guidelines for Evaluating Public Health Surveillance Systems*[11] and the *Framework for Evaluating Public Health Surveillance Systems for Early Detection of Outbreaks.*[12]

The processes and operation of the system at the administrative level were elucidated by informal interviews with: staff at the Royal Australian College of General Practitioners (RACGP) in Adelaide; the ASPREN director in the Department of General Practice, the University of Adelaide; and two previous ASPREN directors. Additional stakeholders were identified from the data distribution list and asked standard questions to ascertain the current use of ASPREN data.

A postal survey of current (2004) and former ASPREN-participating GPs assessed the system performance attributes of usefulness, acceptability and stability. The survey also collected information about GPs' opinions for improving the system and whether its expansion to collect data on additional conditions would be acceptable.

Data analyses comparing ILI diagnoses and laboratory-confirmed influenza data by time and age group (where available) between ASPREN and other influenza surveillance system data in South Australia and New South Wales were performed using MS Excel and STATA version 8. Sentinel practice locations were categorised as metropolitan or regional according to Australian Metropolitan Postcodes.[13] Population data were accessed from the Australian Bureau of Statistics for the 2004 mid-year estimated resident population,[14] and the National Regional Profile and Remoteness Structure from the 2001 census.[15] We defined two geographical categories: 'metropolitan' included major cities and inner regional areas; and 'regional' included the three remaining categories of outer regional, remote and very remote.

Published and unpublished reports using ASPREN data were reviewed. Evaluation reports for other Australian influenza surveillance systems were reviewed (New South Wales,[16] South Australia,[17] Western Australia,[18] NNDSS[7] and Victoria[19]) and the recommendations from these evaluations were considered for their applicability to ASPREN.

Purpose and operation of the system

The Australian Sentinel Practice Research Network is a surveillance system that is owned and operated by the RACGP and managed by its South Australian

and Northern Territory Faculty in Adelaide. Since the mid-1990s, the Director of ASPREN has been based in the Department of General Practice at the University of Adelaide but maintains strong links with, and is a member of, the RACGP. Since 2004, the University of Adelaide has made a financial contribution to the running of ASPREN and is considered a full partner in the enterprise by the RACGP. The current director of ASPREN is a member of the RACGP National Standing Committee on Research.

Objective

ASPREN was established by the RACGP as a national surveillance system in 1991. Each year, a meeting of interested bodies—including RACGP members, academic GPs and epidemiologists—selects 10 to 12 conditions for surveillance. The original objectives of the surveillance program were to:

- provide a rapid monitoring scheme for infectious diseases that can also serve to warn public health officials of epidemics in their early stages;
- provide information about conditions that are seen in general practice;
- measure changes over time for conditions that present to medical practitioners;
- help answer research questions; and
- measure the impact of public health campaigns.

Some conditions such as ILI and measles were listed for surveillance with the intention for ongoing inclusion, whereas others, such as those to answer research questions, were short-term. ILI has been included in the list of reported conditions annually since 1991.

Stakeholders

Stakeholders of ASPREN include:

- The Royal Australian College of General Practitioners;
- current and former Directors of ASPREN;
- current and former participants in ASPREN;
- the Department of General Practice, the University of Adelaide.

The users of the ASPREN data include:

- the Editor of *Communicable Diseases Intelligence,* Australian Government Department of Health and Ageing;
- Communicable Disease Control Branch, Department of Health, South Australia;
- the WHO Collaborating Centre for Influenza Reference and Research;
- researchers from the Department of General Practice, Flinders University; and
- researchers from the University of Western Australia and the University of Melbourne.

Recruitment of GPs

Participation of GPs in ASPREN is voluntary and has been since the program's inception in 1991. For the 2002 to 2004 triennium this activity was approved for 20 RACGP QA-CPD category 1 (clinical audit) points and 56 GPs received points for completing the requirements. In instances where GPs did not complete the contribution, points were awarded on a pro rata basis. The RACGP is yet to make a determination on the points to be awarded for ASPREN participation in the forthcoming 2005 to 2007 triennium, or on the precise requirements for achieving approved points. However, it is likely that a minimum of 30 points will be awarded for the full triennium participation.

Active recruitment of GPs for ASPREN has not been undertaken for several years due to uncertainties about the future of ASPREN and lack of resources. Previously, GP recruitment occurred via bulletins and mail-outs to practices, and advertisements in the RACGP's 'Friday Fax' bulletin to its members. Due to the decline in participating GPs it has been inappropriate and not possible to exclude participants in order to improve the representation by location.

Reportable conditions

The list of reportable conditions and their specific case definitions are mailed to participating GPs at the start of each year along with documentation describing the ASPREN system and reporting requirements. In most years there have been 12 reportable conditions, although there were 13 in 2003 and 14 in 2000.

Data collection

In addition to the list of reportable conditions and associated documentation, each participating GP receives three-monthly batches of reporting forms, with the week number, GP's name and doctor code already completed, and a supply of reply-paid envelopes. For each patient meeting one of the ASPREN condition criteria, the GP is required to record the sex, age bracket and ASPREN-reportable condition by filling in boxes on the form. There are 40 columns

into which patients with ASPREN reportable conditions can be recorded each week. The doctor must also record the total number of consultations made in that week. The form is then folded in a particular way (marks are provided on the form where folds should be made so they scan correctly) and mailed back in the reply-paid envelope. Electronic reporting is not available and forms cannot be returned by facsimile as they cannot be scanned.

The end of the surveillance week is Sunday, and most data collection forms are returned to the RACGP by the following Wednesday. The RACGP administration officer manually checks each form prior to scanning to ensure data points will scan. Records that do not scan properly are amended and an output generated in Microsoft DOS. A report is then automatically generated in both Microsoft Word and Excel formats that provide the number of patients and rates of ILI diagnoses and other ASPREN reportable conditions (measured per 1,000 consultations). The report stratifies the rates by state/territory and age-group and sex.

There is no legal authority for the collection of ASPREN data. No approval to conduct ASPREN surveillance has ever been sought from Human Research Ethics Committees. This is largely based on historical precedent but has also been justified on the grounds that participation in ASPREN is voluntary and the limited patient data collected are anonymous. However, ethics approval or provision of informed patient consent to collect and use their data may need to be considered in light of increasingly stringent privacy provisions.

Reporting and dissemination

The reports generated in Microsoft Word format are disseminated to those on the mailing list on the same day as the data entry process. Recipients of the data include: the Surveillance Section of the Australian Government Department of Health and Ageing; the Communicable Disease Control Branch, Department of Health, South Australia; university researchers from Departments of General Practice and Rural Health; a medical news reporter from Medical Observer; a representative from CSL; and, the administration officer from the WHO Collaborating Centre for Reference and Research on Influenza.

ASPREN data are published quarterly in *Communicable Diseases Intelligence* (monthly publication prior to 2001). An ASPREN annual report provides an overview of statistical data, including reporting practices of GPs and reported rates for the conditions under surveillance. These data may be stratified into age-, sex- or state/territory-specific rates and compared to rates observed in previous years as part of more in-depth analysis. The reporting format was upgraded in 2002 and is reflected in some of the evaluation analyses. Selected ASPREN findings have been published in the *Australian Family Physician*; however, this is not a regular occurrence.[20,21]

ASPREN ILI data are one of four data sources reported in the National Influenza Surveillance Scheme.[22] Graphical presentation of ASPREN ILI data per 1,000 consultations is available via the Australian Government Department of Health and Ageing (DoHA) website, which is updated fortnightly during the influenza season (http://www.health.gov.au/internet/wcms/Publishing.nsf/Content/cda-surveil-ozflu-flucurr.htm).

Resources required to operate system

Three personnel contribute part-time to the management and operation of ASPREN. The clinical director of ASPREN is based at the Department of General Practice, the University of Adelaide and spends approximately one to two hours per week working on ASPREN, although this may be more during production of the annual report and mail-outs, and less at other times in the year. The day-to-day operation involves two RACGP staff members based in the South Australian and Northern Territory Faculty office in Adelaide and overseen by the Faculty manager. The administrative officer spends approximately three to four hours per week receiving, checking and scanning the data collection forms and emailing the reports to those on the distribution list and the project officer spends approximately one day per month troubleshooting computer problems, coordinating mail-outs of annual reports and data collection forms and liaising with the ASPREN administrator and Director. Technical support and maintenance of the scanner is provided by a contract computer technician/programmer; the annual cost for which is from $3,000 to $4,000 per annum.

There is little direct financial support provided for the operation of ASPREN. The ASPREN director's time spent working on the system is voluntary and the unit of the RACGP of which ASPREN is part absorbs salaries for the RACGP personnel. In 2004, the Department of General Practice at the University of Adelaide (in which since 1996, the two ASPREN directors have worked) provided $5,000 from a Primary Healthcare Research Education and Development grant to the RACGP to help cover the administrative costs of maintaining the system. The GPs who participate in the surveillance do not receive payment for their time.

Data analysis

GP participation and reporting practices

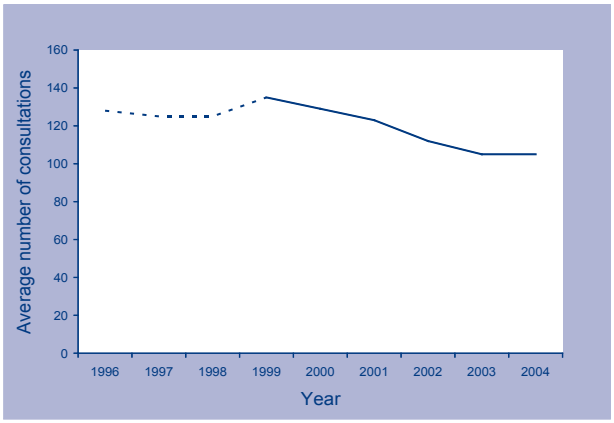
ASPREN annual reports from 1992 to 2002 were available for review. Preliminary data analysis completed in preparation for the 2003 annual report was provided by the ASPREN director, in addition to raw data for 2003 and 2004. Due to changes in the annual report format, and therefore the information available, comparisons were made from 1996 to 2004 with more detailed analysis done for 2003 and 2004.

The number of GPs participating each year has declined from a peak of 110 (1994) to 51 (2004). The average number of weekly consultations per GP has also declined (Figure 1). Data about the total number of consultations monitored were not collected from 1996 to 2001; however, a decline of 41 per cent between 2002 (296,342) and 2004 (173,870) was observed, possibly a reflection of increased consultation length.

As the number of participating GPs has declined, so has the number of forms returned each year (Table 1). The form return rate varied by week throughout the year. The lowest weekly return rate occurred consistently in weeks 52 and one, which correspond to the Christmas and New Year period (Figure 2).

The number of participating GPs decreased from 73 in 2003 to 51 in 2004. The average form return rate varied between 81 and 87 per cent from 1996 to 2000, but had declined to 60 per cent in 2004 (Table 1).

Figure 1. Average number of consultations per general practitioner per week,* 1996 to 2004



* Interrupted line indicates estimated value as reported in the respective annual report.

Figure 2. Number of report forms returned each week, ASPREN, 2003 and 2004

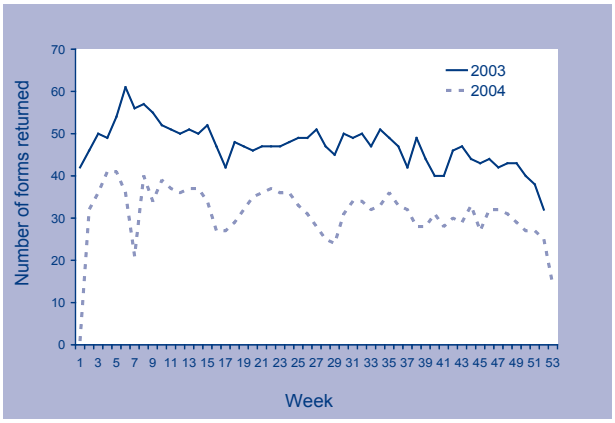


Table 1. Number of participating general practitioners and average number of forms returned per general practitioners, ASPREN, 1996 to 2004

Year	Number of forms returned	Number of participating general practitioners	Average number of forms returned per general practitioners	
			n	%
1996	3,427	81*	42	81
1997	3,168	71*	45	87
1998	2,763	62*	45	85†
1999	2,397	55*	44	85
2000	2,821	66*	43	83
2001	2,754	71*	39	75
2002	2,654	91	29	56
2003	2,456	73	34	65
2004	1,654	50*	33	60†

* When the number of participating general practices was not specifically stated in the annual report the figure was estimated from the maximum number of general practitioners reporting in any one week.

† Years with 53 weeks.

The form return rates were not consistent across states; South Australian, Queensland and Australian Capital Territory GPs had a twofold higher rate than Tasmania (Table 2).

The majority of GPs participating in ASPREN have been practising in metropolitan areas. Participation, as determined by the form return rate, was the same for both groups, although regional GPs had a lower average number of consultations per week (Table 3).

As a proportion of all consultations, those in which an ASPREN-reportable diagnosis was made was approximately 10 per cent during 1996 to 1999 but varied more in subsequent years, ranging from a high of 12.8 per cent in 2002 to a low of 5.7 per cent in 2004. Given that there were 13 reportable conditions in 2004, this drop in the proportion of ASPREN reportable conditions may be an indication of incomplete data collection by the participating GPs.

ASPREN surveillance for influenza-like illness

ILI diagnoses are presented as rates (measured as cases per 1,000 consultations). The peak rate usually occurred around week 30 (end of July) of each year, although outliers included week 23 in 1992 and week 37 in 2000 (Table 4). In general, the ILI season was observed between weeks 15 and 40 each year.

Table 4. Peak rates of influenza-like illness reported by ASPREN, 1991 to 2003

Year	Peak rate of influenza-like illness per 1,000 consultations*	Peak week number	Proportion of influenza cases diagnosed in those aged greater than 64 years
1991	24.9	30	n/a
1992	18.5	23	n/a
1993	22.0	34	n/a
1994	37.2	31	n/a
1995	28.4	25	n/a
1996	30.8	29	n/a
1997	33.8	31	8.1
1998	34.5	27	7.7
1999	17.5	34	8.2
2000	25.0	37	7.3
2001	15.5	30	5.6
2002	16.9	28	4.4
2003	25.0	34	6.3

* ASPREN case definition (see Box 1).

n/a Not available.

Table 2. Number of participating general practitioners and form return rate, ASPREN, 2004, by state

State	Number of participating general practitioners	Number of forms returned	Average number of forms per general practitioner	Proportion of all possible forms returned %
ACT	1	45	45	85
NSW	15	488	33	61
Qld	5	193	39	73
SA	15	543	36	68
Tas	4	73	18	34
Vic	9	247	27	52
WA	2	65	33	61
Total	51	1654	32	60

Table 3. Comparison of metropolitan and regional based ASPREN participating general practitioners, 2004

	Number of general practitioners	Total consultations	Average consultations per week	Average number of forms returned per general practitioner (%)	
				n	%
Metropolitan	37	132,564	110	32	60
Regional	14	41,306	91	32	60
Ratio M:R	2.6:1	3.2: 1	1.2: 1	–	–

M = Metropolitan.

R = Regional

Box 1. International Classification of Health Problems in Primary Care influenza-like illness case definition

Inclusion requires one of the following:

a. viral culture or serological evidence of influenza virus infection; or

b. influenza epidemic, plus *four* of the criteria in (c); or

c. *six* of the following:

i. sudden onset (within 12 hours);

ii. cough;

iii. rigors or chills;

iv. fever;

v. prostration and weakness;

vi. myalgia, widespread aches and pains;

vii. no significant respiratory physical signs other than redness of nasal mucous membrane and throat;

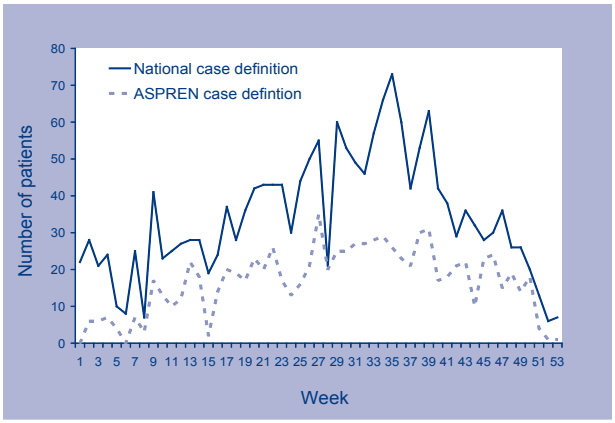
viii. influenza in close contacts.

Influenza-like illness case definition

Since its inception, ASPREN has used the International Classification of Health Problems in Primary Care (ICHPPC-2) ILI case definition (Box 1).[23] During 2004, ILI was reportable using either or both of two different case definitions; patients meeting the ASPREN case definition as above and/or the 2004 nationally agreed ILI surveillance case definition of fever, cough and fatigue.[9]

The nationally agreed ILI case definition, included in the 2004 ASPREN surveillance alongside the previous ICPPHC-2 case definition, increased the number of ILI diagnoses reported. Whilst the new case definition was apparently less specific, the overall seasonal pattern of ILI did not change (Figure 3).

Figure 3. Comparison of the two clinical influenza-like illness case definitions used in 2004

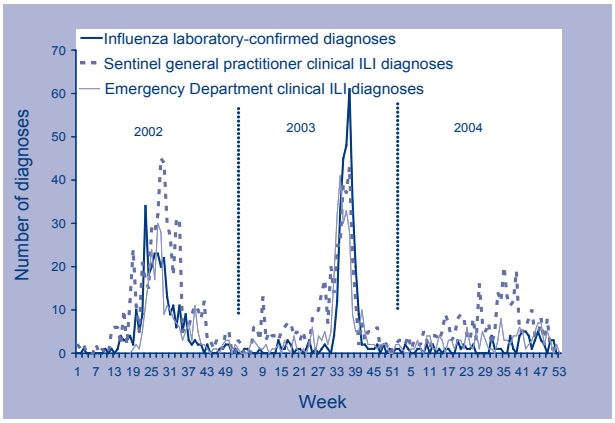


Comparison of ASPREN influenza-like illness surveillance with state-based influenza-like illness surveillance in New South Wales and South Australia

ASPREN ILI data were compared with data from the New South Wales and South Australian influenza surveillance programs; these two states having the highest proportion of ASPREN GPs. ASPREN ILI data recorded using the national case definition were used for the 2004 comparison.

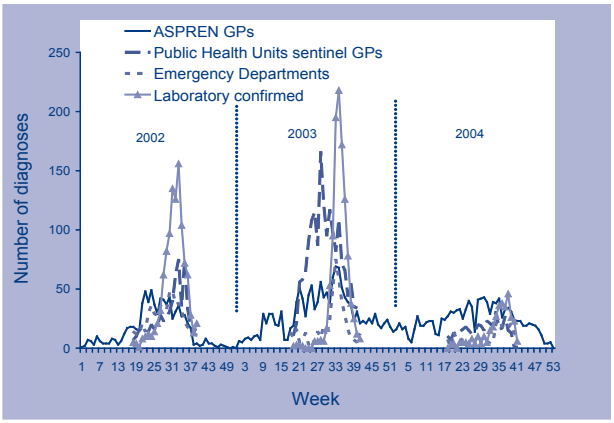
Influenza activity in South Australia is monitored through notifications of laboratory-confirmed influenza and clinical diagnoses of ILI in emergency department attendees in addition to the ASPREN ILI data. ASPREN ILI data provided the earliest indication of the onset of seasonal influenza for each of the three years in the review period (2002 to 2004) (Figure 4). However, as the case definition for ILI is non-specific, the increased activity indicated by sentinel practitioner diagnoses in 2004, which was not supported by a rise in laboratory-confirmed influenza notifications, may have been due to non-influenza respiratory illness.

Figure 4. Influenza clinical and laboratory diagnoses, South Australia, 2002 to 2004, by week



New South Wales influenza surveillance comprises diagnoses of clinical ILI by sentinel GPs through the public health units (PHU) and GPs participating in ASPREN; 12 hospital emergency departments from within the greater Sydney region; and laboratory-confirmed influenza diagnoses collected via the direct virological surveillance system (the latter ceased in 2003) (Figure 5). Surveillance via the PHU sentinel GPs and emergency departments is conducted from May to October each year. In 2004 the PHU sentinel GPs used the nationally agreed ILI case definition; prior to 2004 ILI was defined using an ASPREN-like case definition of: cough and myalgia and no abnormal respiratory physical signs other than redness of nasal mucous membranes and throat; and two of the following: sudden onset; rigours or chills or fevers; prostration or weakness; or influenza in close contact.

Figure 5. Influenza clinical and laboratory diagnoses, New South Wales, 2002 to 2004, by week



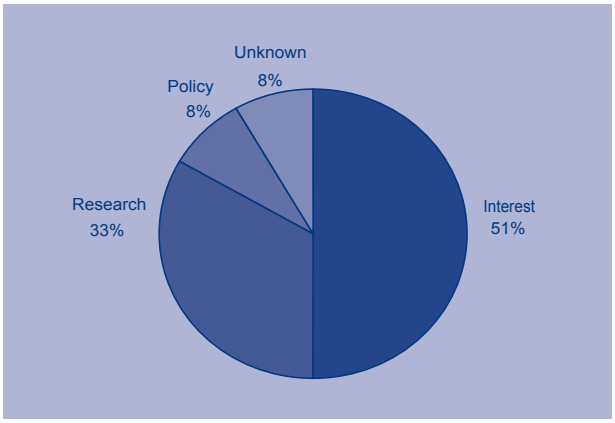
Surveillance system attributes

System attributes were elucidated from stakeholder interviews conducted with the current and former operators of the system; 11 of the 12 individuals or institutions that received ASPREN data each week; and a postal survey of current and former participating GPs. The GP survey response rate was 93 per cent (91/98) overall with 98 per cent (49/50) of current and 88 per cent (42/48) of former ASPREN GPs responding (three were no longer at the same address, therefore data were available for 39 former GPs).

Usefulness

Only one research paper using ASPREN data cited in published literature was identified;[20] however, the system operators perceive that ASPREN data are accessed and used by researchers in support of their work. The 1999 and 2000 ASPREN annual reports listed specific requests for ASPREN data having been received from pharmaceutical companies (influenza), a RACGP training programme registrar (rubella, measles, pertussis and Ross River virus), the Monash Medical School Clinical Research Centre for Water Quality and Treatment (gastroenteritis in Melbourne) and the New South Wales Department of Health (influenza). Of those receiving weekly data, the majority (6/12) do so for personal interest. Four receive the data specifically to support research activities and one institution utilises the data to inform policy, primarily in regard to identification of at-risk groups for vaccination campaigns (Figure 6).

Figure 6. Primary use of ASPREN data, ASPREN, 2004, by weekly recipient list



ASPREN data are published in the quarterly *Communicable Diseases Intelligence* publication and posted on the Communicable Diseases Australia website. The WHO Influenza Centre include ASPREN influenza data in their bi-annual WHO reports. It was not possible to determine how these published data are utilised, however, there is anecdotal evidence of media interest in data accessible via the DoHA website (personal communication: Paul Roche, DoHA).

Due to its biased geographic representativeness (see below) and its current format, it is likely that ASPREN data are neither as useful nor as well utilised as they might be.

Simplicity

ASPREN has been operating for 15 years and is administered with minimal resources, indicating the simplicity of the system. Participating GPs were well aware of the objectives of the system and 80 per cent (39/49) of current GPs perceived participation to be easy. Although only 28 per cent (11/39) of former GPs perceived participation to be easy, the main issue was finding time to do administrative tasks within a busy practice schedule, rather than the complexity of the system itself.

Completion of the forms is uncomplicated, however, remembering the detailed criteria for a reportable condition was an issue raised by both current and former GPs.

Flexibility

Each year new forms, listing the reportable conditions for that year, are prepared and distributed to the participating GPs. In theory the system has the capacity to add reportable conditions at three-month intervals. There is therefore potential flexibility to add new conditions (such as emerging infections) not included in the annual review, although this has never been tested. The process of reprinting and mailing revised data collection forms to all participating GPs would not only require considerable expense, but may also result in confusion among the GPs due to duplicate versions of the form and therefore poorer data quality.

The majority, (80%, 39/49) of current participating GPs were willing to extend surveillance to additional conditions (such as SARS) if requested to do so. This could be facilitated by leaving a blank section in the conditions list that could be used for other new or urgent conditions to be added upon request.

Data quality

Returned forms are checked manually and problems relating to ability to scan the forms are resolved at that time. Data recording issues identified include:

- total number of consultations missing;
- total consultations equal the number of patients reported with ASPREN conditions;
- age-category missing; and
- condition category missing.

The data quality will also be affected by the adherence to the specific case definition criteria for the reportable conditions; it was not possible to assess this. Equally it was not possible to assess completeness of data collection. However, the decline in the proportion of ASPREN reported conditions compared to total consultations, from an average of 10 per cent between 1996 and 2003 to 5.7 per cent in 2004 could be indicative of incomplete reporting, or alternatively that fewer or less common conditions were selected for surveillance in that year.

Initiatives to improve GP reporting and data quality have not been undertaken recently. The system should be adequately resourced to permit follow-up of incorrect or incomplete forms. The clarity of condition definitions should be considered carefully to facilitate rapid and accurate recall, particularly if the condition is rare.

Acceptability

Acceptability was ascertained from three sources: retention of participating GPs; the number of forms returned by participating GPs; and through the responses to direct questioning in the survey of current and former participating GPs.

The number of participating GPs has declined by 45 per cent over recent years from 91 in 2002 to 50 in 2004. The decision to leave ASPREN was, for the majority (76%, 28/37) of former participants surveyed, due to time constraints rather than dissatisfaction with the system. More than a quarter (26%, 7/27) of these respondents cited specific events such as retiring or ill health that prevented their continued participation. Most (85%, 73/86) former and current participants described the importance of ASPREN as either very or somewhat important. The main reason given for participating was an interest in public health (80%, 70/88) and also to gain CPD points (51%, 45/88) [Note that more than one reason to participate could be cited].

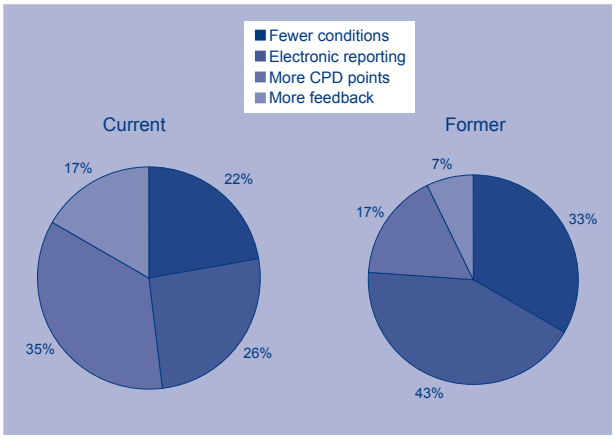
The highest form return rate for any year was 87 per cent (45/52) in 1997; however, this had steadily declined to 60 per cent (32/53) by 2004. Without mandatory zero reporting, it is not possible to account for this decline. GPs should be encouraged to return their forms even if they have not seen patients in that week.

The majority, 73 per cent (36/49), of practices use email and have access to the Internet (83%), mainly by broadband (54%). Whilst the majority, 63 per cent (31/49), of current participants were satisfied with the paper-based method of data reporting, 31 per cent (15/49) of current and 54 per cent (21/39) of former participants would be interested in electronic reporting. Antiquated data collection forms (boxes must be filled in) and collation methods (using a scanner) have become less appealing as familiarity with computer technology in practices has increased.

The level of GP satisfaction with the timeliness, content and delivery method of feedback was good; 82 per cent (40/49) and 74 per cent (29/39) of current and former GPs respectively stated they were either very satisfied or satisfied with these system attributes. The most frequently desired improvements were electronic reporting and electronic feedback (Figure 7).

In summary, ASPREN has a high level of acceptability, with the decline in participation being predominantly due to lack of resources to maintain recruitment. Increased use of technology may be required to maintain the level of acceptability.

Figure 7. Desired improvements to ASPREN identified by current and former ASPREN participants



Sensitivity

Sensitivity is the proportion of actual cases detected by the surveillance system and the ability to detect outbreaks and changes in the activity of influenza over time. The sensitivity is a function of diagnostic reliability and recording compliance and is therefore likely to be compromised by the behaviour of both patients and the participating GP. Although a disease of public health importance, individual clinical presentation of influenza may vary from mild to severe or atypical, affecting the treatment seeking behaviour of the individual patient and hence the opportunity to be detected by ASPREN surveillance. In addition, the manual paper-based method of data collection relies on GPs remembering to mark the appropriate boxes on the data collection forms when they see a patient who meets the case definition. There may be multiple factors that prevent this occurring, including how busy the doctor is, the ease of finding the ASPREN data collection form and the form's ability to act as a visual prompt.

Sensitivity is also dependent on the case definition used. In 2004 ASPREN moved towards using the nationally agreed case definition of fever, cough and fatigue. This case definition was determined to be 44–71 per cent sensitive and 47–80 per cent specific for influenza, over two influenza seasons characterised by influenza A H3N2 circulation in Victoria and Western Australia.[9]

Specificity

Increasing the sensitivity of an ILI case definition may compromise specificity; however, this can be overcome by combining clinical ILI surveillance data with laboratory-confirmed influenza data. State-based ILI surveillance systems in Victoria and Western Australia collect nose and throat swabs (NTS) from a sample of patients presenting with ILI to a sentinel practitioner. Sampling is either at the GPs' discretion (Victoria) or from the first ILI patient presenting on specified days (Western Australia). NTS are transported to the state reference laboratory in viral transport medium and analysed by multiplex polymerase chain reaction for viral respiratory pathogens, including influenza.[10] Data from the Victorian influenza surveillance program has demonstrated that up to 50 per cent of patients with an ILI will have laboratory-confirmed influenza.[24,25] While laboratory support provides the specificity that syndromic case definitions lack it also requires resources and coordination. When such conditions cannot be met laboratory supported surveillance is not recommended.[16] The use of rapid, point-of-care, influenza diagnostic platforms may revolutionise the capacity to confirm ILI diagnoses, or at least to exclude influenza when the test result is negative.[26] However, rapid point-of-care tests are generally not yet sensitive or specific enough, except for use where other tests are not available.

The ability to obtain NTS from a representative sample of ASPREN ILI reported cases and/or presentation of ASPREN ILI data alongside laboratory-confirmed influenza data sourced from other surveillance systems, or from rapid tests, should be considered.

Positive predictive value

The positive predictive value (PPV) is the proportion of cases reported by the system that actually have influenza. PPV is dependent on the laboratory tests used and the prevalence of disease: when influenza is prevalent in the community the PPV of clinical signs and symptoms increases. There is no international consensus on a case definition for ILI, although several exist, including those of WHO. The ILI case definition nationally accepted for Australia in 2004, was determined to have a PPV between 25 and 60 per cent in a setting of H3N2 influenza circulation.[9] A similar case definition of fever, cough and rapid onset was determined to have a higher PPV (35%) compared to the ICHPPC-2 case definition (18%) previously used by ASPREN, thereby confirming the validity of the new simpler case definition, at least in the elderly.[23]

Different strains of influenza, for example H1N1 and influenza B, may have milder presentations with less systemic symptoms and may therefore be systematically under-evaluated.[27] Non-respiratory symptoms must also be considered; gastroenteritis may be a clinical feature of human H5H1 avian influenza cases and SARS.[28–30] Modification to the case definition may be required when more specific information on the circulating subtype and clinical syndrome becomes known.

Representativeness

ASPREN, although aiming to be a national surveillance system, captures data predominantly from south-eastern Australia with New South Wales and South Australia having the highest number of sentinel practices in the network (Figure 8). This has been recognised with the following statement included in the 1998 to 2000 annual reports.

> 'Analysis of the reports on a weekly basis indicated that it is only possible to make comments on New South Wales, South Australia, Victoria and Queensland with any degree of reliability, as the other states have intermittent reporting.'

However, representativeness is misleading when assessed as the number of sentinel GPs per state. New South Wales and South Australia have the highest number of participating GPs, but when sentinel practices are considered against the resident population, South Australia and Tasmania are the only two states to reach the influenza pandemic plan target for metropolitan areas of one sentinel practice per 200,000[1] (Table 5). When viewed as a proportion of consultations monitored, South Australia has by far the highest percentage and Tasmania and New South Wales fall to third and fourth positions respectively: a reflection of the low rate of form return by the Tasmanian participants (Table 2).

The ratio of sentinel GPs in metropolitan and regional practices in 2004 of 2.6:1 approximates the ratio of metropolitan to rural resident population of 2.3:1.[15] No analysis to determine representativeness by socioeconomic status was undertaken.[31]

In order to improve geographic representativeness GP recruitment must consider the state, urban or regional locality and the number of consultations that will be monitored each year, in addition to a commitment to weekly reporting.

Representativeness of the participating GPs and the patients that they see compared to the general GP and Australian populations is also an important consideration but is not analysed further in this report.

ASPREN is managed by the RACGP with GP incentive provided through the award of RACGP CPD points. The RACGP has approximately 11,000 members, including over 3,000 in rural and remote Australia. A separate college, the Australian College for Rural and Remote Medicine (ACRRM) was established in 1997 (www.acrrm.org.au). ACRRM has approximately 2,000 members, representing approximately 50 per cent of rural medical practitioners in Australia. The ACRRM professional development program was formally accepted in its own right for maintenance of vocational recognition in 2002. ACRRM and RACGP use the same professional development triennium period: however, the award categories and required number of points per triennium differ. Points are not interchangeable.

Figure 8. Location of ASPREN participating general practitioners, 2004

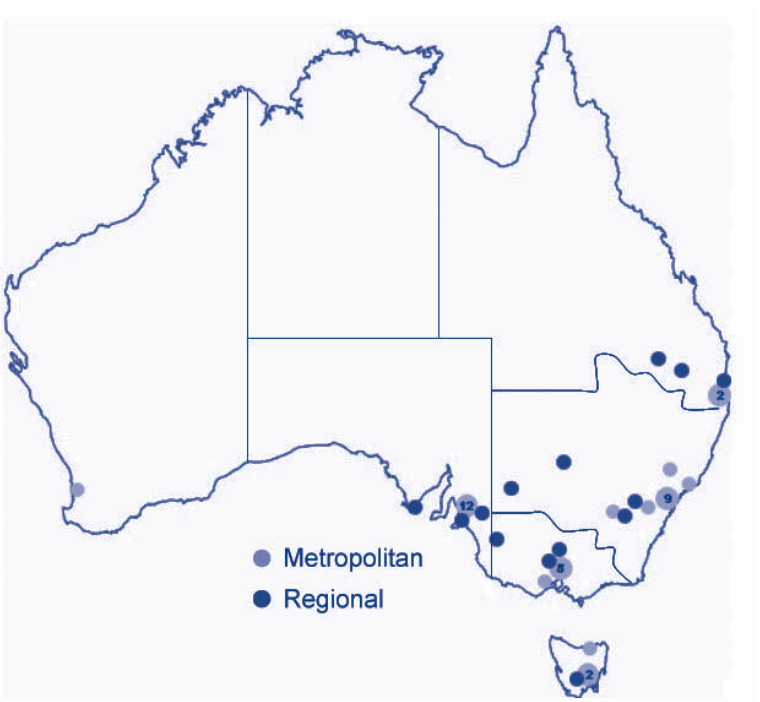


Table 5. Sentinel practices and consultations monitored through ASPREN, 2004, by state

State	Sentinel practices	Consultations	Population (ERP 2004)	Practices per 100,000	Consultations per 100,000	Practices required to attain one per 200,000 or (100,000)*
ACT	1	6,072	324,021	0.31	1,874	5 (2)
NSW	15	56,836	6,731,295	0.22	844	67 (34)
Qld	5	21,234	3,882,037	0.13	547	37 (19)
SA	15	48,943	1,534,250	0.98	3,190	15 (8)
Tas	4	8,845	482,128	0.83	1,835	5 (2)
Vic	9	25,941	4,972,779	0.18	522	50 (25)
WA	2	5,999	1,982,204	0.10	303	20 (10)
Total	50	173,870	19,908,714	0.25	873	199 (100)

* Pandemic planning target is one per 100,000 for regional and one per 200,000 for metropolitan areas.

ERP Estimated residential population. (ABS)

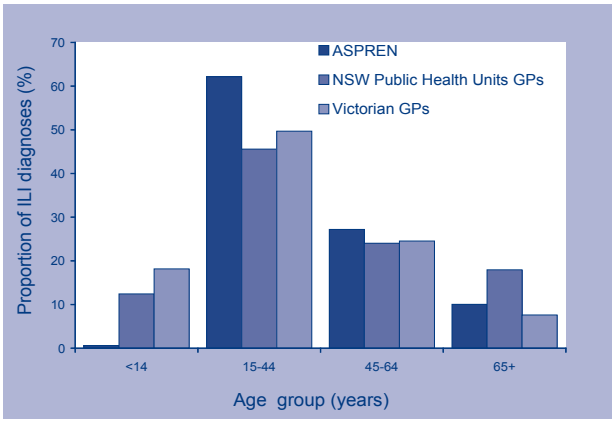
Recruitment of rural GPs is important to provide geographic representation; therefore it is also important that recruitment and incentives not be limited to a single professional organisation.

It is difficult to speculate if the poor representativeness of GP sentinel sites impacts on the representativeness of ILI reported by ASPREN compared to ILI cases presenting at GP surgeries across the country. However, the seasonal pattern of ASPREN ILI data is similar to that of the New South Wales and Victorian influenza sentinel GP surveillance programs (Figure 5),[32] and the age-group distribution of ASPREN is similar to that of the New South Wales and Victorian sentinel GP programs with the exception of under-representation of children in the ASPREN data (Figure 9). The ratio of male to female ILI diagnoses was also similar between the three surveillance systems (ASPREN 1:1, New South Wales 0.9:1, Victoria 0.8:1). This implies that the type of ILI patient seen by ASPREN GPs is not dissimilar to those seen by other GPs, except perhaps in the under-representation of children.

Timeliness

Retrospective analysis of influenza data from 2002 to 2004 indicates that ASPREN can achieve timely detection of increased influenza activity. However, data collection and reporting methods do not allow this information to be accessed in a timely way. Despite the manual paper-based data collection methods that ASPREN employs (forms returned by mail, scanning of forms) the turnaround from data collection to reporting of two weeks is quite reasonable. However, the timeliness of the system could be vastly improved by the adoption of new technologies such as web-based reporting or data extraction directly from practice software. This would also alleviate the problem created by slow return of data collection forms.

Figure 9. Proportion of influenza-like illness diagnoses, 2004, by age-group and sentinel GP surveillance system



Stability

ASPREN surveillance is a stable system that has operated for 15 years. The low turnover of administrative staff has facilitated consistency of the system.

Data have been collected by a loyal group of GPs with more than half (52%, 25/48) of current participants estimating their ongoing commitment to ASPREN being for 6 to 10 years or longer. The decline in participation over recent years has been compounded by cessation of active recruitment due to the uncertainty of resource availability for ASPREN to continue. If recruitment remains suspended the sustainability of the system may be compromised. The RACGP had implicitly recognised this situation and had appointed a project officer in 2004 to improve and expand the network, including reviewing the feasibility of electronic reporting. However, work had not commenced at the time of this evaluation.

Despite the personal commitment of the current administrative staff and participating GPs, without formal financial provisions to support necessary resources, the continued stability of ASPREN may be placed in jeopardy.

Discussion

ASPREN provides an established and stable framework for syndromic surveillance that is currently useful for monitoring selected endemic diseases in some areas of Australia. The potential for the system to contribute to national bioterrorism surveillance or detect an emerging infectious disease is dependent on the:

- ability to improve the system's representativeness;
- appropriate case definition attributes;
- timeliness and utilisation of data for decision-making; and
- availability of adequate resources for system redevelopment and management.

The findings of this evaluation provided 12 primary recommendations to maximise the potential ASPREN ILI surveillance contribution to the national Biosecurity Surveillance System (Box 2).

Representativeness and recruitment of sentinel practices

ASPREN currently provides ILI data comparable to other surveillance systems operating in south-eastern Australia. To be representative of communities throughout Australia, intensive recruitment will be required, with specific targeting of particular locations, accompanied by acceptable and appropriate incentives. Opening the recruitment process to the rural college ACRRM, may assist in ensuring geographical representativeness. Currently geographic representativeness is measured as practices per population; however, this measure is not evidence based and does not account for the type of practice, number of consultations or number of GPs within the practice. Investigation into the most appropriate method to measure sentinel site representativeness of community population is needed.

Box 2. Summary of recommendations

Representativeness and recruitment of sentinel practices

1. Expand the network to improve representativeness
2. Link ASPREN with existing sentinel GP networks
3. Maintain or increase the professional incentive

Case definition sensitivity, specificity and positive predictive value

4. Consider inclusion of laboratory support to improve ability to analyse specificity
5. Use the national ILI case definition (fever + cough + fatigue)

Data timeliness and utility for decision-making

6. Explore risks and benefits of automated data extraction and electronic reporting
7. Develop a structure for analysis and presentation of surveillance data
8. Enhance dissemination of feedback and summary data analysis

System coordination and resources

9. Consider alternative models for coordination of biosecurity surveillance in general practice
10. Provide minimum annual funding commitment for a minimum defined period of time
11. Explore risks and opportunities for income generation
12. Consider the privacy legislation and ethical implications of current and proposed surveillance systems

Maintaining a stable body of participating GPs is important for system stability and continuity. The provision of professional incentives was deemed important by participating GPs. Exploration of educational opportunities through meetings or research projects may attract additional points. Alternatively direct payment to GPs for participation in surveillance could be considered. However, this evaluation highlighted that compliance with the current manual data collection system was not optimal, nor was it consistent by state. Compliance to explicit reporting requirements, for example zero reporting, should be a condition of participation and award of professional incentives or payment.

Conformity to the nationally agreed ILI case definition permits comparison across ASPREN and state-level ILI surveillance systems. This conformity may also permit amalgamation of the data from state-level ILI surveillance systems to provide national ILI surveillance complimentary to ASPREN. For example, ASPREN could specifically recruit GPs from states and territories with no influenza surveillance systems or where coverage is limited, and combine these data with that from GPs participating in state-based systems such as Victoria and New South Wales. Ultimately, the question will arise whether ILI surveillance should be conducted centrally, removing the need for state-based systems, or whether a collaboration of national and state systems can function efficiently.

Case definition sensitivity, specificity and positive predictive value

The evidence supports universal adoption of the nationally agreed ILI case definition of fever, cough and fatigue.[9] However, inclusion of laboratory support to confirm influenza diagnosis or comparison of ILI surveillance data with confirmed influenza data sources is necessary to assure appropriate interpretation of sentinel ILI surveillance data. Evidence based reviews are required to investigate the case definition applicability when influenza strains other than H3N2 predominate and utility of the case definition when applied to children.

Data timeliness and utility for decision-making

This evaluation identified poor timeliness of data collation and reporting as an issue. Electronic data collection methods, with their advantage of timeliness and automation are an obvious alternative to current paper-based methods. However, several limitations such as: the ability to identify incident cases from follow-up visits; application of a standard case definition; the cost of establishing the system, including capital costs; and compliance to federal and state privacy legislation for accessing health data, need to be overcome.

Automated data extraction from a database such as Medical Director only permits access to a summary diagnosis field stating derivations of 'influenza' based upon the opinion of the treating physician and not necessarily conforming to a prescribed case definition. However, evaluation of electronic syndromic GP surveillance system in New Zealand concluded that ILI data extracted corresponded well with their manual paper-based GP ILI surveillance,[33] as did evaluation of a medical locum service ILI surveillance used in Victoria.[34]

One systematic review and critical evaluation of published literature about surveillance systems for illnesses and syndromes related to bioterrorism identified 13 systems that collected influenza-related data; five of these have been described in peer-reviewed evaluation reports. These reports did not provide sufficient evidence to favour any given source of ILI data (school absenteeism, sick-leave prescriptions, GP consultations for ILI) or method of collection or analysis. There was an indication that electronic reporting methods were more timely than manual systems.[35]

By focusing on symptoms, rather than confirmed diagnoses, syndromic surveillance aims to detect bioterrorism events or newly emerging diseases earlier than would be possible from traditional surveillance systems. The clinical presentation of ILI can be loosely considered as a bioterrorism-related syndrome: anthrax and respiratory agents may present with fever, cough and fatigue with rapid onset.[35] However, there is limited evidence, based on evaluation of surveillance systems specifically designed for collecting and analysing data for the early detection of bioterrorism events, that they will be effective in detecting such events.[35,36] American studies demonstrated that only 5 per cent of outbreaks, and none of five recent examples of emerging infectious diseases, were detected via surveillance.[37,38] WHO estimates 65 per cent of the world's first news about infectious disease events come from informal sources such as press reports and the Internet.[39]

There is presently a high risk for emergence of a new influenza strain to cause a pandemic. ILI surveillance in Victoria has demonstrated good capacity for monitoring endemic influenza seasonal activity;[40] however, ability to detect a new strain in a timely fashion is untested. ASPREN ILI surveillance does not include, nor is it linked to, provision of laboratory support to confirm influenza diagnoses. In addition to confirming the proportion of ILI that is attributable to influenza, laboratory support provides the opportunity to test influenza negative samples for other respiratory viruses or emerging diseases.[41,42] The more samples that are tested the higher likelihood there may be of detecting a new respiratory virus or influenza virus drift. Provision of an established link

between surveillance and laboratories will facilitate collaboration and coordination in an outbreak or emerging infectious disease situation.

There are no formal structures within ASPREN, such as thresholds or specified periods at which to review data,[40,43] to facilitate the ability of ASPREN to inform and impact on decision-making and initiate public health action. The poor timeliness of data acquisition and reporting equally impacts on data utility for decision-making, with the exception of retrospective comparisons to validate trends observed from different influenza surveillance data sources.

However, the stability of the network provides a potential platform for the rapid gathering of national community-level data for a known or hypothesised syndrome. Flexibility can be easily enhanced to permit rapid dissemination of additional condition case definitions in the instance of a bioterrorism or pandemic event. These capacities should be recognised and developed immediately and prior to any comprehensive redevelopment of ASPREN.

System coordination and resources

ASPREN, in its current format, is under-resourced and heavily reliant on the goodwill of its director and the institution in which it is housed. The resources required will be dependent on the level of redevelopment undertaken and whether laboratory support is included. Allocation of resources to support initiatives to maximise the quality of data collected should also be included. The Victorian Influenza Surveillance Program, including laboratory support, has an estimated annual operating cost of $125,000 for approximately 40 sentinel practices and testing 500 specimens.[19] Extrapolating from this estimate, ASPREN would require up to $300,000 annually to support 100 to 199 sentinel practices with state-based laboratory support. Capital costs to establish electronic data extraction, analysis and reporting systems would be additional.

Conclusions

ASPREN comprises a small group of dedicated GPs and administrators providing consistent data on select conditions. The network is not representative of Australia. Compliance to the current manual data collection system is not optimal. Resource input is minimal. Redevelopment to maximise the potential to contribute to biosecurity surveillance would require targeted intensive recruitment of GPs to achieve geographic representativeness by state and between rural and urban areas and exploration of alternative technology for data collection. The main potential of ASPREN is to permit rapid dissemination of a syndromic case definition and acquisition of nationwide community level clinical presentation data.

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Annual report: surveillance of adverse events following immunisation in Australia, 2004

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Abstract

This report summarises Australian passive surveillance data on adverse events following immunisation (AEFI) for 2004 and describes reporting trends over the five years, 2000 to 2004. AEFIs are notified to the Adverse Drug Reactions Advisory Committee by state and territory health departments, hospitals, doctors and other health providers, vaccine manufactures, and the public. There were 975 AEFI records for vaccines received in 2004. This is an annual AEFI reporting rate of 4.8 per 100,000 population, the lowest since 2000, and a 33 per cent decrease compared with 2003 (1,460 records; 7.1 AEFI records per 100,000 population). Dose-based AEFI reporting rates in 2004 were 1.8 per 100,000 doses of influenza vaccine for adults aged ≥18 years and 11.8 per 100,000 doses of scheduled vaccines for children aged <7 years. The majority of records described non-serious events while nine per cent (n=88) described AEFIs defined as 'serious'. There were no reports of death related to immunisation. The most frequently reported individual AEFI was injection site reaction in children following a fifth dose of an acellular pertussis-containing vaccine (67 reports per 100,000 doses). The marked reduction in the AEFI reporting rate in 2004 coincided with the removal of the fourth dose of acellular pertussis vaccine, due at 18 months of age, from the vaccination schedule in September 2003 and fewer people receiving meningococcal C vaccine through the national catch-up vaccination program for those aged 1–19 years in 2004, compared with 2003. The consistently low reporting rate of serious AEFIs demonstrates the high level of safety of vaccines in Australia. *Commun Dis Intell* 2005;29:248–262.

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

Introduction

Ongoing surveillance of adverse events following immunisation (AEFI), and regular analysis and reporting of these data, are integral to the management of immunisation programs. The aim of AEFI surveillance is to monitor vaccine and immunisation program safety and to detect population-specific, rare, late-onset or unexpected adverse events that may not be detected in pre-licensure vaccine trials.[1–3] An 'adverse event following immunisation' 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 can be *coincidentally* associated with the *timing* of immunisation without necessarily being caused by the vaccine or the immunisation process.

In Australia, AEFIs are notified to the Adverse Drug Reactions Advisory Committee (ADRAC) by state and territory health departments, health care professionals, vaccine manufacturers and members of the public.[4,5] All reports received by ADRAC are evaluated using internationally consistent criteria[6] and are reviewed at regular meetings. These passive AEFI surveillance data have been collated in the ADRAC database since 2000 and are used to monitor trends, detect signals and generate hypotheses. Reports summarising national AEFI surveillance data have been published regularly since 2003.[7–9] This report summarises AEFI data reported to ADRAC for vaccines received during 2004 and trends in AEFI reporting for the five year period 2000–2004.

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Several important changes both to AEFI surveillance methods and to the Australian childhood vaccination schedule occurred during 2003 and 2004 that affect the AEFI surveillance data presented in this report. In September 2003, the 8th edition of the *Australian Immunisation Handbook*[5] was released. The case definitions of several AEFIs, including those for anaphylactic reaction and allergic reaction, differed from those shown in the 7th edition of the *Australian Immunisation Handbook*.[4] Coinciding with the release of the 8th edition of the Handbook were a number of changes to the immunisation schedule, including the removal of the 4th dose of DTPa (due at 18 months of age) and new recommendations that all children receive three vaccines not included in the funded National Immunisation Program at the time (i.e. 7-valent pneumococcal conjugate vaccine (7vPCV), varicella vaccine and inactivated poliomyelitis vaccine).[5] Also the meningococcal C conjugate vaccine catch-up immunisation program for those aged 1–19 years, which commenced in 2003, was completed for most school-aged and pre-school children during 2004.[10]

Methods

Adverse events following immunisation data

De-identified information was released to the National Centre for Immunisation Research and Surveillance for all drug and vaccine adverse event notifications received by ADRAC between 1 January 2000 and 31 March 2005. Readers are referred to previous AEFI surveillance reports for a description of the AEFI surveillance system and methods used to evaluate AEFI reports received by ADRAC.[7,8]

ADRAC database records* were eligible for inclusion in the analysis 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 2004 *or* (b) if no vaccination date was recorded, the date of onset of symptoms or signs occurred between 1 January 2000 and 31 December 2004.

* The term 'AEFI record' is used throughout this report because a single AEFI notification to ADRAC can generate more than one record in the database. For example if a notification describes an injection site reaction plus symptoms and signs of a systemic adverse event (e.g. fever or generalised allergic reaction), two records will appear in the database: one record containing information relevant to the injection site reaction and one record for the systemic adverse event.

Study definitions of adverse events following immunisation 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[6] and the US Vaccine Adverse Events Reporting System (VAERS).[11] In this report, an AEFI is defined as 'serious' if the record indicated the person had recovered with sequelae, been admitted to hospital, experienced a life-threatening event, or died.

Typically, each AEFI record listed several symptoms, signs and diagnoses that had been re-coded from the reporter's description into standardised terms using the Medical Dictionary for Regulatory Activities (MedDRA®).[12] To simplify data analysis, we grouped MedDRA® coding terms to create a set of reaction categories. Firstly, reaction categories were created that were analogous to the AEFIs listed and defined in the *Australian Immunisation Handbook* (8th edition).[5] The category created for 'allergic reaction' for this report differs from previous reports to reflect the change in the definition of 'allergic reaction (generalised)' in the 8th edition of the Handbook, where both skin and gastrointestinal symptoms and signs are included.[5] A new category was created, 'severe allergic reaction', to capture reports of a generalised allergic reaction that involved symptoms and signs of the circulatory and/or respiratory system but had not been coded in the dataset as 'anaphylactic reaction'. Reaction categories were not created for two new AEFIs listed in the 8th edition of the Handbook ('extensive limb swelling' and 'nodule') due to the lack of specific MedDRA® coding terms for these AEFIs. Instead, these AEFIs are included in the category of 'injection site reaction'. A separate reaction category was created where symptoms and signs describing a hypotonic-hyporesponsive episode (HHE) or HHE-like event were mentioned but the specific terms of HHE was not present. This definition was *based* on the Brighton Collaboration case definition for HHE (level 2 of diagnostic certainty).[13] A 'possible HHE' in this report is defined as hypotonia plus terms describing colour change (pallor, cyanosis) and/or hyporesponsiveness (somnolence, hypokinesia), in the absence of terms related to other AEFIs such as convulsion.

Additional categories were created for MedDRA® coding terms that were listed in more than one per cent of AEFI records (e.g. headache, irritability, cough). Reaction terms listed in less than one per cent of records were grouped into broader categories based on the organ system where the reaction was manifested (e.g. gastrointestinal, neurological).

Data analysis

All data analyses were performed using the SAS version 8 computer program.[14] 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 less than seven years for seven childhood vaccines funded through the National Immunisation Program (DTPa, DTPa-HepB, Hib, Hib-HepB, polio, MMR and MenCCV), and for adults aged 18 years and over for influenza vaccine. The number of administered doses of each of the seven 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.[15] Vaccine doses administered between 1 January and 31 December 2004 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 2004 annual national influenza coverage survey[16] for the 18–39 years, 40–64 years and ≥65 years age groups. Dose-based AEFI reporting rates were not determined for other vaccines and age groups due to the lack of reliable denominator data for the number of vaccine doses distributed or administered.

Dose-based AEFI reporting rates for vaccines received in 2004 were compared to 2003 reporting rates and to the average annual reporting rate for the four years 2000 to 2003 inclusive.

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 2004. 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.[7,8,17]

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 8th edition of the *Australian Immunisation Handbook*.[5] These reaction categories are similar to but 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 was administered.[7] Because children in particular receive several different vaccines at the same time, all vaccines tend to be listed as 'suspected' of involvement of a systemic adverse event, as it is usually not possible to ascribe the AEFI to a single vaccine.

Results

Summary of data

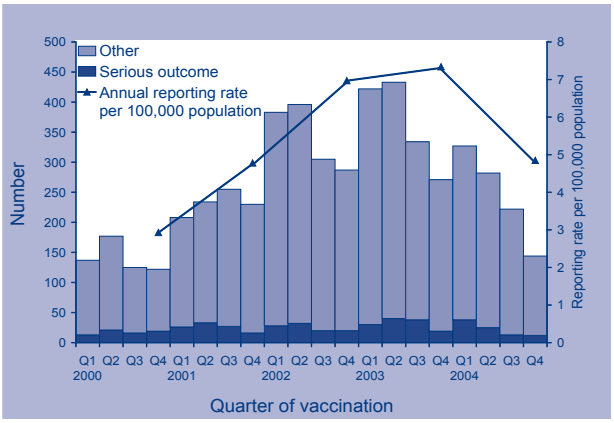
There were a total of 975 AEFI records in the ADRAC database for 2004. This is a decrease of 33 per cent compared with 2003 when there were 1,460 AEFI records. In 2004, approximately four per cent of AEFI notifications resulted in more than one AEFI record in the database (usually of an injection site reaction and a systemic reaction). This was lower than in 2003 when approximately 10 per cent of notifications resulted in more than one AEFI record.[8]

Eighty-eight (9%) of the 975 AEFI records for 2004 were defined as 'serious' (i.e. recovery with sequelae, requiring hospitalisation, experiencing a life-threatening event or death). A total of 444 (46%) AEFI records were assigned causality ratings of 'certain' (n=363, 37%) or 'probable' (n=81, 8%).

Adverse events following immunisation reporting trends

The AEFI reporting rate for 2004 was 4.8 per 100,000 population and was lower than for the previous three 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).

Figure 1. Adverse events following immunisation, ADRAC database, 2000 to 2004, by quarter of vaccination



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

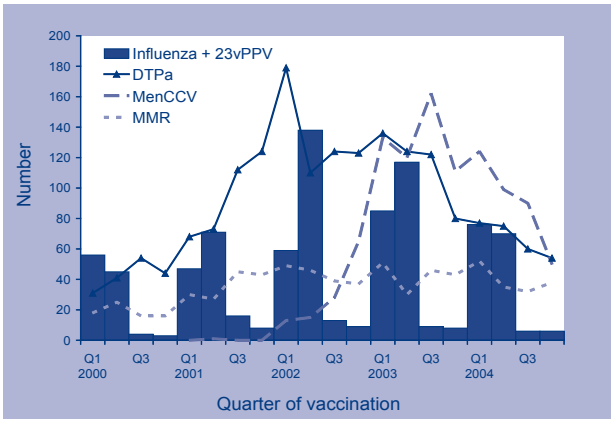
A seasonal pattern of AEFI reporting was apparent in 2004 and previous years, with the highest number of AEFI notifications for vaccinations administered in the first half of the year (Figure 1). The seasonal peak corresponds to the months when more vaccinations are administered in Australia, particularly among 5-year-old children receiving DTPa and 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).

Age and gender distribution

The AEFI reporting rate in 2004 was highest among children aged <1 year (40.9 per 100,000 population), the age group that receive the greatest number of vaccinations. The annual AEFI reporting rate decreased for all age groups in 2004 compared with 2003 (Figure 4). The largest decrease was among children aged 2 to <7 years (91.1 and 23.1 per 100,000 population in 2003 and 2004, respectively).

The overall male to female ratio was 1.0:1.2. There were more reports for females in all age groups except the 2 to <7 year age group (male:female 1.0:0.9).

Figure 2. Frequently suspected vaccines, adverse events following immunisation, ADRAC database, 2000 to 2004, by quarter of vaccination



See appendix for abbreviations of vaccine names.

Figure 3. Selected frequently reported adverse events following immunisation, ADRAC database, 2000 to 2004, by quarter of vaccination

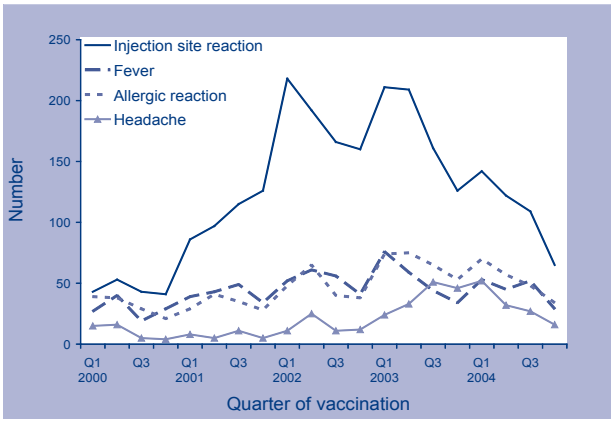
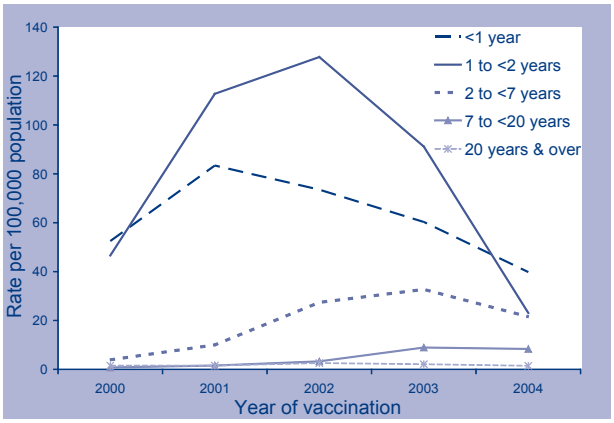


Figure 4. Reporting rates of adverse events following immunisation per 100,000 population, ADRAC database, 2000 to 2003, by age group and quarter of vaccination



Geographical distribution

As seen in previous years, AEFI reporting rates varied between the states and territories for vaccines received during 2004 (Table 1). The Australian Capital Territory and Northern Territory had the highest reporting rates (35.9 and 17.5 per 100,000 population, respectively) while Tasmania and Victoria had the lowest rates (1.2 and 2.5 per 100,000 population, respectively). Reporting rates were lower in 2004, compared with 2003,[8] for all states and territories except the Australian Capital Territory where the overall reporting rate increased from 23.0 to 35.9 per 100,000 population and the rate for children aged <7 years increased from 151 to 180 per 100,000 population. The majority of the increase in reports from the Australian Capital Territory in 2004 described adverse events following MenCCV among school-aged children.

Adverse events following immunisation outcomes

Fifty-nine per cent of reported AEFIs in 2004 were defined as 'non-serious' while nine per cent were defined as 'serious' (Table 2) – the same as seen in 2003. No deaths related to immunisation were reported in 2004. Fewer 'serious' AEFIs were assigned 'certain' or 'probable' causality ratings compared with 'non-serious' AEFIs (21% versus 50%).

Vaccines and adverse events following immunisation

Twenty-three vaccines were recorded as 'suspected' of involvement in the adverse events described in the 975 AEFI records for vaccines received in 2004 (Table 3). They included all vaccines recommended in the ASVS, plus vaccines recommended to travellers and specific risk groups.

The most frequently suspected individual vaccine was MenCCV with 363 (37%) records (Table 3). Vaccines containing pertussis, diphtheria and tetanus antigens (i.e. DTPa, DTPa-HepB and dTpa) were suspected in 342 (35%) reports. The proportion of reports where only one vaccine was suspected of involvement in the adverse event differed by vaccine, as did the proportion assigned causality ratings of 'certain' or 'probable', and the proportion defined as 'serious' (Table 3).

AEFI reporting trends differed by vaccine (Figure 2). The number of reports of AEFIs where DTPa vaccine was suspected declined in 2004 following a peak in the first quarter of 2002. Reports related to MMR vaccine remained relatively constant while reports for MenCCV increased in 2003 following the addition of this vaccine to the national immunisation program and delivery of a catch-up campaign through provider and school-based immunisation programs, then declined during 2004 as the catch-up programs were completed for most of those aged 1–19 years (Figure 5).

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

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

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

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

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

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

na Not available.

Table 2. Outcomes of adverse events following immunisation (AEFI), ADRAC database, 1 January to 31 December 2004

Outcome	AEFI records		'Certain' or 'probable' causality rating†		Age group‡			
					< 7 years		≥ 7 years	
	n	%*	n	%§	n	%§	n	%§
Non-serious	577	59	286	50	269	47	297	51
Not recovered at time of report	222	23	96	43	90	41	129	58
Not known (missing data)	88	9	41	47	46	52	38	43
Serious:	88	9	21	24	36	41	49	56
recovered with sequelae	(2)		(1)		(0)		(2)	
hospital treatment – admission	(81)		(20)		(35)		(46)	
life-threatening event	(5)		(0)		(1)		(4)	
death	(0)		(0)		(0)		(0)	
Total	975	100	444	46	437	45	513	53

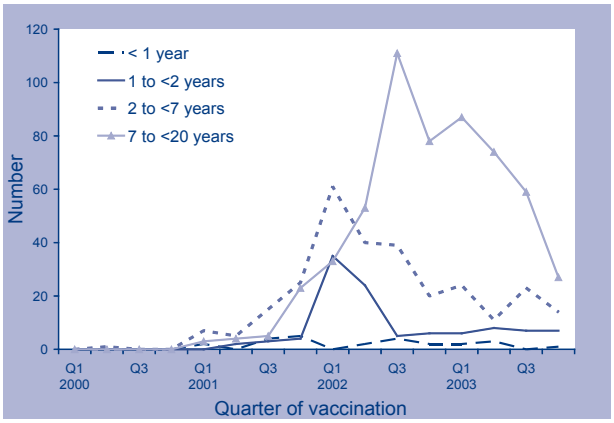
* Percentages relate to the total number of adverse events following immunisation records (n=975).

† Causality ratings were assigned to AEFI records using criteria described previously.[7]

‡ AEFI records where both age and date of birth were not recorded are not shown.

§ Percentages relate to the number of AEFI records with the specific outcome e.g. of 577 AEFI records with a 'non-serious' outcome, 50 per cent had causality ratings of 'certain' or 'probable' and 47 per cent were for children aged less than 7 years.

Figure 5. Number of adverse events following immunisation records for meningococcal C conjugate vaccine, ADRAC database, 2001 to 2004, by age group and quarter of vaccination



Adverse events following immunisation reactions

The distribution and frequency of reactions listed in AEFI records for 2004 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.

The most frequently reported adverse events were injection site reaction (45% of 975 AEFI records) followed by allergic reaction (23%), fever (18%) headache (14%) and rash (9%) (Tables 2 and 5). The relationship between the most frequently reported vaccines and adverse events is shown in Figure 6. Injection site reactions were the most commonly reported adverse event following receipt of MenCCV, DTPa, MMR, 23vPPV and influenza vaccines – over 60 per cent of reports for both DTPa (176/237) and 23vPPV (62/93) listed injection site reaction. Headache was mainly reported following receipt of MenCCV (88/363).

Figure 6. Most frequently suspected vaccines, records of adverse events following immunisation, ADRAC database, by most frequent reaction categories

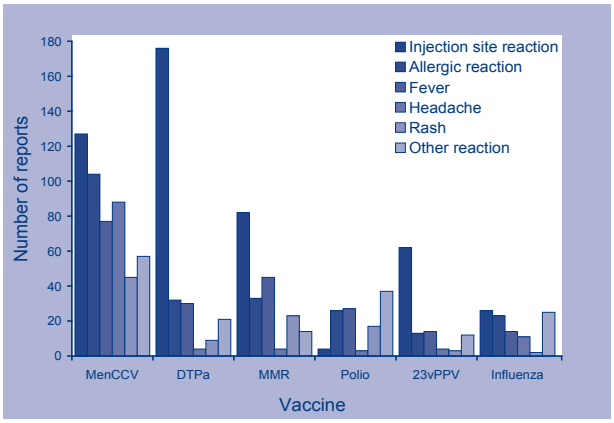


Table 3. Vaccine types listed as 'suspected' in records of adverse events following immunisation (AEFI), ADRAC database, 1 January to 31 December 2004

Suspected vaccine type*	AEFI records	One suspected vaccine or drug only†		'Certain' or 'probable' causality rating‡		'Serious' outcome§		Age group‖ < 7 years		Age group‖ ≥ 7 years	
	n	n	%¶	n	%¶	n	%¶	n	%¶	n	%¶
MenCCV	363	280	77	168	46	33	9	106	29	250	69
DTPa	237	114	48	103	43	6	3	221	93	0	-
MMR	157	23	15	15	10	11	7	146	93	7	4
Polio	93	2	2	1	1	13	14	88	95	2	2
23vPPV	93	71	76	59	63	4	4	1	1	88	95
Influenza	83	63	76	27	33	15	18	1	1	80	96
Hib	77	2	3	2	3	11	14	75	97	1	1
DTPa-hepatitis B	66	12	18	9	14	12	18	65	98	0	-
Hepatitis B	52	28	54	13	25	8	15	8	15	43	83
dTpa	39	31	79	20	51	1	3	0	-	39	100
7vPCV	29	17	59	6	21	6	21	28	97	0	-
Hib-hepatitis B	24	3	13	3	13	2	8	19	79	0	-
Varicella	23	19	83	1	4	4	17	13	57	9	39
dT	15	13	87	9	60	1	7	1	7	14	93
Rabies	13	9	69	3	23	0	-	0	-	13	100
JE	9	6	67	4	44	1	11	1	11	8	89
Hepatitis A	7	1	14	0	-	4	57	2	29	4	57
Q fever	7	7	100	2	29	4	57	0	-	6	86
Hepatitis A + B	5	3	60	0	-	0	-	0	-	4	80
BCG	2	2	100	1	50	0	-	2	100	0	-
Typhoid	2	0	-	0	-	1	50	0	-	1	50
Yellow fever	2	2	100	0	-	0	-	0	-	2	100
Tetanus	1	1	100	1	100	0	-	0	-	1	100
Total**	975	709	73	444	46	88	9	437	45	513	53

* See appendix for abbreviations of vaccine names.

† Adverse events following immunisation 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.[7]

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

‖ AEFI records not shown if both age and date of birth were not reported.

¶ 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 363 AEFI records; this was the only suspected vaccine in 77 per cent of the 363 AEFI records, 46 per cent had 'certain' or 'probable' causality ratings, 9 per cent were defined as 'serious' and 29 per cent were for children aged less than 7 years.

** Total number of AEFI records analysed, not the total in each column as categories are not mutually exclusive and one AEFI record may list more than one vaccine.

Table 4. Reaction categories of interest* mentioned in records of adverse events following immunisation (AEFI), ADRAC database, 1 January to 31 December 2004

Reaction category*	AEFI records	Only reaction reported†		Certain/probable causality rating‡		Age group§			
						< 7 years		≥ 7 years	
	n	n	%‖	n	%‖	n	%‖	n	%‖
Injection site reaction	438	272	23	324	74	233	53	196	45
Allergic reaction¶	197	23	12	58	29	69	35	123	62
Severe allergic reaction¶	12	0	0	2	17	4	33	8	67
Fever	179	6	3	54	30	88	49	83	46
Rash	92	35	38	24	26	52	57	34	37
Abnormal crying	28	4	14	3	11	26	93	2	7
Convulsions	27	8	30	17	63	8	30	19	70
Arthralgia	26	3	12	13	50	1	4	24	92
Lymphadenopathy/itis**	21	4	19	9	43	7	33	12	57
HHE††	7	0	–	7	100	7	100	0	–
Possible HHE††	10	0	–	0	–	10	100	0	–
Abscess	6	0	–	1	17	6	100	0	–
Arthritis	4	0	–	0	–	1	25	3	75
Anaphylactic reaction	2	0	–	0	–	0	–	1	50
Brachial neuritis	2	0	–	1	50	0	–	2	100
Guillain-Barré syndrome	2	0	–	1	50	1	50	1	50
Encephalitis	1	0	–	0	–	1	100	0	–
Parotitis	1	0	–	0	–	1	100	0	–
Sepsis	1	0	–	0	–	0	–	1	100
Thrombocytopenia	1	0	–	1	100	0	–	1	100
Acute flaccid paralysis	0	–		–		–		–	
Death	0	–		–		–		–	
Encephalopathy	0	–		–		–		–	
Meningitis	0	–		–		–		–	
Orchitis	0	–		–		–		–	
Osteitis	0	–		–		–		–	
Osteomyelitis	0	–		–		–		–	
SSPE‡‡	0	–		–		–		–	
Toxic shock syndrome	0	–		–		–		–	
Total§§	975	415	43	444	46	437	45	513	53

* Reaction categories were created for the adverse events following immunisation of interest listed and defined in the *Australian Immunisation Handbook*, (8th edition, p 22–3 and 271–5)[5] as described in Methods section.

† Adverse events following immunisation records where only one reaction was reported.

‡ Causality ratings were assigned to AEFI records using criteria described previously.[7]

§ Not shown if both age and date of birth were not recorded.

‖ Percentages relate to the number of AEFI records in which the specific reaction term was listed e.g. of 438 AEFI records listing injection site reaction, 62 per cent listed only one type of reaction while 74 per cent had causality ratings of 'certain' or 'probable' and 53 per cent were for children aged less than 7 years.

¶ Allergic reaction includes skin and/or gastrointestinal (e.g. diarrhoea, vomiting) symptoms and signs.[5] The category 'severe allergic reaction' includes allergic reaction with symptoms and signs indicating involvement of the circulatory and/or respiratory system but not recorded in the ADRAC database as 'anaphylactic reaction'. [5]

** Includes lymphadenitis following Bacille Calmette-Guèrin vaccination and the more general term of 'lymphadenopathy'.

†† Hypotonic-hyporesponsive episode (HHE). The separate reaction term of 'possible HHE' indicates records where 'HHE' was not listed but other terms describing an HHE or similar event were (e.g. hypotonia plus pallor or cyanosis or somnolence or hypokinesia not coded as convulsion).[13]

‡‡ Subacute sclerosing panencephalitis.

§§ Total number of AEFI records analysed, not the total in each column as categories are not mutually exclusive and one AEFI record may list more than one reaction term.

Table 5. 'Other'* reaction terms listed in records of adverse events following immunisation (AEFI), ADRAC database, 1 January to 31 December 2004

Reaction term*	AEFI records	Only reaction reported†		Certain/probable causality rating‡		Age group§ < 7 years		Age group§ ≥ 7 years	
	n	n	%‖	n	%‖	n	%‖	n	%‖
Headache	127	10	8	53	42	7	6	116	91
Malaise	86	0	–	25	29	31	36	53	62
Oedema	62	9	15	29	47	28	45	32	52
Pain	52	2	4	16	31	7	13	43	83
Nausea	48	0	–	22	46	2	4	46	96
Myalgia	42	0	–	17	40	2	5	39	93
Syncope	33	3	10	16	52	3	10	28	90
Irritability	30	1	3	6	20	22	73	6	20
Dizziness	29	0	–	14	48	0	–	29	100
Anorexia	23	0	–	7	30	11	48	12	52
Reduced sensation	22	0	–	12	55	0	–	22	100
Pallor	21	1	5	8	38	7	33	13	62
Erythema	20	2	10	5	25	10	50	9	45
Somnolence	15	1	7	6	40	7	50	7	25
Increased sweating	15	0	–	6	40	4	27	11	73
Cough	14	0	–	4	29	5	36	8	57
Pharyngitis	11	0	–	3	27	3	27	8	73
Fatigue	11	0	–	5	45	0	–	10	91
Weakness	11	0	–	1	9	2	18	8	73
Heart rate/rhythm change	10	1	10	2	20	5	50	5	50
Influenza-like illness	10	0	–	3	30	1	10	9	90
Other									
general non-specific	43	1	2	15	35	11	26	29	67
respiratory	34	4	12	8	24	9	26	24	71
eye or ear	33	1	3	12	36	8	24	25	76
cardiovascular	28	1	4	15	54	7	25	21	75
neurological	27	4	15	8	30	8	30	8	30
psychological	26	0	–	6	23	13	50	13	50
skin	19	4	21	6	32	7	37	12	63
gastrointestinal	12	3	25	1	8	5	42	7	58
infection	11	3	27	0	–	3	27	6	55
musculoskeletal	11	2	18	5	45	1	9	1	9
metabolic/endocrine	6	0	–	0	–	4	67	1	17
renal/urogenital	4	0	–	4	100	0	–	4	100
haematological	3	0	–	1	33	0	–	3	100
miscellaneous	1	0	–	1	100	0	–	1	100
pregnancy/congenital	1	0	–	0	–	0	–	1	100

* Reaction terms not listed in the *Australian Immunisation Handbook*[5] but included in adverse events following immunisation records in the ADRAC database. The top part of the table shows reaction terms included in one per cent or more of AEFI records; the bottom part of the table shows reaction terms grouped by organ system that were included in less than one per cent of AEFI records.

† AEFI records where only one reaction was reported.

‡ Causality ratings were assigned to AEFI records using criteria described previously.[7]

§ Not shown if both age and date of birth were not recorded.

‖ Percentages relate to the number of AEFI records in which the specific reaction term was listed e.g. of 438 AEFI records listing injection site reaction, 62 per cent listed only one type of reaction while 74 per cent had causality ratings of 'certain' or 'probable' and 53 per cent were for children aged less than seven years.

More severe AEFIs reported included anaphylactic reaction (n=2), severe allergic reaction involving the respiratory and/or circulatory system (n=12), hypotonic-hyporesponsive episode (HHE, n=7), possible HHE (n=10), thrombocytopenia (n=1), encephalitis (n=1) and convulsion (n=27). The two reports of anaphylactic reaction were for adults: one following 23vPPV and one following receipt of typhoid and hepatitis A vaccines. DTPa-HepB vaccine was the most commonly suspected vaccine in AEFI records of HHE (5/7, 71%) and possible HHE (7/10, 70%). Of the 27 reports of convulsion, 17 (63%) listed MenCCV and 3 (11%) listed dTpa as a suspected vaccine: 19/27 (70%) vaccinees were aged 7 to <20 years and had received one or more of MenCCV, dTpa and hepatitis B vaccines.

Reactions mentioned in fewer than one per cent of AEFI records for 2004 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', which included reaction terms such as 'feeling hot', 'feeling cold' and 'discomfort'.

The trends in the most frequently reported types of reactions changed over time (Figure 3). There were fewer reports of injection site reaction in 2004 compared with previous years. Reports of allergic reaction, fever and rash were less variable over time and reports of headache were lower in 2004 compared with 2003, consistent with the decrease in reporting of adverse events following MenCCV.

Dose-based adverse events following immunisation reporting rates

Influenza vaccine and adults aged ≥18 years

Influenza vaccine was suspected of involvement in 78 AEFI records for people aged ≥18 years. The dose-based AEFI reporting rates by age group are shown in Table 6. The AEFI reporting rate was lower among influenza vaccinees aged ≥65 years than for younger vaccinees (Table 6), as seen previously.[7,8] The most frequently reported adverse events were injection site reaction, fever and allergic reaction (0.6, 0.3 and 0.3 per 100,000 doses, respectively). The reporting rate of injection site reactions was highest among younger vaccinees aged 18–39 years (1.2 per 100,000 doses) than for the 40–64 year and ≥65 year age groups (0.4 and 0.3 per 100,000 doses, respectively). There was one report of Guillain-Barré syndrome following influenza vaccination in a person aged 65 years or more. This corresponds to a reporting rate of 0.05 per 100,000 doses for persons aged 65 years or more and 0.03 per 100,000 doses for persons aged 40 years or more, the same as in previous years.[7,8]

Table 6. Dose-based reporting rates of adverse events following immunisation (AEFI) with influenza vaccine,* 18 years and over, ADRAC database

AEFI category†	Age group	AEFI records‡ n	Vaccine doses n	Rate per 100,000 doses§		
				2004	2003	Ratio of 2004 to 4-year mean‖
Overall	≥ 18 years	78	4,447,500	1.8	– ¶	–
	18 to 39 years	20	732,700	2.7	–	–
	40 to 64 years	36	1,653,300	2.2	2.8	0.7
	≥ 65 years	22	2,061,500	1.1	1.6	0.7
Serious	≥ 18 years	12	4,447,500	0.3	–	–
	18 to 39 years	0	732,700	0.0	–	–
	40 to 64 years	7	1,653,300	0.4	0.2	2.5
	≥ 65 years	5	2,061,500	0.2	0.3	1.2

* Number of administered doses of influenza vaccine estimated from the 2004 national influenza survey.[16]

† Adverse events following immunisation (AEFI) category includes all records, and those defined as 'serious' where influenza vaccine was suspected of involvement in the reported adverse event. 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 in 2004.

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

‖ Ratio of the reporting rate per 100,000 doses for 2004 and the average (mean) reporting rate per 100,000 doses for the previous 4 years (2000–2003).

¶ Influenza immunisation rates for the 18–39 year age group were not estimated before 2004, therefore AEFI reporting rates for this age group have not been estimated for 2000–2003.

Scheduled vaccines for children aged <7 years

Dose-based AEFI reporting rates are shown in Table 7 for seven funded vaccines received during 2004 by children aged less than seven years. The overall reporting rate and those for all vaccines and categories decreased in 2004 compared with 2003. The highest AEFI reporting rate and largest change in reporting rate was for DTPa vaccine (Table 7). The reporting rate for MMR vaccine was slightly higher than the average annual rate for the previous four years (2000–2003) (Table 7).

Dose-based reporting rates of the most commonly reported AEFIs differed by vaccine type (Figure 7). Injection site reaction following DTPa vaccine was reported at a rate of 32.9 per 100,000 doses of DTPa vaccine for children aged <7 years, down from 47.9 per 100,000 doses for 2003.[8] The decrease was greatest among children aged 1 to <2 years following the removal from the immunisation schedule of the 4th dose of the vaccine, due at 18 months of age, in September 2003 (Figure 8). In 2004, only one report of a local reaction following DTPa was received for a child in this age group. The reporting rates of injection site reaction for the <1 year and 2 to <7 year age groups were 1.1 and 66.7 per 100,000 doses of DTPa vaccine, respectively.

The reporting rate of injection site reaction following MMR vaccine was highest among children aged 2 to <7 years (30 per 100,000 doses). Over 85 per cent (65/75) of these children had received DTPa and/or other vaccines at the same time as MMR but the specific injection sites of each of the vaccines were not reported or could not be differentiated.

Figure 7. Reporting rates of adverse events following immunisation per 100,000 doses of vaccine recorded on the Australian Childhood Immunisation Register, children aged <7 years, ADRAC database, 2004

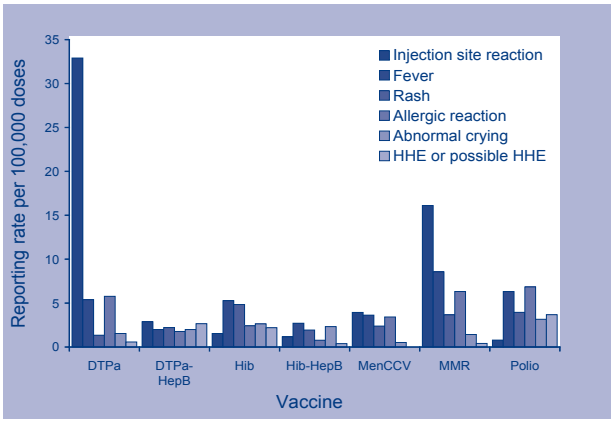


Table 7. Reporting rates of adverse events following immunisation (AEFI) per 100,000 vaccine doses,* children aged less than 7 years, ADRAC database

Suspected vaccine type† or AEFI category‡	AEFI records n	Vaccine doses n	Rate per 100,000 doses§		
			2004	2003	Ratio of 2004 to 4–year mean‖
DTPa	221	519,708	42.5	63.3	0.9
DTPa-HepB	65	451,793	14.4	18.5	0.6
Hib	75	454,667	16.5	21.0	0.7
Hib-HebB	19	258,483	7.4	10.7	0.5
Polio	88	964,591	9.1	14.6	0.7
MMR	146	490,500	29.8	32.0	1.2
MenCCV	106	380,023	27.9	33.3	na
Total‡	400	3,519,765	11.4	18.5	0.7
'Serious' outcome‡	30	3,519,765	0.9	1.1	0.7
'Certain' or 'probable' causality rating‡	160	3,519,765	4.5	9.2	0.6

* Number of vaccine doses recorded on the Australian Childhood Immunisation Register and administered between 1 January and 31 December 2004.

† Adverse events following immunisation (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.

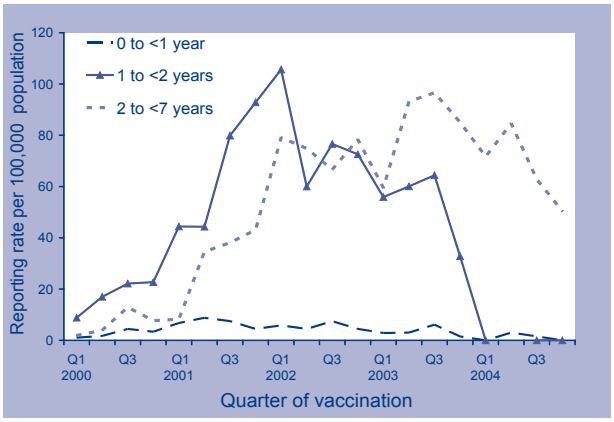
‡ AEFI category includes all records (i.e. total), those assigned 'certain' or 'probable' causality ratings, and those defined as 'serious' where at least one of the seven vaccines shown in the table was suspected of involvement in the reported adverse event. Causality ratings were assigned using the 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.

‖ Ratio of the reporting rate per 100,000 doses for 2004 and the average (mean) reporting rate per 100,000 doses for the previous 4 years (2000–2003).

na Not estimated as the program commenced on 1 January 2003.

Figure 8. Reporting rate of injection site reaction per 100,000 doses of DTPa vaccine, by age group and quarter of vaccination, ADRAC database, 2000 to 2004



The reporting rate for HHE following DTPa-HepB vaccine was 1.13 per 100,000 doses for children aged <1 year. This is the same as for the previous three-year period (2001–2003) of 1.12 per 100,000 doses of DTPa-HepB vaccine and 1.23 per 100,000 doses of DTPa vaccine. Only one report of HHE following DTPa vaccine was received in 2004.

Discussion

The data show that there was a significant decrease in reports of AEFIs to ADRAC in 2004 compared with 2002 and 2003. This was evident across all age groups and all states and territories except the Australian Capital Territory, and was most apparent in reports of adverse events following DTPa among children aged 1 to <2 years and MenCCV among people aged 2 to <20 years. This decrease coincides with the removal of the 4th dose of DTPa vaccine (due at 18 months of age) from the ASVS in September 2003 and the completion in most states and territories of the catch-up MenCCV immunisation program for older children and adolescents during 2004 (Figures 2 and 5).

The data demonstrate a high level of safety of vaccines in Australia, with the majority of AEFIs reported being injection site reaction and other non-serious events. The reporting rate of AEFIs defined as serious remains low (0.9 per 100,000 doses of vaccines for children aged <7 years and 0.3 per 100,000 for influenza vaccinees aged ≥18 years), while the proportion of all reported AEFIs that are defined as serious has remained at 9–10 per cent over the past five years.

Although the number of AEFI reports for MenCCV and DTPa were substantially lower in 2004 than 2003 (363 and 237 in 2004 vs 536 and 416 in 2003), they remained the most frequently suspected vaccines with injection site reactions still the most often commonly reported AEFI for both vaccines. The types of adverse events reported for both vaccines is consistent with those detected in other AEFI surveillance systems and in observational studies.[18–20] Any impact that removal of the DTPa dose due at 18 months of age from the ASVS might have on rates of injection site reaction among children receiving DTPa at 4–5 years of age will not be seen in the national AEFI surveillance data until 2006 when the first cohort of children affected by the schedule change become due for their 4th dose prior to school entry. It is expected that the total number of AEFI reports for MenCCV will decline in 2005 when the catch-up program is completed. Reporting has stabilised for the age group who receive MenCCV as part of the routine immunisation schedule at 12 months of age with six to eight reports per quarter for the 1 to <2 year age group during 2004 (Figure 5).

There has been a decrease in AEFI reporting for children aged <1 year each year for 2002–2004, following a peak in 2001 (Figure 4). The reason for this decease is unclear, as it has occurred in all states and territories and for all vaccines scheduled for this age group (data not shown). Immunisation coverage rates have not changed substantially for this age group over that time.[15] The decrease since 2001 could relate to increased reporting during 2001 following a major change to the ASVS in May 2000 with the introduction of the universal hepatitis B vaccination program (given as a monovalent vaccine at birth and combined with either DTPa or Hib at two, four and six months of age). An increase in reporting of AEFIs often occurs after the introduction of a new vaccine, followed by a decrease in reporting as providers become more familiar with the vaccine.[2,17,21]

The recent addition of 7vPCV to the National Immunisation Program in January 2005 for all children[22] and further changes to be implemented in November 2005 are likely to be reflected in AEFI reporting patterns. These changes include a national childhood varicella vaccination program[23] and replacement of the current monovalent and combination vaccines used in the infant schedule (i.e. DTPa, DTPa-HepB, Hib, Hib-HepB and polio) with a single hexavalent vaccine at two, four and six months of age.[24]

The implementation of the nationally funded 7vPCV program for all children from January 2005 has seen an increase in AEFI reports mentioning this vaccine compared with previous years (45 reports were received in the first three months of 2005 compared with a total of 72 reports for 2000–2004; data not shown). The frequency and type of adverse events following 7vPCV reported to ADRAC since 2000 are similar to those reported to the US VAERS with injection site reaction, fever, allergic reaction and irritability or fussiness the most frequently reported adverse events.[25] Data for adverse events following 7vPCV reported to ADRAC will be included in future surveillance reports.

Differences in AEFI surveillance practices among key stakeholders also affect the interpretation of AEFI trend data. In November 2002 there were major changes with the implementation of a new database and the MedDRA® dictionary used to code reaction terms. In September 2003, the definitions of some AEFIs listed in the *Australian Immunisation Handbook*, which many reporters use as a guide to notify an AEFI, were changed to reflect current opinion. These mainly affected coding and reporting of anaphylactic reaction and allergic reaction. Our assessment of the data suggests that these changes have had little impact on AEFI trends.

AEFI surveillance practices differ markedly between the states and territories and are reflected in their reporting rates (Table 1). Victoria and Tasmania consistently have the lowest AEFI reporting rates – both states request general practitioners and others to report directly to ADRAC. In contrast, the Australian Capital Territory and South Australia have significantly higher reporting rates, particularly for children aged <7 years, and have similar systems where reporting of AEFIs to the state or territory health department is requested and reporting by parents is encouraged.

AEFI surveillance is complex compared with most public health surveillance systems as there are multiple exposures and multiple outcomes of interest.[2,3,17] Further, the association between the reported exposure(s) and outcome(s) is temporal but not always causal. The quality of the information contained in AEFI notifications to ADRAC is very important as inadequate or misleading information can impact on the interpretation of AEFI surveillance data. For example, based on data from clinical trials and observational research, the apparently high reporting rate of injection site reaction following MMR vaccine in children aged 2 to <7 years could, at least partly, be attributed to co-administration of a 5th dose of DTPa vaccine and insufficient information reported to ADRAC about the sites of injection of the co-administered vaccines.

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 consistently show low rates of vaccine preventable diseases in Australia and the substantial impact of national immunisation programs in reducing the incidence, morbidity and mortality of diseases such as Hib, invasive pneumococcal disease, meningococcal C disease and measles.[26–30] AEFI surveillance data over a five year period also show consistently low reporting rates of serious AEFIs and that the most frequently reported AEFIs in Australia are injection site reaction, allergic reaction, fever and other non-serious and transient events.

This is the fourth regular report analysing AEFIs in Australia detected by the national passive surveillance system.[7–9] 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. The data reported here demonstrate that the system is sufficiently sensitive to detect both known rarer adverse events, including HHE and thrombocytopenia, and expected changes in AEFI reporting trends, such as those related to changes in immunisation programs for DTPa and MenCCV. It is expected that the system will provide valuable information to assist in the management of the hexavalent and varicella immunisation programs that are to commence in November 2005.

Acknowledgments

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Appendix

Abbreviations of vaccine types

BCG	Bacille Calmette-Guèrin
dT	diphtheria-tetanus
DTPa	diphtheria-tetanus-pertussis (acellular) – paediatric formulation
dTpa	diphtheria-tetanus-pertussis (acellular) – adolescent and adult formulation
DTPa-HepB	combined diphtheria-tetanus-pertussis (acellular) and hepatitis B
HepB	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 vaccine
MenCCV	meningococcal C conjugate vaccine
MMR	measles-mumps-rubella
7vPCV	7-valent pneumococcal conjugate vaccine
23vPPV	23-valent pneumococcal polysaccharide vaccine
polio	poliomyelitis (oral and inactivated)

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Annual report of the Australian National Poliovirus Reference Laboratory, 2004

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Abstract

The Australian National Poliovirus Reference Laboratory at the Victorian Infectious Diseases Reference Laboratory is the World Health Organization designated laboratory for the isolation and testing of poliovirus from clinical specimens within Australia, the Pacific Island countries and Brunei Darussalam. Surveillance for acute flaccid paralysis (AFP) within Australia, the main clinical manifestation of poliomyelitis, is also coordinated at the Victorian Infectious Diseases Reference Laboratory in conjunction with the Australian Paediatric Surveillance Unit. The annual non-polio acute flaccid paralysis rate after classification of cases by the Polio Expert Committee was 1.0 per 100,000 population, reaching the expected World Health Organization annual target for a non-polio endemic country. During 2004, 64 specimens from 30 AFP cases were referred to the National Polio Reference Laboratory. A mixture of poliovirus types 1 and 2 was isolated from an infant with AFP from New South Wales. Both isolates tested as Sabin-like and the case was subsequently classified as infant botulism by the Polio Expert Committee. The laboratory isolated adenoviruses from seven AFP cases. A coxsackievirus B5 and an echovirus 18 were identified from a further two AFP cases. During 2004, 1,266 cases of poliomyelitis due to wild poliovirus were reported world-wide. Many of these resulted from wild poliovirus importations, which continued in 2005, including to Indonesia. This highlights the need for maintaining high poliovirus vaccination coverage to prevent the transmission of poliovirus and high quality AFP and laboratory surveillance for the detection of poliomyelitis due to an imported wild poliovirus. *Commun Dis Intell* 2005;29:263–268.

Keywords: poliovirus, acute flaccid paralysis, surveillance, enterovirus

Introduction

The National Poliovirus Reference Laboratory (NPRL) located at the Victorian Infectious Diseases Reference Laboratory (VIDRL) is responsible for the testing of specimens from patients with acute flaccid paralysis (AFP). AFP is the main clinical manifestation of poliovirus infection and occurs in approximately one per cent of infections. In addition to poliovirus, other viruses and microorganisms can cause AFP. Non-polio enteroviruses such as echoviruses 11, 18 and enterovirus 71 have been associated with AFP.[1] Other diseases presenting as AFP include transverse myelitis, Guillain-Barré syndrome and infant botulism.[2]

Surveillance for AFP is coordinated at VIDRL and conducted in collaboration with the Australian Paediatric Surveillance Unit. The World Health Organization (WHO) target for a non-polio-endemic country such as Australia is one AFP case per 100,000 children aged below 15 years. Based on this figure, we would anticipate 40 AFP cases per annum in Australia.[3]

The Australian standard immunisation schedule recommends administration of Sabin oral poliovirus vaccine (OPV) at two, four and six months of age with an additional booster dose prior to school entry. OPV contains live attenuated strains of all three poliovirus serotypes that replicate in the gut and are excreted in the faeces. Therefore, it is possible to isolate strains of poliovirus from individuals recently immunised with OPV. These may be considered as incidental isolations during routine specimen testing. Laboratories with uncharacterised polioviruses or enterovirus isolates may refer them to the NPRL for further characterisation. This will ensure that no poliovirus remains undetected and any poliovirus isolated in Australia, has been tested to differentiate between wild and vaccine strains.

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Methods

The NPRL is responsible for coordinating AFP surveillance in collaboration with the Australian Paediatric Surveillance Unit. Doctors are requested to notify by telephone, all AFP cases in children aged less than 15 years and residing in Australia, or a person of any age suspected of an acute poliomyelitis infection, to the AFP co-ordinator at VIDRL. AFP cases are also reported to the Australian Paediatric Surveillance Unit via a monthly reporting system. The clinicians, who notify a case, 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 or vaccine-associated paralytic poliovirus or (iii) non-AFP.

Due to intermittent shedding of the virus, two faecal specimens are collected 24 to 48 hours apart and up to 14 days after onset of paralysis for virological testing in a WHO accredited laboratory. The specimens are extracted in a 10 per cent v/v chloroform solution and inoculated onto a series of continuous cell lines. The main cell line employed for the isolation of poliovirus is L20B,—a mouse epithelial cell line with cell surface expression of the poliovirus receptor, CD155.[4] Another cell line used by the WHO network for the isolation of poliovirus and other enteroviruses is RD (human rhabdomyosarcoma). Other laboratories within Australia refer enteroviruses of unknown serotype to the NPRL for further characterisation. Polioviruses identified amongst these isolates are tested to differentiate between wild and vaccine strains.

All polioviruses, whether isolated from AFP cases or other sources, are tested by a WHO-accredited process known as intratypic differentiation (ITD) that distinguishes between wild and vaccine strains of poliovirus. ITD involves a genetic based method, [polymerase chain reaction (PCR)] and an antigenic based method, [enzyme–linked immunosorbent assay (ELISA)]. The ELISA utilises cross-absorbed polyclonal antisera for the specific detection of wild and OPV strains of poliovirus. The poliovirus ELISA is sensitive to mutations within the capsid of the OPV strains, resulting from nucleotide substitutions during virus genome replication. The mutations can result in vaccine strains with discordant ITD results; for example, Sabin-like by PCR and non-Sabin-like or double reactive by ELISA. A poliovirus strain that displays equal avidity for the Sabin and non-Sabin-like cross-absorbed antisera in the ELISA is described as double reactive. Sequencing of the VP1 capsid gene is performed for poliovirus isolates with discordant ITD results. Sabin polioviruses with more than one per cent nucleotide changes from the parental OPV strain within the VP1 gene, are classified as vaccine-derived polioviruses by the WHO.[5]

The NPRL is accredited annually by the WHO as a national and regional polio reference laboratory. The accreditation process includes proficiency panels for the main laboratory techniques of poliovirus isolation and identification, ELISA and PCR. In addition, an annual on-site laboratory review by WHO is conducted.

Results

Acute flaccid paralysis surveillance

According to the WHO criteria, eligible AFP cases are patients who are Australian residents and aged less than 15 years at the onset of paralysis. However, the PEC reviews cases of suspected poliomyelitis in people of any age. Sixty-two notifications of AFP in Australia were received in 2004. Forty-nine of the AFP cases notified were from patients aged less than 15 years, four cases were patients 15 years or older and nine duplicate notifications were received. Of the 49 eligible cases, the PEC classified 45 as non-polio AFP (Table 1). No clinical information has

Table 1. AFP surveillance in Australia compared with WHO indicator targets for children aged less than 15 years, 2004

WHO indicator target for AFP cases of children less than 15 years	Australia's surveillance for AFP cases with onset in 2004	Australia's AFP surveillance rates for 2004
Non-polio AFP case rate of 1 per 100,000 population (40 cases for Australia in 2004).	49 unique cases of AFP notified. 45 cases classified by the PEC as non-polio AFP.*	AFP notification rate: 1.2 per 100,000 population. Non-polio AFP case rate: 1.1 per 100,000 population.
More than 80 per cent of notified AFP cases with 2 adequate stool specimens collected at least 24 hours apart within 14 days of onset of paralysis.	18 AFP cases with 2 or more specimens per case.	Referral of adequate specimens from AFP cases: 40 per cent (18/45) of the cases classified by the PEC.

* Four cases require clinical information from the referring doctor before cases can be classified by the PEC.

AFP Acute flaccid paralysis.

been received for the remaining four cases. Thus, the annual AFP notification rate in Australia was 1.2 cases per 100,000 children aged less than 15 years. The annual non-polio AFP rate in 2004 after classification of cases by the PEC was 1.1 cases per 100,000 children aged less than 15 years, reaching the expected WHO target for a non-polio endemic country.[6] The WHO target for the non-polio AFP rate has only been met on two previous occasions, in 2000 and 2001. Twenty of the 45 (44%) cases classified as non-polio AFP by the PEC were diagnosed as Guillain- Barré syndrome.

Differences between the rates of notification of AFP by the various Australian states and territories[7] noted in previous years, were not as striking in 2004. New South Wales was responsible for 23/49 (47%) eligible notifications involving children aged less than 15 years. This is equivalent to an annual notification rate of 1.8 per 100,000 New South Wales residents aged less than 15 years. All Australian states and territories except for Western Australia, the Australian Capital Territory and Victoria reached or exceeded the target rate. Paediatricians in Victoria notified nine cases, equivalent to 0.9 cases per 100,000 children aged less than 15 years, the highest rate the state has achieved since the introduction of AFP surveillance in 1995.

Laboratory testing of specimens

Acute flaccid paralysis cases

During the reporting period, 62 specimens from 30 AFP cases within Australia were referred to the NPRL. This included six specimens collected from two AFP cases aged greater than 15 years, which is outside the WHO standard criteria for AFP surveillance.

A mixture of poliovirus types 1 and 2 was isolated from an infant with AFP from New South Wales (Table 2). While the poliovirus type 1 isolate tested as Sabin-like by PCR, it was double reactive by ELISA. Sequencing of the VP1 gene revealed 99.7 per cent nucleotide homology compared with the prototype Sabin strain and the isolate was therefore classified as Sabin-like. The poliovirus type 2 tested as Sabin-like by both methods of ITD. The Polio Expert Committee classified the case as infant botulism based on the detection of *Clostridium botulinum* serotype B toxin and isolation of *C. botulinum* serotype B organism from a faecal specimen of the infant.

A coxsackievirus B5 was isolated from two specimens of a 12-year-old child with AFP from Queensland. The virus was identified by nucleotide sequencing and confirmed by monovalent antisera neutralisation. Specimens were collected from a six-year-old child with AFP from New South Wales. The first specimen yielded a non-polio enterovirus that was subsequently sequenced and identified as echovirus 18. No virus was isolated from the second specimen of the same patient.

In 2004, adenoviruses were isolated from seven AFP cases, with confirmation by PCR (Table 2). The seven cases represented 23 per cent of the 30 Australian AFP cases that were tested by the laboratory in the reporting period. This, and the fact that five of the seven cases were from Victoria, led us to consider whether a particular serotype was circulating or had an association with AFP. Monovalent antisera were used to type the adenoviruses from four of the cases from Victoria and one from Queensland. The viruses from the Victorian cases belonged to adenovirus species C – types 1, 2 (two cases) and 5 – while adenovirus type 4 of species E was identified from the Queensland case.

Table 2. Test results of specimens and isolates referred to the Australian National Poliovirus Reference Laboratory, 2004

Result	Isolations from AFP cases	Isolations from referred samples	Total
Poliovirus Sabin-like type 1	–	1	1
Poliovirus Sabin-like type 1 & 2	1	–	1
Poliovirus Sabin-like type 2	–	3	3
Poliovirus Sabin-like type 3	–	1	1
Adenovirus*	11	1	12
NPEV	3	23	26
No virus isolated	47	2	49
Total	62	31	93

* Eleven adenoviruses were isolated from a total of 15 specimens collected from seven acute flaccid paralysis cases.

NPEV Non-polio enterovirus. Coxsackievirus B5 was isolated from two faecal specimens of one AFP case and echovirus 18 from another case. Nucleotide sequence homology results of NPEV from sources other than AFP identified coxsackievirus A16 (4 isolates), coxsackievirus B4 (2 isolates), echovirus 7 (1 isolate), coxsackievirus B5 (3 isolates), echovirus 3 (2 isolates) and echovirus 11 (5 isolates).

AFP Acute flaccid paralysis.

No virus was isolated after 14 days in culture from a total of 47 specimens, including specimens collected from the remaining 20 AFP cases.

Sources other than acute flaccid paralysis

Five polioviruses were identified from 31 specimens and isolates referred from sources other than AFP and all isolates tested as Sabin-like (Table 2). This included two polioviruses (type 2) isolated from faecal specimens collected from a three-month-old infant with continual diarrhoea, following vaccination with OPV.

Amongst the 31 referred samples from sources other than AFP cases (Table 2), two faecal specimens collected one month apart, were from an eight-month-old infant who had been vaccinated with OPV and had undergone a transplant. These specimens were referred to the NPRL to determine if shedding of vaccine poliovirus was ongoing. Poliovirus type 3 was isolated from the first specimen and tested as Sabin-like by ITD. The second specimen collected 32 days later, yielded an adenovirus that was confirmed by PCR and subsequently identified as adenovirus 1 by antisera neutralisation. No poliovirus was isolated from the second specimen.

Twenty-six of the 31 isolates and specimens were referred by a laboratory in South Australia for further identification. Of these, two polioviruses (types 1 and 2) were identified amongst the isolates and both tested as Sabin-like. This highlights the importance of referring untyped enteroviruses to the NPRL for the detection of polioviruses within Australia. To date, sequencing of 17 of the referred isolates has identified coxsackievirus A16, B4 and B5 and echovirus 3, 7 and 11. A further six non-polio enteroviruses are yet to be identified. One referred isolate failed to passage, which may have been due to loss of virus titre in transit.

A cerebrospinal fluid specimen collected from an adult with symptoms of meningitis, who had a child vaccinated with OPV six weeks prior to onset of symptoms, tested positive for enterovirus by PCR at the referring laboratory. The enterovirus PCR result was confirmed by the Viral Identification Laboratory at VIDRL. The cerebrospinal fluid specimen was tested by the NPRL and did not yield any enterovirus in cell culture.

A summary of enteroviruses tested at the NPRL between 1995 and 2004 is described in Table 3.

Regional reference laboratory activities

In its role as a WHO regional reference laboratory, the NPRL received a total of 406 specimens and isolates during January to December 2004, from national poliovirus laboratories and hospitals in the Western Pacific Region. This included 27 specimens from 14 AFP cases from the Pacific Islands, two specimens from an AFP case from Brunei Darussalam, three specimens and isolates from the Philippines and 48 specimens and isolates from Malaysia. A further 51 specimens and isolates from Papua New Guinea and 275 from Ho Chi Minh City, Viet Nam, were referred as part of an ongoing laboratory quality assurance program.

Table 3. Summary of enterovirus testing at the Australian National Poliovirus Reference Laboratory, 1995 to 2004

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
2004	6		26	61	93

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

† Two poliovirus isolates had discordant results by ITD. Sequencing confirmed the isolates as Sabin-like, with <1.0 per cent variation from the parental Sabin strain.

Laboratory accreditation

The NPRL at VIDRL retained its full accreditation status for 2004 as a national and regional reference laboratory following a performance-based review by a member of the WHO. The laboratory successfully isolated and identified all viruses in a proficiency panel, referred by the National Institute of Public Health and Environmental Protection, the Netherlands, as part of a laboratory quality assurance program. In addition, the laboratory successfully completed proficiency panels referred by the National Institute of Public Health and Environmental Protection and the Centers for Disease Control and Prevention, USA, for the WHO-approved ITD methods of ELISA and PCR, respectively.

In preparation for laboratory containment of vaccine strains of poliovirus, an inventory has been completed of all specimens and isolates stored by the laboratory since 1993. This is in addition to an inventory of wild polioviruses prepared for Australia's certification as polio-free in 2000.

Discussion

In 2004, Australia achieved the WHO standard criteria for AFP surveillance in a non-polio endemic country, by detecting one case of AFP per 100,000 children aged less than 15 years. Since the introduction of AFP surveillance in 1995, the target has been reached only twice before, in 2000 (1.15/100,000) and 2001 (1.13/100,000).[8] In the intervening years, the non-polio AFP rate dropped to 0.75 per 100,000 population less than 15 years of age in 2002[9] and to 0.68 notifications per 100,000 in 2003.[7]

The rate of adequate faecal sampling in 2004 was 40 per cent, well below the WHO target of 80 per cent of notified AFP cases in children less than 15 years of age (Table 1). Nevertheless, this is the highest rate reported since the introduction of AFP surveillance in Australia. Previously, the rate of adequate faecal sampling had varied from 24 per cent to 36 per cent.[7]

A recent publication reported the isolation of adenovirus from AFP specimens and a possible link between the virus and the condition. Ooi, *et al* described an investigation of eight children who presented with AFP during an outbreak of enterovirus 71-associated hand-foot-and-mouth disease in Malaysia in 1997.[10] The laboratory identified adenovirus 21 from two of the cases and adenovirus species B, of which adenovirus 21 is a member, from a further three AFP cases. It was concluded that adenovirus 21 may cause AFP by anterior horn cell damage or neuropathy of the brachial or lumbosacral plexus. The NPRL isolated adenovirus from 23 per cent of Australian AFP cases in 2004. No single serotype or members of adenovirus species B were identified amongst the Australian isolates. Until now, the isolation of adenovirus has been considered incidental to enterovirus isolation in relation to the WHO polio eradication program. The NPRL will continue to review all adenovirus isolations from AFP cases.

The number of wild poliovirus confirmed cases reported globally increased from 784 in 2003 to 1,266 in 2004.[11] This was mainly due to the increase in wild poliovirus transmission in Nigeria, which accounted for 62 per cent of the global 2004 case count, as well as the transmission into other African countries.[12] An outbreak in Sudan, subsequently led to virus importations into Ethiopia and Saudi Arabia.[12]

In April 2005, an outbreak of polio in Indonesia, caused by wild poliovirus 1, was reported by the WHO.[13] The index case from West Java, was unimmunised and genetic analysis of the poliovirus, indicated it was imported from Sudan and was similar to recently isolated viruses in Saudi Arabia and Yemen. A wild poliovirus has not been isolated in Indonesia since 1995. Indonesia's national routine vaccination coverage is reported to be at least three doses of OPV in 74 per cent of children below one year of age.[14] Routine immunisation rates below 90 per cent increase the risk of an outbreak in the event of a polio re-introduction.[14] However, the AFP surveillance system of Indonesia continues to meet the global minimum standard of detecting at least one AFP case per 100,000 children below 15 years of age and was sufficiently sensitive to detect a wild poliovirus importation.[14]

The risk of importations of wild poliovirus into non-endemic countries remains as long as polio exists anywhere in the world. For Australia to retain its polio-free status, it is imperative that it maintains high national vaccination coverage, currently 93 per cent[15] and conducts sensitive AFP surveillance and high quality laboratory procedures for the detection of poliovirus.

The Global Polio Eradication Initiative Strategic Plan for 2004–2008 outlines the main activities required to interrupt poliovirus transmission, achieve global certification and prepare for the global cessation of OPV.[16] In order to implement the safe cessation of OPV, all six WHO regions need to be declared free of circulating wild poliovirus.[17] The WHO Biosafety Advisory Group has recommended that the strategy used for the containment of wild polioviruses be used as a basis for containment of all polioviruses, including vaccine-derived polioviruses and Sabin strains.[17] Once polio is eradicated globally, laboratories will be the only remaining source of the virus. Successful laboratory containment will prevent the transmission of poliovirus from the laboratory into the community.

The NPRL has completed an inventory of all Sabin polioviruses stored at VIDRL in preparation for the post-global certification phase.

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Creutzfeldt-Jakob disease: Australian surveillance update to 31 December 2004

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Australian National Creutzfeldt-Jakob Disease Registry

Abstract

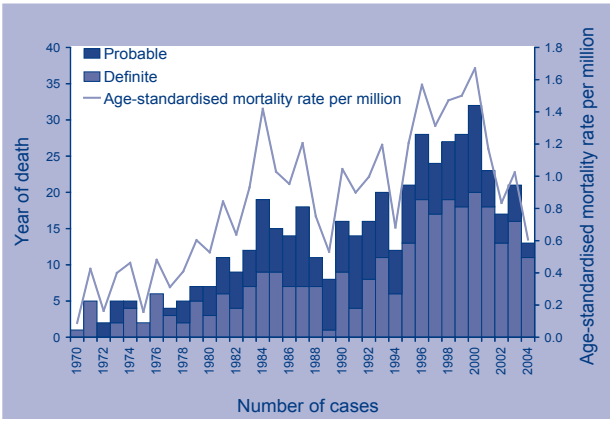
The Australian National Creutzfeldt-Jakob Disease Registry (ANCJDR) was established in October 1993 after the identification of probable iatrogenic CJD in recipients of human pituitary hormones. Since this time and with the recommendations of the Allars inquiry into CJD in Australia,[1] the registry has performed surveillance of CJD in Australia with retrospective ascertainment to 1970 and ongoing prospective ascertainment of all human prion diseases or transmissible spongiform encephalopathies (TSEs). Prion diseases include CJD, Gerstmann-Straussler-Scheinker syndrome, fatal familial insomnia and Kuru. This brief summary presents the epidemiological findings of the ANCJDR based on data from 1970 to 31 December, 2004. *Commun Dis Intell* 2005;29:269–271.

Keywords: Creutzfeldt-Jakob disease; disease surveillance; transmissible spongiform encephalopathy

From 1 October 1993 to 31 December 2004, 1,004 suspected transmissible spongiform encephalopathy (TSE) cases acquired between 1970 and 2004, have been notified to the Australian National Creutzfeldt-Jakob Disease Registry (ANCJDR) for investigation. Of these, 293 definite cases and 186 probable cases have been classified (Table 1) and comprise of 434 sporadic (91.0%), 36 familial (7.3%) and 9 iatrogenic cases (1.7%). Seven cases of possible CJD have been identified of which six were sporadic and one iatrogenic and a total of 86 cases were still under investigation with 47 of these cases still alive. After detailed follow-up and investigation, 432 suspect cases (43%) were excluded from the registry as non-TSE cases. As of December 2004, no further cases of iatrogenic CJD have been detected since the last identified case in 2000. Australia remains free of variant CJD (vCJD).

Between 1970 and 2000, a steady increase in the annual incidence of spongiform encephalopathies can be observed (Figure). 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.[2] Since 2000, a decline in numbers, in particular probable cases, has been apparent. This may relate to a number of issues, including broadened surveillance responsibilities and difficulties encountered following changes to privacy legislation. The average annual age-adjusted mortality rate during the period from 1970 to 2004 is 0.84 deaths per million per year. During the prospective period of ANCJDR surveillance from 1993 to 2004, the average annual rate of mortality was 1.19 deaths per million persons. The rate for this prospective ascertainment epoch is considered to be a more robust representation of Australian CJD incidence as during this period standardised approaches to case classification and ascertainment were implemented nationally.[3]

Figure. Australian National Creutzfeldt-Jakob Disease Registry definite and probable cases: number and age-standardised mortality rate, 1970 to 2004



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

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Table 1. Classification of cases on the Australian National Creutzfeldt-Jakob Disease Registry, 1 January 1970 to 31 December 2004

Classification	Sporadic	Familial	Iatrogenic	Variant CJD	Unclassified	Total	Cases classified during 2004*
Definite	260	28	5†	0	0	293	+19
Probable	174	8	4	0	0	186	+6
Possible	6	0	1	0	0	7	+1
Incomplete	0	0	0	0	86‡	86	+27
Total	440	36	10	0	86	572	+53

* Describes the classifications made during the 2004 surveillance year (includes cases notified in 2004 or previous years).

† Includes one definite iatrogenic case who received pituitary hormone treatment in Australia but disease onset and death occurred while a resident of the United Kingdom. This case is not included in the statistical analysis since morbidity and mortality did not occur within Australia.

‡ Includes 47 living cases.

The duration of illness for CJD cases varies depending on aetiology and other determinants. The median length of illness duration for all CJD cases was four months. For sporadic cases, the 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 eight 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 sex differences have been observed. Overall, 47.2 per cent of cases were male and 52.8 per cent were female. The average age of death in sporadic cases by sex was 65 years (range, 25-89) for males and 66 years (range, 33-89) for females. Over the period of 1970 to 2004, there was no difference between the average age-specific mortality rates of males (0.62 deaths/million/year) and females (0.68 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 (4.6 deaths/million/year).

In comparison to sporadic cases, the average death age of familial cases was 51 years (range, 20-82 years) in males and 62.5 years (range, 42-82 years) in females. Peak mortality rates occurred in the 65-69 year age group in both males (0.26 deaths/million/year) and females (0.41 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 geographical distribution of sporadic CJD cases showed no significantly increased risk for any individual Australian state or territory. The number of total TSE deaths by state or territory between 1993 to 2004 is shown in Table 2 and reflects geographical population distributions. Crude incidence rates show little variability in the larger regions of Australia and are similar to international rates where similar surveillance mechanisms are in place. The lowest rates were observed in Tasmania and the Northern Territory and may suggest lower ascertainment. No geographical birth region of sporadic CJD cases demonstrated a significantly increased or decreased rate of sporadic CJD incidence.

The notification of suspect cases to the ANCJDR initially peaked (132 cases) during the first year of the registry's surveillance. This was the result of the investigation of the Australian Institute Health and Welfare (AIHW) death certificate searches, which ascertained cases retrospectively to 1988. Further peaks of referrals were observed in 1995-1996 (129 and 125 cases respectively) and again in 1999 (103 cases). The 1995-1996 consecutive peaks were a direct result of AIHW death certificate and hospital and State Morbidity data searches while the 1999 peak was representative of an increased level of acceptance and utilisation of the 14-3-3 cerebrospinal fluid (CSF) diagnostic test by clinicians. More recently, referrals have plateaued with around 60–70 cases referred to the registry each year for evaluation. Overall, the large majority of notifications of suspect cases have been obtained by personal communication from clinicians (34.5%), CSF 14–3–3 protein test request

Table 2. Transmissible spongiform encephalopathy deaths, 1993 to 2004, by state and territory

State/ territory	TSE cases by year of death												Total	Mean crude mortality rate (deaths/ million/yr)
	1993	1994	1995	1996	1997	1998	1999	2000	2001	2002	2003	2004		
ACT		1					1			1			3	0.80
NSW	2	3	7	6	10	10	13	12	9	4	7	7	90	1.17
NT						1							1	0.44
Qld	5	2	5	6	3	3	7	7	3	3	3		47	1.14
SA	1	3	2	3	3	1	3	2			1	1	20	1.12
Tas				1						2			3	0.53
Vic	10		4	8	5	9	3	9	10	5	8	3	74	1.32
WA	2	3	3	4	3	3	1	2	1	2	2	2	28	1.29

(34.1%), death certificates (13.3%) and hospital and health department searches (12.2%). Since 1998, the diagnostic CSF test has been the most dominant initial notification source of definite and probable cases (45–86%) of CJD cases. Compulsory notification of suspect CJD cases has been implemented in four Australian states and territories since 2003–2004. The effect of scheduling CJD as a notifiable disease will be closely monitored by the ANCJDR. At present, there has been no demonstrable change to the number of referrals.

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A comparison of a rapid test for influenza with laboratory-based diagnosis in a paediatric population

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Abstract

The rapid and accurate detection of influenza A and B in a hospital setting is useful to confirm infection, exclude other diseases and assist in the management of patient illness including the possible use of specific antiviral therapy. We evaluated the use of the Directigen Flu A+B in a paediatric hospital laboratory in comparison with the established diagnostic tests direct immunofluorescence, viral culture and reverse transcriptase-polymerase chain reaction. A total of 193 respiratory specimens were examined and the Directigen test detected positive samples with an 80.8 per cent sensitivity and a specificity of 100 per cent. This study confirms other paediatric studies which have found the Directigen Flu A+B to be less sensitive than traditional laboratory tests but nevertheless to have a potential role in patient management especially when a positive result is obtained. *Commun Dis Intell* 2005;29:272–276.

Keywords: influenza, rapid tests, Directigen, point of care, diagnostic

Introduction

Influenza is a major cause of respiratory disease outbreaks in the winter months and while it is usually a self-limiting disease in healthy individuals, it can cause severe illness and mortality in the elderly, immunosuppressed and very young patients.[1,2] In children, influenza has been associated with increased outpatient visits, hospital admissions and antibiotic usage.[3,4] However, rapid diagnosis of influenza has been shown to significantly alter the management of the patient's illness, resulting in a reduction in diagnostic tests performed, reduced antibiotic use, increased antiviral use and reduced length of stay in a hospital emergency department.[5]

A number of laboratory tests are used for the diagnosis of influenza but most require highly skilled laboratory staff and equipment, and are often too time consuming to be useful in determining timely treatment options. Recently however, a number of rapid tests for influenza have become available which are simple and can be performed outside the laboratory without specialised equipment or extensive training.[6,7] The major limitations in using these tests have been the lack of sensitivity and specificity when compared to standard laboratory tests. Their performance has also been shown to be variable depending on the age of the study group and the type of sample being tested. The highest sensitivity with rapid test kits has been reported in studies from young children using nasopharyngeal aspirates (NPA) or swabs as the respiratory sample and even when testing is limited to these types of samples, some variation has been reported.[6–13] We undertook the current study to evaluate the Directigen Flu A+B rapid test using mainly NPA samples from children in comparison with three laboratory diagnostic tests used for influenza diagnosis, direct immunofluorescence (DIF), rapid enhanced tissue culture combined with immunofluorescence (RETCIF) and a multiplex reverse transcriptase polymerase chain reaction (RT–PCR) for the differential detection of influenza A and B viral genes.

Materials and methods

Respiratory samples were obtained from patients with acute respiratory symptoms attending the Royal Children's Hospital, Melbourne, between August and the beginning of October 2003. Samples were processed for routine viral diagnosis in the virology laboratory using DIF and RETCIF as previously described.[14] Samples were tested using the Directigen Flu A+B rapid test (Becton Dickinson and Co., Maryland, USA) as per the manufacturer's recommendations. A test was performed using internal kit positive and

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negative controls on each new kit. Specimens for the rapid test were prepared by aliquoting 200 µl of NPA (or bronchoalveolar lavage) into a tube and adding eight drops of extraction buffer (Reagent E) according to the manufacturer's instructions. Swabs were extracted in 16 drops of extraction buffer. The preparation and testing procedure takes approximately 10–15 minutes to perform depending on the number of samples tested and the result is read by eye. An aliquot (300–500 µl) of the original specimen was stored at –70° C for the RT–PCR assay. A non-nested, in-house multiplex RT–PCR assay was used for the detection of influenza type A and influenza B. Briefly, primers used to detect the influenza A matrix gene (amplicon size 322 bp) and the influenza B NS gene (amplicon size 109 bp) were modified from Poddar[15] (primer sequences available on request). Viral RNA was extracted from 140 µl of clinical sample using the QIAamp® Viral RNA Mini Kit (QIAGEN, Australia) in accordance with the manufacturer's instructions. RT–PCR was carried out using the Titan One Tube RT–PCR System (Roche, Australia) according to the manufacturer's recommendations using 5 µl of the extracted RNA per reaction with an PTC–200TM thermocycler (MJ Research, Waltham, MA). PCR product was analysed by gel electrophoresis using 10 µl of amplified product which was run on a 2.5 per cent agarose gel containing ethidium bromide with control influenza A and B samples.

Results

In this study we analysed a clinical sample from each of 193 paediatric patients aged from nine days to 15 years (66% of samples were from patients aged two years or younger) of which 53.4 per cent were male and 46.6 per cent were female. The sample types consisted of 183 nasopharyngeal aspirates, four nasal swabs, three throat swabs and three bronchoalveolar lavages. Of the 193 specimens examined, 99 were considered positive for influenza by DIF. All influenza isolates were influenza A with no influenza B viruses and when sub-typed using DIF, all were A(H3) with no A(H1) viruses. A small number (less than 10%) of samples required re-testing after dilution as they gave invalid results initially but on dilution and re-testing gave valid results. The Directigen kit was easy to use and detected influenza A in 80 samples and no influenza B. When this was compared to the results obtained with DIF testing (Table 1) the Directigen kit showed a sensitivity of 80.8 per cent and a specificity of 100 per cent (Table 2). No false positives were obtained with the kit, giving a 100 per cent PPV (positive predictive value) but only an 83.2 per cent NPV (negative predictive value) when compared to DIF (Table 2). All of the samples that were positive by DIF also yielded positive isolates using cell culture in combination with IF using the RETCIF method, therefore comparisons with Directigen and RETCIF were identical to those made with Directigen and DIF (Tables 1 and 2). The sensitivity of the Directigen test compared to DIF and RETCIF was also analysed in three different age groupings. Children 0–2 years had an 87.5 per cent sensitivity, children five years and below had a sensitivity of 83.5 per cent and children 6–15 years had a sensitivity of 62.5 per cent.

Table 1. Detection of influenza A virus in clinical samples by DIF/RETCIF, multiplex RT–PCR* and Directigen Flu A+B

	DIF or RETCIF +	DIF or RETCIF –	RT–PCR+	RT–PCR–
Directigen +	80	0	78	2
Directigen –	19	94	16	95

* Note that two samples were unavailable for RT–PCR testing.

Table 2. Comparison of performances of the Directigen Flu A+B rapid test kit to DIF or RETCIF or multiplex RT–PCR, and comparison of the performance of multiplex RT–PCR to the DIF or the RETCIF assay

	Directigen Flu A+B compared to:		RT–PCR compared to:
	DIF or RETCIF	Multiplex RT–PCR	DIF or RETCIF
Sensitivity %	80.8	83.0	95.9
Specificity %	100	97.9	100
PPV %	100	97.5	100
NPV %	83.2	85.6	95.9
Accuracy %	90.2	90.6	97.9

A comparison of the Directigen test with an in-house non-nested multiplex RT–PCR assay yielded similar results to the use of DIF or RETCIF as the comparators with a sensitivity and specificity of 83 per cent and 97.9 per cent, respectively (Tables 1 and 2). When the RT–PCR assay was compared to the DIF or RETCIF results, the concordance was very good with discrepant results seen in only four of 191 samples, giving the RT–PCR assay a sensitivity of 95.9 per cent and specificity of 100 per cent with PPV and NPV of 100 per cent and 95.9 per cent respectively (Table 2). All four of these discrepant results were from samples that were positive by DIF or RETCIF and two were also positive by Directigen. Two of these samples (NPA's) yielded smeared PCR product on both initial testing and repeat testing which were considered inconclusive and scored as negative while the other two samples failed to produce detectable PCR product. Twenty-four non-influenza viruses were also detected using routine DIF and culture with IF [11 respiratory syncytial virus (RSV), five cytomegalovirus (CMV), five parainfluenza–3 (PI–3) and three dual infections – RSV+PI–3, RSV+CMV and RSV+herpes simplex virus 1]. The Directigen kit and the RT–PCR were negative for all these samples.

Discussion

The results in this study using the Directigen Flu A+B compared favourably with other studies in paediatric populations where influenza A was detected. Studies which also used DIF or viral culture as the 'gold standards' have reported sensitivities and specificities of 96 per cent and 99.6 per cent,[9] 86.6 per cent and 100 per cent,[10] 95 per cent and 88 per cent,[11] 43.8 per cent and 99.7 per cent,[12] 60 per cent and 100 per cent[13] respectively, for paediatric patient groups compared to our results of 80.8 per cent and 100 per cent. The studies by Cazacu *et al*[12] and Landry *et al*[13] obtained much lower sensitivity with the Directigen Flu A+B kit than in our study and other similar studies.[9,10,11] The reasons for this large difference are not apparent however some differences in the sample type were present with one study[12] using mainly nasal washes in their trial and the other[13] using a mixture of NP swabs and aspirates. In the test results contained in the BD Directigen Flu A+B kit booklet, when compared to virus isolation, NPA's gave the highest sensitivity followed by nasopharyngeal washes and nasopharyngeal swabs followed by throat swabs and lower nasal swabs. Also reported in the booklet is up to a 1,000-fold difference in the detection limits (as measured by $CEID_{50}$) for different viruses [A(H1N1), A(H3N2) and B], for example the 2 A(H3N2) viruses listed showed a 100-fold difference in detection limit. As the various studies were conducted at different times, different viruses may have been circulating in the studies that may be detected at varying levels with the Directigen rapid test. Influenza B has been reported to be detected at a similar[12] or lower level[9,10,13] than influenza A using the Directigen Flu A + B kit however as no B viruses were detected in our study this can not explain the discrepancy with some of these other studies. The proportion of younger children in each study may also affect the sensitivity of the Directigen Flu A+B kit. A consistent finding between studies has been the higher sensitivity of the kit when used on samples from children[16] compared to adults, especially in young patients (≤5 years[11]) or those aged under two years.[12] In our study the sensitivity increased to 87.5 per cent for samples from patients ≤2 years compared to an overall sensitivity of 80.6 per cent and decreased to 62.5 per cent with older children (6–15 years). As the majority of our samples were obtained from ≤2-year-old children, this may have contributed to the higher overall sensitivity compared to other studies.

The high PPV seen in our study (100% i.e. no false positives) and others (100%[10], 96%[9] and 90.5%[13]) with influenza A detection by the Directigen test, should give confidence to technicians and paediatricians that when they obtain a positive test, they can confidently confirm a clinical diagnosis or begin appropriate treatment, with the option of using a specific influenza antiviral drug immediately. A rapid positive result could reduce the need for further laboratory testing which would help offset the cost of the kit and performing the test. A previous study has shown rapid diagnosis of respiratory viral infections in children can result in significant reductions in hospital stays and antibiotic use as well as laboratory savings.[17] On the other hand a more cautious approach is warranted if a negative Directigen result is obtained, in view of the higher proportion of false negative results seen when using this test (NPV in our study was 83.2%, and in others was 99.6%,[9] 92.1%,[10] and 96.9%[13]). Previous studies using the Directigen kit have also noted that the test can produce a number of indeterminate or invalid tests initially Ruest *et al*[10] found that eight per cent of samples tested fell into this category and required diluting and re-testing. In our study we found less than 10 per cent of samples gave invalid results initially but on dilution and re-testing gave valid results.

When the rapid test was compared to a multiplex RT–PCR assay, the sensitivity and specificity was improved slightly due to the RT–PCR not detecting 4/97 of the DIF/RETCIF positive samples, two of which were positive by the rapid test. One possible explanation for this might have been the extra freeze thaw step these samples had prior to RT–PCR assay, which may have caused degradation of the viral RNA. Others have reported a lower sensitivity than our study when comparing the Directigen kit with a multiplex real-time PCR[18] however this is not surprising given the added sensitivity of real-time PCR.

The impact of influenza in children has been highlighted in recent studies especially in those under five years[19] and during the winters in Australia and in the United States of America (USA) in 2003–04, where a number of deaths were associated with influenza. Some 152 deaths were reported in children under 18 years in the USA[20] while three deaths were reported in one hospital in Sydney, Australia.[21] Hence methods that will rapidly and accurately diagnose influenza in children would be a useful addition to our current range of tests both in the laboratory and also in the wider community. During outbreaks, hospitals might even consider outpatient testing of children who present with respiratory illness to allow segregation of any who test positive to reduce nosocomial infections and reduce further testing.[5,22,23] In conclusion, the Directigen Influenza A+B is a relatively simple test that performed well when using samples that would be expected to contain the highest viral loads (NPA samples from a paediatric population) but still failed to detect influenza A in around 20 per cent of positive samples as detected by DIF or RETCIF or RT–PCR. Newer rapid tests for influenza which are now available, promise even better results than the current ones.[24]

Acknowledgements

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SARS and biothreat preparedness – a survey of ACT general practitioners

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Abstract

In late 2003 and early 2004 the ACT Division of General Practice and ACT Health conducted two concurrent surveys designed to identify knowledge, attitudes and practices of Australian Capital Territory (ACT) general practitioners around severe acute respiratory syndrome (SARS) and biothreat preparedness. One survey asked individual general practitioners (GPs) about how they gathered information about SARS in 2003, how they preferred to receive information, current practices, and how they perceived the threat of SARS and other infectious agents. The second survey asked practice principals how they organised their practice to respond to the SARS threat in 2003, any difficulties they had with implementing this response, use of SARS infection control guidelines, and current policies. The response rate for the individual GP survey was 48 per cent (184/381) and the response rate for the practice organisation survey was 54 per cent (74/136). GPS used many sources of information on SARS during the 2003 outbreak. Facsimiles from the ACT Division of General Practice were the primary source (17%) and facsimile was the preferred method of receiving information in future outbreaks. The majority of GP respondents felt adequately informed about SARS during the 2003 outbreak, but many general practices did not follow the national guidelines on telephone screening of patients, warning signs and having infection control kits available. The majority of practices reported that they had policies or procedures in place to isolate potentially infectious patients from others in the waiting room. GPs rated an influenza pandemic as a threat to themselves and their patients much more highly than SARS or bioterrorism. Suggestions and comments on how ACT GPs could be better prepared to respond to future outbreaks included the need for timeliness of information, information delivery mechanisms, communication issues, education, the availability of guidelines and protocols, planning, role delineation, the use of response teams, provision of equipment, and vaccination. Planning for future infectious disease outbreak events in the Australian Capital Territory should incorporate general practitioners so that the plans reflect what is a feasible response in the general practice setting. *Commun Dis Intell* 2005;29:277–282.

Keywords: communicable diseases, disease control, severe acute respiratory syndrome

Background

In 2003, an outbreak of a new infectious disease, severe acute respiratory syndrome (SARS), caused a global public health emergency.[1] In Australia, an extensive response was mounted to the potential threat of SARS.[2] The SARS outbreak was also seen as a test for other potential infectious disease threats, such as the possibility of an influenza pandemic, or deliberate release of a bioterrorism agent such as smallpox or anthrax.

SARS is an example of an emerging disease with a potentially significant impact on primary health care, including general practice. There was an expectation in Australia that general practitioners (GPs) would be prepared to deal with possible cases of SARS, including screening patients and having infection control equipment available. Guidelines for general practitioners on SARS were posted on the Australian Government Website in April 2003[3] and GPs were encouraged to access this site. In the Australian Capital Territory (ACT) information was distributed from the Deputy Chief Health Officer to the ACT Division of General Practice, which subsequently sent this information via facsimile to all ACT general practitioners. In addition, many other sources of information were available, including medical journals, medical newspapers and the general media.

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Little was known about the effectiveness of various methods of rapid communication with GPs, and what communication methods are preferred by GPs in situations of rapid change. It was also not known whether general practices were adequately prepared to deal with possible SARS cases and what would help them prepare for any similar event in the future.

During the outbreak, GPs in the Australian Capital Territory raised a number of concerns around preparedness for emerging disease events. These concerns included information dissemination, guideline implementation, infection control, equipment requirements and costs, occupational health and safety, and workforce issues. GPs were also concerned about appropriate roles, relationships, and the onus of responsibility between the acute care, primary care and public health systems. Similar issues were raised in other countries.[4]

In late 2003 and early 2004 the ACT Division of General Practice and the ACT Health conducted two concurrent surveys designed to identify knowledge, attitudes and practices of ACT general practitioners around SARS and biothreat preparedness. The intention of the surveys was to provide information to help plan for a more cohesive and consistent response to any future outbreak or bioterrorism event.

Methods

The study was approved by the ACT Health and Community Care Human Research Ethics Committee.

Two concurrent surveys were mailed from the ACT Division of General Practice in November 2003, a time at which the crisis of the SARS outbreak had abated:

- an anonymous mail survey to all 381 ACT general practitioners on the ACT Division of General practice database (the Individual GP Survey). The questionnaire asked about individual knowledge, attitudes and practices around SARS and biothreat preparedness. It also asked about preferences for communication with health authorities in an outbreak situation, and for suggestions on improving preparedness.
- an anonymous mail survey to all 136 ACT general practices on the Division of General Practice database, to be completed by the practice principal and/or practice nurse and/or practice manager (the Practice Organisation Survey). Practice principals were asked about how their practice responded to the SARS outbreak of 2003, and current policies and resources for SARS response and biothreat preparedness.

Practice principals were asked to complete both surveys—one in their capacity as practice principal from the perspective of the practice, and the other from the perspective of an individual GP.

The ACT Division of General Practice database of general practitioners was considered the most comprehensive available at the time for GPs' names and contact details. The database included all ACT GPs whether they were Divisional members or not.

Both surveys included a covering letter co-signed by the President of the ACT Division of General Practice and the ACT Deputy Chief Health Officer, a self-completion survey and a reply paid envelope. Two weeks after the initial mail-out reminder letters and duplicate surveys were sent out.

In late January and early February 2004, following an initial low response rate, all 136 ACT general practices were telephoned as a follow-up and encouraged to respond to the Practice Organisation Survey in particular.

Data entry and analysis were done in Epi Info 2002.[5] Analyses were based on the number of respondents who completed each question rather than the total number of respondents.

Results

Response rates

The response rate for the Individual GP Survey was 48 per cent (184/381), while the response rate for the Practice Organisation Survey was 54 per cent (74/136). Twenty-seven surveys were posted following the telephone reminder calls. This lifted the response rate for the practice organisation survey from 40 per cent to 54 per cent. Response rates to individual questions ranged from 91 per cent to 100 per cent.

Individual GP Survey

Of the GPs who responded to demographic questions, 54 per cent were female, 69 per cent were in the 41–60 years age group and 61 per cent worked seven or more sessions per week in general practice.

GPs reported that during the 2003 SARS outbreak they used many sources of information, particularly facsimiles and newsletters from the ACT Division of General Practice, but also websites, the Australian Government hotline, medical journals, medical newspapers and the mainstream media (Figure 1). When asked how they would prefer to receive information in the future in the event of a serious outbreak GPs nominated facsimile (38%), the Division of General Practice newsletter (13%), the Australian Government Department of Health and Ageing website (8%)

Figure 1. Sources of information on severe acute respiratory syndrome used by Australian Capital Territory general practitioners during the 2003 outbreak (n=875)

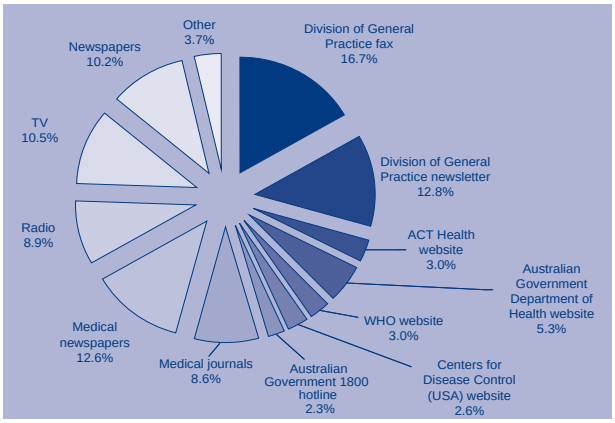
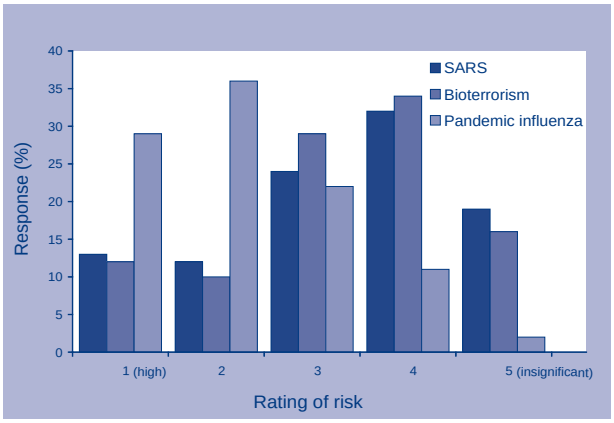


Figure 2. Australian Capital Territory general practitioners' ratings of the risk of severe acute respiratory syndrome, bioterrorism and pandemic influenza as a threat to themselves and their patients (n = 173)



and the ACT Health website (7%). Other responses included the mainstream media, the medical media, other websites, a hotline, email and mobile phone messaging.

The majority of respondents stated that they felt adequately informed about the SARS outbreak in 2003 (83%), about the threat to health care workers (76%) and about the recommended response to a suspected SARS case presenting to their practice (70%).

When asked about current practices, 32 per cent of GP respondents reported they always asked patients with a fever about travel (66% sometimes ask) and 19 per cent asked patients with respiratory symptoms about travel (74% sometimes ask).

Fifty-five per cent of GP respondents reported having an influenza vaccine every year, 23 per cent most years and 22 per cent reported that they never have an annual influenza vaccine. Seventy-two per cent of respondents had previously been vaccinated against smallpox and 59 per cent would be prepared to be vaccinated against smallpox if a realistic threat were identified. Fifteen per cent responded that they were not prepared to be vaccinated against smallpox and 25 per cent said they did not know.

GPs rated an influenza pandemic much more highly as a threat to themselves and their patients than SARS or bioterrorism. (Figure 2).

There were 67 suggestions or comments about how ACT GPs could be better prepared to respond to future outbreaks. These covered a number of topics including: the need for timely information; effective information delivery mechanisms; better communication within and between health agencies; education and training needs; the need for appropriate and useful guidelines and protocols; disaster and outbreak planning; the need for clear role delineation in outbreak responses; the use of response teams or centralised assessment centres; funding and provision of specialised equipment; and vaccination.

Practice Organisation Survey

Practice principals or their representatives reported that during the 2003 SARS outbreak there was variability in the way patients were screened and in the way the practices prepared for possible SARS cases. Patients were more likely to be screened when they presented at the surgery than by phone, and 30 per cent or more of patients were not screened (Table). Many practices did follow other preparedness recommendations, such as the placement of SARS advisory signs and having surgical masks available (Table).

The majority of respondents (67%) reported no problems with implementing a screening process to identify suspected SARS cases in the practice. Screening measures ceased within two months of the end of the outbreak in 49 per cent of practices.

The *Australian Government SARS Infection Control Guidelines for General Practice* were accessed by 46 per cent of respondents and, of these, 79 per cent found them useful. Of the four practices that did not find the guidelines useful, a number of reasons were cited, including that: there were other sources of guidelines, the guidelines were not appropriate for general practice, the guidelines were 'overkill', and the practice had no suspected cases.

Table. Reported patient screening and preparedness responses consistent with *Australian Government SARS Infection Control Guidelines for General Practice* by Australian Capital Territory general practices during the 2003 severe acute respiratory syndrome outbreak

Question: During the 2003 SARS outbreak did your practice	Number of respondents	Yes		Don't know		No	
		n	%	n	%	n	%
Ask patients about travel when they rang for an appointment?	72	30	42	3	4	39	54
Ask patients about fever when they rang for an appointment?	73	21	29	5	7	47	64
Ask patients about travel when they presented at the surgery?	71	44	62	4	6	23	32
Ask patients about fever when they presented at the surgery?	73	39	53	7	10	27	37
Have an identified person in the practice who regularly checked which countries / regions were currently SARS affected?	73	31	43	0		42	58
Have a SARS advisory sign at the entrance to the surgery?	74	43	58	0		31	42
Have surgical masks available for suspected SARS cases to put on in the waiting room?	74	52	70	0		22	30
Buy new equipment specifically to deal with SARS?	70	35	50	0		35	50

SARS Severe acute respiratory syndrome.

Fifty per cent of respondents reported buying equipment specifically to deal with SARS. Equipment purchases included surgical masks (22%), disposable gowns (17%), disposable gloves (11%), hand cleaning products (10%), thermometers or thermometer covers (9%), disinfectants (8%), protective eyewear (8%) and P2 (N95) masks (8%). Cost of the new equipment ranged from $10 to $1,000 (median $200). Many respondents had problems obtaining equipment, including reduced availability, cost and long waiting times.

The majority of respondents (65%) reported that they currently had policies or procedures in place in their practice to isolate potentially infectious patients from others in the waiting room, and 81 per cent reported they had a separate room available for isolation. Forty-nine per cent of respondents reported the practice had a practice nurse and, of these, 61 per cent of practice nurses were trained in triage.

When asked for comments on how general practices could be assisted to better prepare for future outbreaks, there were 38 responses. Many of these echoed the responses in the Individual GP Survey. Comments included the need for timely information, detailed guidelines appropriate for general practice, workshops and practical scenario style education, organised supply of equipment, greater public education, planning which includes GPs, more money and response/crisis teams.

Discussion

These surveys demonstrate general practice responses in the Australian Capital Territory to the 2003 SARS outbreak, and GP knowledge, attitudes, policies and practices regarding biothreat preparedness.

Targeted and timely information dissemination to health practitioners from a recognised authority is important during a public health emergency such as the SARS outbreak. GP respondents reported facsimile to be the most frequent method by which they obtained information about SARS during the 2003 outbreak. Facsimile was also GPs' preferred method for receiving timely information about any future outbreak or event. A facsimile stream from the ACT Division of General Practice to all general practices in the ACT requires relatively small resources to achieve fairly comprehensive coverage. However, this should not preclude exploration of other methods of rapid communication with GPs for use in public health emergencies. In particular, electronic information dissemination may become more common as its day-to-day use increases in general practice. Methods such as email and mobile phone messaging (SMS) were preferred by some respondents and these have also been suggested in New South Wales.[6]

For the purposes of this study, and for rapid public health communication with GPs, the ACT Division of General Practice database was considered the most comprehensive list of ACT GPs and general practices. Other possible sources of GP lists were pathology provider or hospital databases.[7] Since the survey, the Australian Capital Territory has moved ahead with the development of a single service provider database, which will be used across the Australian Capital Territory by public hospitals and the ACT Division of General Practice. This should increase the accuracy of mailing lists as the database will have daily use and regular updating. In South Australia, a GP registry has similarly been developed for rapid communication between public health authorities and primary care providers.[8]

While the majority of GP respondents felt adequately informed about SARS during the 2003 outbreak, fewer than 50 per cent of practices accessed the national SARS guidelines for general practice on the Australian Government website. Practices may have had access to the guidelines from other sources, but many practices did not routinely follow the recommendations in the guidelines. While this survey did not probe the reasons for compliance (or non-compliance) with the guidelines, it is possible GPs did not perceive SARS as a significant enough threat to themselves or their patients and were reluctant to change routine practices. In Hong Kong at the time of the SARS outbreak, GPs independently instituted preventive measures in the absence of specific guidelines.[9] In this case the perception of risk to GPs was realistically high. In Australia, lack of compliance with the guidelines may also reflect difficulties in their implementation in the general practice setting.

The National Health and Medical Research Council recommends that health care providers are vaccinated against influenza[10] but we found that only 55 per cent of GP respondents had an annual influenza vaccination. Such immunisation protects the GP themselves and is recommended in order to protect patients who are at high risk. GPs' reasons for and against their own immunisation is an issue which could be explored further. Similar vaccination levels have been reported in an Australian tertiary hospital, and coverage was not improved by the introduction of a hospital vaccination policy.[11,12] New strategies to improve vaccination coverage in general practice staff also need to be investigated.

Appropriate immunisation of staff and appropriate infection control procedures are linked to general practice accreditation through the Royal Australian College of General Practitioners *Standards for General Practice.*[13] However, even universal application of these standards may not be adequate in special circumstances such as the SARS outbreak, when special precautions need to be put in place. Continuing professional development for GPs about biothreats and other emerging disease events could provide opportunities to raise awareness of biothreat preparedness issues and to engage GPs in biothreat response planning.

General practitioners in the Australian Capital Territory considered pandemic influenza to be a more important threat to themselves and their patients than SARS or bioterrorism. Consequently, GP engagement in planning for infectious disease outbreaks may be higher if based around influenza rather than other agents. Planning for an influenza pandemic is also likely to be applicable to other disease scenarios, such as a SARS outbreak or a bioterrorism event.

Barriers identified by GPs in implementing the SARS guidelines should be taken into consideration when planning for possible outbreak or bioterrorism events. Issues include effective communication methods, clear role delineation for all participants in a response, the use or otherwise of response teams or centralised assessment centres, and supplies of specialised equipment. In addition, issues around remuneration for general practitioners who participate in public health activities (such as response teams) need to be considered in advance of a serious outbreak, particularly where such activities are not billable under the Medicare Benefits Scheme. Many of the barriers to an effective response raised by ACT GPs have been recognised internationally. A review of the SARS outbreak in Hong Kong and Toronto provided recommendations on: improving communication; integration of health services; surge capacity; infection control policies, plans and procedures; and occupation health measures.[14]

Limitations of this study include response rates, overlap between surveys and self-reporting. The response rates of 48 per cent for the Individual GP Survey and 54 per cent for the Practice Organisation Survey mean that the results may not be able to be generalised to the whole of the general practice population of the Australian Capital Territory. As the responses to the surveys were anonymous, it was not possible to obtain information about the non-responders. Reminder telephone calls to general practices improved the response rate to the Practice Organisation Survey and this technique could be used in future surveys. A number of practice principals would have completed both the Individual GP Survey and the Practice Organisation Survey. While the questions in the surveys were different, it is possible that possible that practice principals may have answered differently because they saw both questionnaires. Self-reported behaviour does not neces-

sarily represent actual practice, and could result in an overestimation of compliance with guidelines. As our survey was anonymous and voluntary we consider this is unlikely to have significantly affected the validity of our results.

Australia's only confirmed case of SARS in 2003 was identified retrospectively and was seen not by a hospital but by a general practitioner.[15] This highlights the importance of effectively including general practitioners in preparing for any future serious outbreak of an emerging infectious disease. The findings of our study show some strengths in general practice but also highlight areas where improvements can be made. In particular, planning for future emerging disease outbreak events in the Australian Capital Territory should incorporate general practitioners so that the plans reflect what is a feasible response in the general practice setting.

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Influenza and pneumococcal vaccine coverage among a random sample of hospitalised persons aged 65 years or more, Victoria

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Abstract

This study was undertaken to assess the uptake of influenza and pneumococcal vaccination based on provider records of the hospitalised elderly, a group at high risk of influenza and pneumococcal disease. The study used a random sample of 3,204 admissions at two Victorian teaching hospitals for patients, aged 65 years or more who were discharged between 1 April 2000 and 31 March 2002. Information on whether the patient had received an influenza vaccination within the year prior to admission or pneumococcal vaccination within the previous five years was ascertained from the patient's nominated medical practitioner/vaccine provider. Vaccination records were obtained from providers for 82 per cent (2,804/2,934) of eligible subjects. Influenza vaccine coverage was 70.9 per cent (95% CI 68.9–72.9), pneumococcal coverage was 52.6 per cent (95% CI 50.4–54.8) and 46.6 per cent (95% CI 44.4–48.8) had received both vaccines. Coverage for each vaccine increased seven per cent over the two study years. For pneumococcal vaccination, there was a marked increase in 1998 coinciding with the introduction of Victoria's publicly funded program. Influenza and pneumococcal vaccine coverage in eligible hospitalised adults was similar to, but did not exceed, estimates in the general elderly population. Pneumococcal vaccination coverage reflected the availability of vaccine through Victoria's publicly funded program. A nationally funded pneumococcal vaccination program for the elderly, as announced recently, should improve coverage. However, these data highlight the need for greater awareness of pneumococcal vaccine among practitioners and for systematic recording of vaccination status, as many of these subjects will soon become eligible for revaccination. *Commun Dis Intell* 2005;29:283–288.

Keywords: communicable diseases; disease control; influenza; pneumococcal infections; vaccines

Background

Every Australian aged 65 years or more is considered to be at an increased risk of influenza and invasive pneumococcal disease, with the risk of adverse outcomes likely to be even greater among the hospitalised elderly due to other comorbidities.[1] Influenza and 23-valent polysaccharide pneumococcal vaccines (23vPPV) are both recommended for people in this age group (annually for influenza but only one dose with a single revaccination five years later for 23vPPV).[1] The influenza vaccine has been free for all elderly Australians under the national immunisation program since 1999 while 23vPPV was added to the national program in 2005.[2] Although not nationally funded, a state based program of free 23vPPV for the elderly has operated in Victoria since 1998.[3]

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Two national telephone surveys have estimated influenza and pneumococcal vaccine coverage based on self-report. In 2000, influenza vaccine coverage among persons aged 65 years or more in Victoria was reported at 75 per cent with 40 per cent having received 23vPPV within the previous five years.[4] In the following year, influenza vaccination coverage among elderly Victorians was estimated at 81 per cent.[5]

Since self-reported pneumococcal vaccination status is unreliable[6–9] we aimed to assess the uptake of pneumococcal and influenza vaccines based on provider records. We estimated coverage in Victoria, where both vaccines are publicly funded, among a cohort of hospitalised persons aged 65 years or more.

Methods

We determined influenza and pneumococcal vaccination status from a random sample of persons aged 65 years or more who had been discharged from the Royal Melbourne Hospital or the Western Hospital between 1 April 2000 and 31 March 2002. At the conclusion of each month throughout the study period, subjects were randomly selected (using a random number generator) from a list of persons aged ≥65 years who had been discharged from each hospital in that month. If the subject appeared on the list more than once in any given month (more than one discharge recorded for that month), we selected one admission at random and excluded other admissions. For repeat admissions over numerous months, we retained the first selected admission and excluded all subsequent admissions. We also excluded non-residents of Victoria, those aged <65 years, and day admissions for dialysis or chemotherapy (ICD-10-AM codes Z49.1, Z49.2 and Z51.1).

We contacted each subject or their next of kin by telephone and, after obtaining verbal consent to participate, requested permission to contact the subject's general practitioner or other vaccine providers. We contacted these providers in writing and asked if they had a record of influenza vaccine within the year prior to admission or pneumococcal vaccination within the five years prior to admission. If so, we requested the specific vaccination date.

Our study was a component of a case-cohort study aimed at assessing vaccine effectiveness against community-acquired pneumonia. For this reason we report vaccination coverage of the cohort in terms of protection. Vaccination was considered protective if it was given between 14 days and one year prior to hospital admission for influenza vaccine or 14 days and five years prior for pneumococcal vaccine. Subjects for whom the provider indicated the vaccine was given but gave no vaccination date, an incomplete date or a date within 14 days of admission were excluded.

Since subjects were selected from a monthly list of discharged patients, even if they were selected only once, they were more likely to have been selected if they had been frequently admitted over the study period than if they had been admitted only once. We adjusted for this potential bias at the conclusion of the data collection period by calculating vaccination coverage as a weighted average. The weighting was the inverse of the total number of months where the subject had at least one discharge recorded from the hospital during the study period. We report both weighted and unweighted coverage estimates.

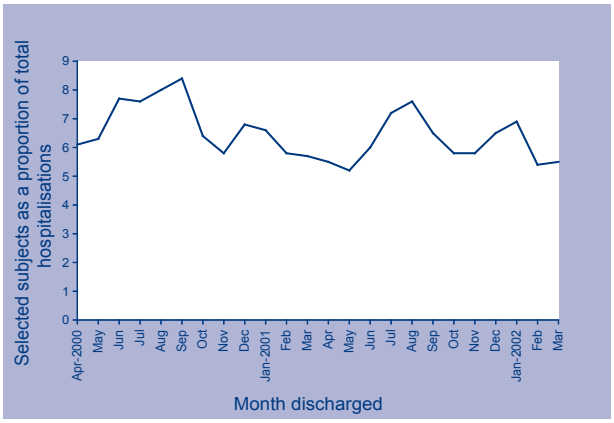
We estimated annual influenza vaccine coverage and 23vPPV coverage within the previous five years. Exact 95 per cent confidence intervals for proportions were calculated using Stata 8.0.[10] We compared 23vPPV coverage by year of vaccination from our study against the number of 23vPPV doses available in Victoria. Vaccine dose data were obtained from two sources: 23vPPV prescriptions issued from 1992 to 2001 by State;[11] and 23vPPV doses distributed through Victoria's publicly funded program (i.e. no prescription required) from 1998 to 2001 (personal communication, Ted Jamieson, Department of Human Services, Victoria, March 2003). Our study was approved by the Human Research Ethics Committee, Royal Melbourne Hospital Research Foundation (ref 2000.022).

Results

There were 83,280 hospital separations coded for persons aged 65 years or more during the two-year study period of which 27,372 (33%) were day admissions for dialysis or chemotherapy and were excluded. A further 6,216 (7%) hospital separations were excluded as repeat separations for the same person in that month. We randomly selected 3,204 (6%) from the remaining 49,692 separations. The proportion of subjects selected from the hospital discharge list each month ranged from 5.2 per cent to 8.4 per cent, with peaks over winter months corresponding to the increase in the number of cases selected in the case-cohort study (Figure 1).

Of the 3,204 randomly selected admissions, 202 (6%) were excluded because they were repeat admissions for previously selected subjects and 68 (2%) were excluded for various other reasons (Figure 2). The median age of the remaining 2,934 eligible subjects was 75 years (range 65–102 years) and 51 per

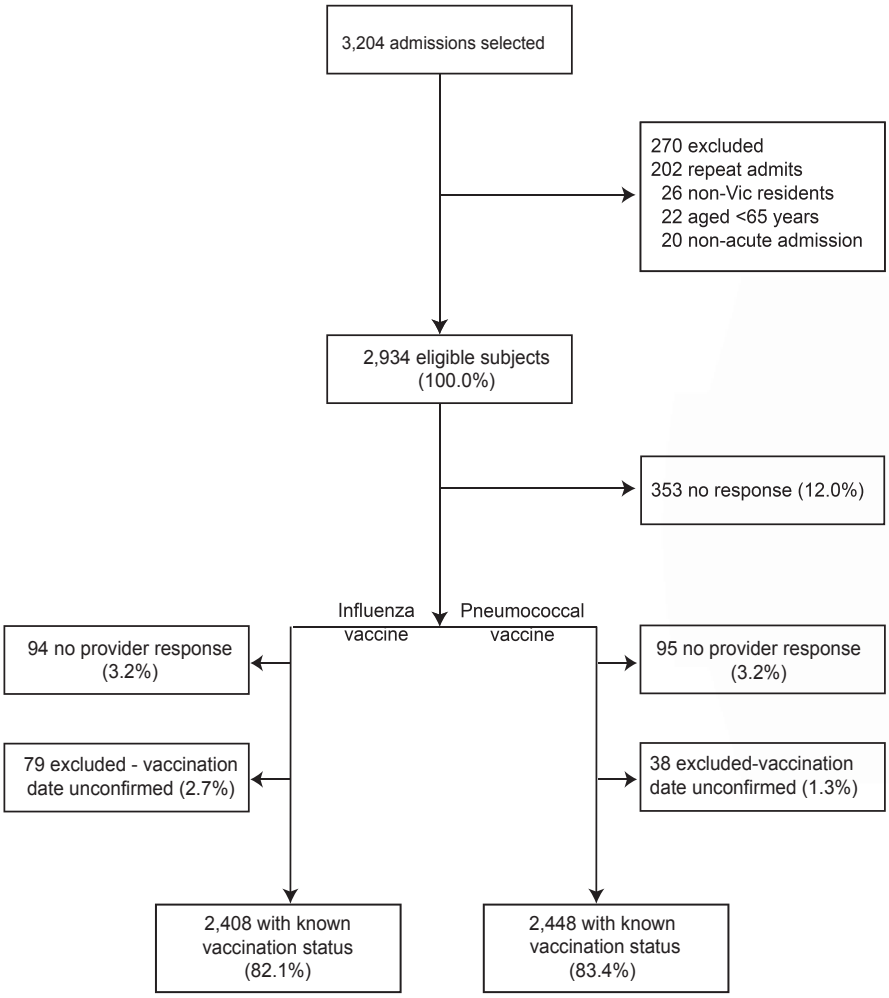
Figure 1. Proportion of hospitalisations selected from monthly list of discharge diagnoses for persons aged 65 years or more, Victoria, 1 April 2000 to 31 March 2002



cent were male. We ascertained influenza vaccination status from nominated providers for 82 per cent (2,408/2,934) of subjects, 23vPPV status for 83 per cent (2,448/2,934) and both vaccines for 81 per cent (2,380/2,934). There was no difference in age and sex distribution between those eligible subjects for whom we ascertained vaccination status and those for whom we did not (data not shown).

The weighted estimates of vaccine coverage were 70.9 per cent (95% CI 68.9–72.9) for influenza vaccination within the year prior to admission, 52.6 per cent (95% CI 50.4–54.8) for 23vPPV within five years prior to admission, and 46.6 per cent (95% CI44.4–48.8) for both vaccines. These estimates were virtually identical to unweighted estimates (unadjusted for selection probability), suggesting subjects with repeated admissions over the study period did not bias the coverage estimate (Table).

Figure 2. Response rate for ascertainment of influenza and pneumococcal vaccination status among hospitalised persons aged 65 years or more, Victoria, 1 April 2000 to 31 March 2002



Coverage for each vaccine increased from the first study year (April 2000-March 2001) to the second (April 2001-March 2002). Even though influenza vaccine coverage was substantially higher, the overall increase in coverage was similar for each vaccine: 7.1 per cent (95% CI 3.1–11.1) for influenza vaccine, 7.2 per cent (95% CI 2.9–11.5) for 23vPPV and 7.9 per cent (95% CI 2.9–11.5) for those who had received both vaccines (Table). Comparison of vaccine coverage by age group suggests the overall increase was evenly distributed across each five-year age stratum over 65 years for influenza vaccination whereas increases in the point estimates for 23vPPV were limited to those under 85 years (Figure 3). The greatest increase in 23vPPV coverage occurred in 1998 coinciding with the commencement of Victoria's publicly funded program and peak in vaccine availability (Figure 4).

Table. Influenza and pneumococcal vaccine coverage (weighted and unweighted) among a cohort of hospitalised persons aged ≥65 years, Victoria, 1 April 2000 to 31 March 2002, by year of discharge

Vaccination coverage	Influenza vaccine		23vPPV		Both vaccines	
	%	(95%CI)	%	(95%CI)	%	(95%CI)
Total (unweighted)	70.7	(68.8–72.5)	52.4	(50.4–54.4)	46.4	(44.4–48.4)
Total (weighted)	70.9	(68.9–72.9)	52.6	(50.4–54.8)	46.6	(44.4–48.8)
Study year 1 (weighted)	67.4	(64.5–70.2)	49.1	(46.1–52.1)	42.8	(39.8–45.8)
Study year 2 (weighted)	74.5	(71.7–77.2)	56.3	(53.2–59.4)	50.7	(47.5–53.9)
Increase (weighted)	7.1	(3.1–11.1)	7.2	(2.9–11.5)	7.9	(3.6–12.3)

Influenza vaccine within the year prior to admission, 23vPPV within five years prior to admission.

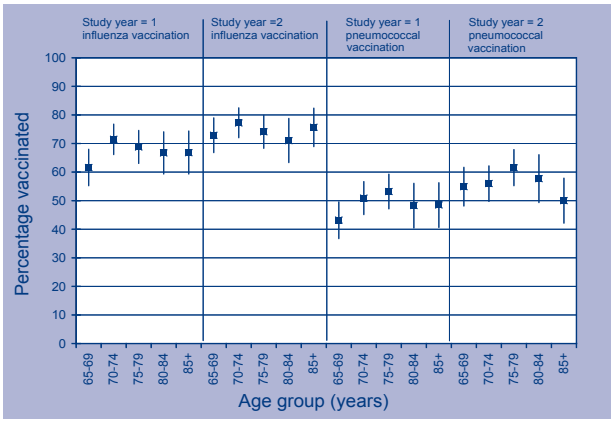
Weighted coverage estimates adjust for probability of selection (see Methods).

Study year 1 = Subjects discharged between 01/04/2000 and 31/03/2001.

Study year 2 = Subjects discharged between 01/04/2001 and 31/03/2002.

Increase refers to increase in coverage between Study year 1 and Study year 2.

Figure 3. Influenza and pneumococcal vaccine coverage among a cohort of hospitalised persons aged 65 years or more, Victoria, 1 April 2000 to 31 March 2002, by age group and year of discharge

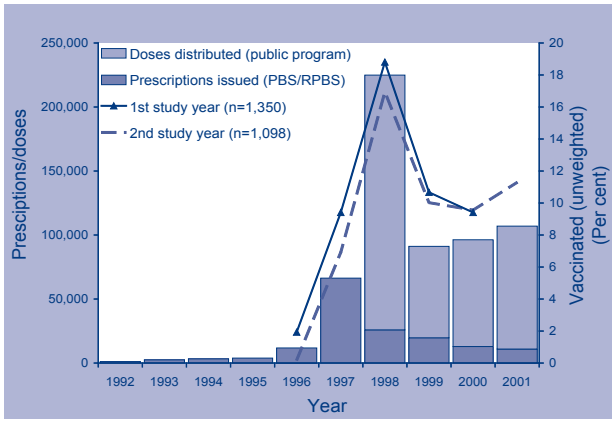


Study year 1 = Subjects discharged between 1 April 2000 and 31 March 2001.

Study year 2 = Subjects discharged between 1 April 2001 and 31 March 2002.

Boxes and vertical lines represent weighted estimate of vaccine coverage with 95 per cent confidence limits.

Figure 4. Comparison of 23vPPV coverage among a cohort of hospitalised persons aged 65 years or more against total doses of 23vPPV available, Victoria, 1992 to 2001, by study year and year of vaccination



Study year 1 = Subjects discharged between 1 April 2000 and 31 March 2001.

Study year 2 = Subjects discharged between 1 April 2001 and 31 March 2002.

PBS (Pharmaceutical Benefits Scheme)/RPBS (Repatriation Pharmaceutical Benefits Scheme)[11]

Doses distributed under Victoria's publicly funded program, i.e. no prescription required, (personal communication, Ted Jamieson, Department of Human Services, Victoria, March 2003).

Discussion

Our study determined influenza and pneumococcal vaccination status using complete vaccination dates from provider records and should therefore be considered minimum coverage estimates among the respondents. Any influenza or pneumococcal vaccinations that were not identified could have only increased coverage among the respondents. We expect that vaccination coverage may be lower among the non-respondents. A similar study among community based elderly in Victoria had a response rate of 72 per cent with the estimated pneumococcal coverage revised from 57.9 per cent (95% CI 52.0–63.6) to 50.5 per cent (44.8–56.1) after accounting for response bias.[8] It should be noted that the response rate in our study was substantially higher with 82.1 per cent of eligible subjects having a known influenza vaccination status and 83.4 per cent having a known pneumococcal vaccination status.

We found influenza vaccine uptake among the respondents had increased by seven percentage points from 68 per cent during the first study year (April 2000–March 2001) to 75 per cent in the second year (April 2001–March 2002). Others have also found evidence of increased influenza coverage in community based surveys in Victoria over a similar time period,[5] although the estimates were higher (78% in 2000 and 81% in 2001) and based on self report. Given that self-reported influenza vaccine status is considered to be reliable,[6] the lower influenza vaccination rates in our study may reflect lower overall coverage among the hospitalised elderly.

Like influenza vaccine, 23vPPV coverage among respondents also increased by seven percentage points between the first study year and the second. The increase was roughly equivalent to the amount of 23vPPV distributed through Victoria's publicly funded program, which may have also influenced the increase in influenza vaccine coverage because persons requiring 23vPPV would almost certainly have been eligible for influenza vaccination. The improvement in coverage appeared to be broad based, with the point estimate increasing across each five-year age stratum over 65 years with the exception of 23vPPV coverage among subjects over 85 years. Our study was limited to a two year observation period but it was encouraging that uptake of both vaccines had increased among the hospitalised elderly in the second year.

Our estimates were consistent with the available doses of 23vPPV each year, the greatest increase in coverage coinciding with the introduction of Victoria's publicly funded program in 1998 when the total number of doses available was greatest. We found the increase in 1998 was consistent for both those subjects discharged during the first study year and those discharged during the second. This is further evidence indicating that the introduction of Victoria's publicly funded program has dramatically increased coverage even though funding has limited the availability of vaccine from year to year.[8,12]

Our results suggest 53 per cent of the hospitalised elderly had received 23vPPV within the five years prior to admission, increasing from 49 per cent in 2000/01 to 56 per cent in 2001/02. In an earlier study, MacIntyre, *et al* reported 23vPPV coverage among a non-random sample of elderly patients at the Royal Melbourne Hospital had increased from four per cent in 1997 to 41 per cent in 1998.[13] As previously noted, a similar population-based survey in Victoria, which also confirmed coverage from provider records, found very similar results to those of our study with 23vPPV coverage among the elderly of 50.5 per cent (95% CI 44.8–56.1) in 2000 after adjusting for response bias.[8] This suggests vaccine coverage among hospitalised patients is similar to but not greater than vaccine coverage in the community. It could be expected that persons regularly admitted to hospital would have more contact with health professionals and therefore be more likely to be vaccinated but we found no evidence of this as demonstrated by the weighted and unweighted coverage estimates being virtually identical.

Victoria's publicly funded 23vPPV program has led to a reduction in the incidence of invasive pneumococcal disease among the elderly in that State.[14] Given that pneumococcal vaccine has been shown to be cost-effective for people aged 65 years or more in other countries[15–17] and is likely to be of similar benefit to influenza vaccine in this age group,[18] our study provides support for the introduction of a fully funded national 23vPPV program for the elderly as announced recently.[2] Our data suggests a national 23vPPV program may also provide further impetus to improve influenza vaccination uptake among the elderly.

The hospitalised elderly are a group at particularly high-risk from influenza and pneumococcal disease. Both vaccines are now available free to all Australians aged 65 years or more. Every opportunity, including hospital admission, should be taken to review vaccination status among this age group and immunise as appropriate.

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Estimates of chronic hepatitis B virus infection in the Northern Territory

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Recent estimates of the prevalence of chronic hepatitis B virus (HBV) infection obtained from the first national serosurvey in Australia in 1996–99 range from 91,500 to 163,500 persons (0.49%–0.87%).[1] A large proportion of these infections is known to occur in selected populations, including Indigenous people. Studies in the 1980s and early 1990s estimated that nearly half of all Indigenous schoolchildren had serological markers of HBV infection.[2,3] A recent report showed that HBV notification and hospitalisation rates in Australia are at least four times higher in Aboriginal and Torres Strait Islander people.[4] The seroprevalence of HBV infection is likely to differ significantly from the national rate in some areas, particularly the Northern Territory, where approximately 25 per cent of the population is Indigenous and universal infant HBV immunisation has been in place since 1990.[5]

The first national serosurvey established baseline seroprevalence of HBV markers for Australia[1] – derived from sera collected opportunistically from laboratories around Australia between July 1996 and May 1999. States and territories were sampled proportionally to their populations,[1] so not all sera collected from Northern Territory laboratories were tested. In the present study, all available sera from the Northern Territory—mainly from Royal Darwin Hospital—were tested for HBV core antibody (HBcAb) (n=150), and HBV surface antibody (HBsAb) (n=161). Sera in which HBcAb was detected were tested for HBV surface antigen (HBsAg). Population prevalence was calculated by weighting the age-specific prevalence estimates by the age distribution of the 1998 Northern Territory population. Table 1 shows that in 1996–1999 the population prevalence of HBsAb was 41 per cent (30.1–51.2%), HBcAb 28 per cent (16.4–39.3%), and HBsAg 0.8 per cent (0–1.7%). These are the first estimates of HBV prevalence since the introduction of universal HBV immunisation in the Northern Territory in 1990. The significantly (0.005) higher prevalence of HBsAb in 1–4-year-olds, compared with the national serosurvey (Table 2) reflects the impact of the Northern Territory immunisation program (a national infant program commenced in 2000, after these sera were collected).

The estimated rate of chronic HBV infection in the Northern Territory (0.8%) was similar to that in the national serosurvey. Although the status of subjects whose sera were collected is not known, it was estimated that approximately 50 per cent would be Indigenous people (personal communication, Dr Gary Lum, Director, Northern Territory Government Pathology Service). Compared with the national serosurvey, there was a higher proportion with evidence of past infection (HBcAb positive) at all ages (Table 2). This was particularly noticeable for children aged under nine years, in whom the proportions were more than 15 times higher in the Northern Territory than nationally, even though hepatitis B infections in this age group are preventable by current vaccination programs. Although the number of sera tested was small and individual clinical data are not available, the prevalence in a random sample of Northern Territory Indigenous children, including those from remote regions, would be likely to be higher. More specific studies are needed to examine the impact of the hepatitis B immunisation program in the Northern Territory in more detail.

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Table 1. Results of the Northern Territory serosurvey for hepatitis B, 1996 to 1999

Age group	HbsAb		HBcAb		HBsAg*		
	Number tested	%pos (95% CI)	Number tested	%pos† (95% CI)	Number tested	%pos‡,§	% pos‡ (adjusted est)‖
1–4	28	71.4 (51.3–86.8)	29	13 (3.9–31.7)	3	3.4	3.6
5–9	21	33.3 (14.6–57)	18	11 (1.4–34.7)	1	0	0
10–14	21	33.3 (14.6 –57)	21	24 (8.2–47.2)	5	0	0
15–19	49	38.8 (25.2 – 53.8)	49	34.7 (21.7–49.6)	14	6.1	6.6
20–39	19	47.4 (24.4 – 71.1)	14	28.5 (8.4–58.1)	2	0	0
Over 40	23	26.1 (10.2 –48.4)	19	36.8 (16.3–61.6)	7	0	0
Total¶	161	40.6 (30.1 –51.2)	150	27.9 (16.4–39.3)	32	0.8	0.8

CI Confidence intervals.

* Testing for HBsAg was restricted to sera positive for HBcAb.

† %positive or weakly positive (n=7) for HbCAb.

‡ %pos (percentage of sera positive for HBsAg) = number of sera positive for HBsAg x 100 ÷ number of sera tested for HBcAb.

§ There were insufficient sera to confirm the HBsAg status of two subjects, (aged 41 and 17 years, both female). These subjects were excluded from the analysis.

‖ Adjusted est = adjusted estimate of prevalence—missing results distributed according to the distribution of known results.

¶ Age group specific prevalence estimates have been weighted by the age distribution of the 1998 Northern Territory population to obtain a population prevalence.

Table 2. Comparison of Australian and Northern Territory serosurvey results for hepatitis B, 1996 to 1999

Age group	HBsAb		HBcAb	
	Australia %pos (95% CI)	Ratio Northern Territory:Australia	Australia %pos (95% CI)	Ratio Northern Territory:Australia
1–4	37.8 (32.9–42.9)	1.9	0.3 (0–1.4)	43.3
5–9	25.2 (21.3–29.3)	1.3	0.6 (0.1–0.7)	18.3
10–14	25.9 (22.0–33.0)	1.3	2.0 (0.4–2.6)	12
15–19	26.8 (22.7–31.2)	1.4	2.9 (1.5–5)	12
20–39	29.9 (24.3–34.7)	1.6	7.9 (1.7–18.2)	3.6
Over 40*	26.8 (21.1–33.1)	0.9	11.1 (5.7–18.4)	3.3
Total†	28.7 (27.0–30.4)	1.4	6.9 (5.4–8.5)	4.1

* National serosurvey results only include those aged 40–59 years, Northern Territory serosurvey results include those age 40–84 years.

† National serosurvey results for the total include those aged 1–59 years, Northern Territory serosurvey results include those aged 1–84 years.

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The incidence of Ross River virus disease in South Australia, 1992 to 2003

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Abstract

Ross River virus (RRV) disease is the most frequently notified arboviral disease in Australia, and the burden of this disease to Australian society is significant. We have studied the incidence of RRV disease between 1992 and 2003 in South Australia. Our findings suggest that the incidence of the disease in South Australia over the study period was relatively stable. There were four epidemics in the study period, with the majority of cases acquired from regions along the River Murray. There was some evidence of spread of the disease to regions in which activity of RRV had not been previously recognised, such as the Mid-North and the South-East. In terms of disease distribution amongst the population, it was found that the highest rates occurred in the 30–49 year age range. There was no significant difference in disease rates between males and females. In order to facilitate further research into RRV disease transmission, we recommend that the suspected region of acquisition be a mandatory component of the national notification dataset. *Commun Dis Intell* 2005;29:291–296.

Keywords: Ross River virus disease, South Australia, epidemiology

Introduction

Ross River virus (RRV) causes a non-fatal disease in humans, and it has been estimated that between 70 and 90 per cent of people infected with RRV have either mild symptoms or no symptoms at all.[1,2] The typical features of RRV disease are joint pain and swelling (mainly in the extremities), lethargy, myalgia, rash (involving the trunk and limbs), fever, headache and depression.[3–6] With thousands of cases occurring in Australia each year, the burden of this disease to Australian society is significant; for example, it has been estimated that the direct and indirect health costs are in the tens of millions of dollars per year, and this is without taking into account the significant but intangible costs of the pain and suffering of the individual cases.[3,7–9]

RRV disease is the most common arboviral disease in Australia, and the virus has been isolated from more than 40 species of mosquito. Being a mosquito-borne disease, the distribution of RRV disease is closely tied to environmental conditions, as the availability of habitat and factors such as rainfall and temperature have a large influence on mosquito populations. The disease is endemic in the tropical regions of Australia, where the climate is conducive to mosquito breeding during the wet season. In the more temperate southern regions of Australia, the disease occurs relatively infrequently outside of epidemics.

In South Australia, the first reported epidemic occurred in 1956, when approximately 200 cases were reported from regions along the River Murray.[10,11] The disease has been notifiable in South Australia since 1980.[5]

The purpose of this study was to describe the incidence and distribution of RRV disease in South Australia for the period July 1992 to June 2003, and so extend the work done by Mudge, Cameron, Weinstein and others, whose descriptions of RRV disease in South Australia cover the period from its first detection in 1956 up to the summer of 1992/93.[5,12,13]

Methods

Data regarding notified cases of RRV disease for the study period were sourced from both the Australian Government and South Australian health departments (see acknowledgements). Data were obtained at the national level so that disease rates in South Australia could be compared with other parts of Australia. This national dataset consisted of the age and sex of the cases, as well as the Statistical Local Area (SLA) of their residence and the date of onset of symptoms. In addition to these fields, the South Australian dataset recorded the SLA where the infection was thought to have been acquired, as recorded on the form completed by the notifying

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medical practitioner. For cases where this suspected region of acquisition had not been recorded, the place of residence was used as a substitute.

The analytical approach applied to these data was the traditional epidemiological method of classifying and comparing cases by time, person and place. Disease rates were derived from 2001 census data (SLA residential populations by age and sex) obtained from the Australian Bureau of Statistics. Electronic maps were produced using a computer software tool ('Csmart') developed by the South Australian Department of Health.

Results

There were 2,294 notifications of RRV disease to the South Australian health department during the study period (Figure 1), with the median annual rate being 3.8 cases per 100,000 population (Figure 2); these were considered confirmed cases on the basis that there was either a fourfold or greater change in serum antibody titres between acute and convalescent-phase serum specimens or there was demonstration of specific IgM antibodies in cerebrospinal fluid (CSF) or acute-phase serum or there was isolation of the virus from blood, CSF or tissue specimens.

Temporal distribution

Figure 1 highlights the four epidemics that occurred during the study period, which accounted for almost 90 per cent of the cases during the study period. The 1992/93 epidemic, with over 800 notifications, was the largest epidemic in South Australia on record.[10,11] Epidemics followed in 1996/97, with over 650 cases notified, and in 1999/00 and 2000/01, with more than 250 cases notified in each. These data suggest a general pattern of epidemics in South Australia every three to four years, and that over the study period the epidemics became smaller in terms of the number of notified cases.

Figure 1. Ross River virus notifications per month, South Australia, July 1992 to June 2003

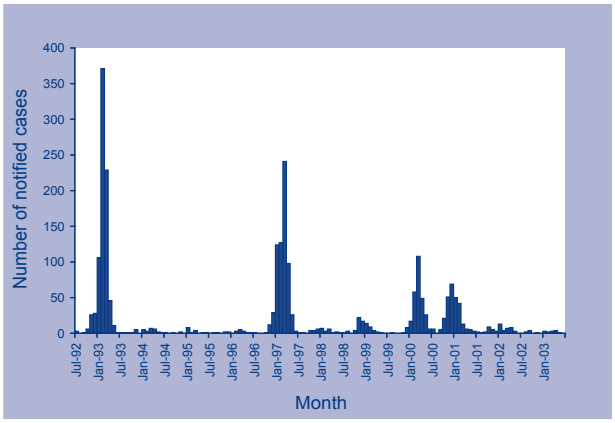
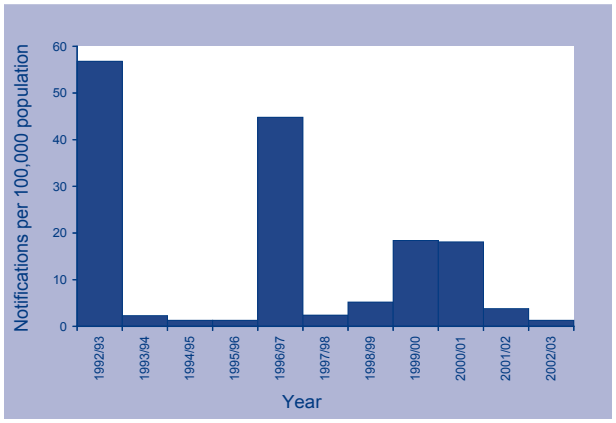


Figure 2. Ross River virus notifications per 100,000 per year, South Australia, July 1992 to June 2003

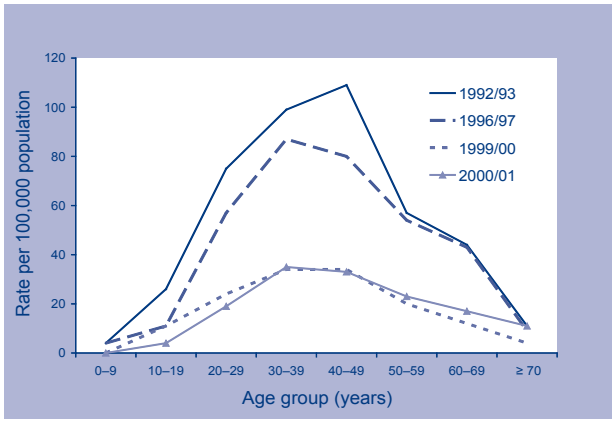


The peak months for the first three epidemics were February and March. For the 2000/01 epidemic however, the peak period was between November and February (Figure 1). Across the entire study period, over half the cases occurred in February and March, and almost 80 per cent occurred in the months January to April.

Age and sex distribution

Figure 3 shows the age-specific rates for each of the four epidemic years. In each of these epidemics the rates in young children, teenagers and people aged over 70 years were relatively low, and the highest rates consistently occurred in the 30–49 years age range. For Australia for the study period, the highest annual rates also occurred in the 30–39 years (38 cases per 100,000 population) and the 40–49 years (37 cases per 100,000 population) age ranges.

Figure 3. Ross River virus age-specific rates per 100,000 per epidemic year, South Australia, July 1992 to June 2003



The male to female ratio for the study period was 1.1:1, which was consistent with an underlying ratio of 1 (χ^2 = 2.26, df = 1, p > 0.1) and with the ratio for Australia for the same period (1:1).

There was no significant difference in the male: female ratio across age groups (Figure 4). The age group with the male to female ratio furthest from one was children aged less than ten years; this age group had a ratio of 1.7:1, but this value was based on only 16 cases.

Geographical distribution

Of the 2,294 South Australian notifications, 208 had a suspected region of acquisition outside of South Australia, and another 32 had neither a region of acquisition nor a place of residence recorded. Of the remaining 2,054 cases, 538 cases had no region of acquisition recorded and a further 155 cases had region of acquisition recorded as 'indeterminate' (e.g. 'Riverlands (indeterminate)', 'Far north (indeterminate)'). For these 693 cases (34% of 2,054), the region of acquisition was set to the place of residence. For the 1,569 cases where the region of acquisition had been recorded, the region of acquisition differed from the region of residence in 647 (41%) cases.

The regions of acquisition most commonly reported were the Riverland (730 cases) and the Murray Mallee (321 cases). The next most prominent regions were the Eyre Peninsula, Adelaide, the Far North and the Flinders Ranges, with 151, 143, 127 and 117 cases respectively (see Figure 5).

Figure 4. Ratio of males to females, South Australia, 1992 to 2003, by age group

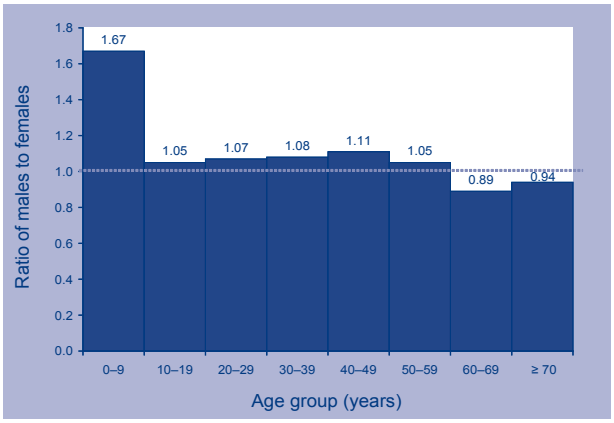
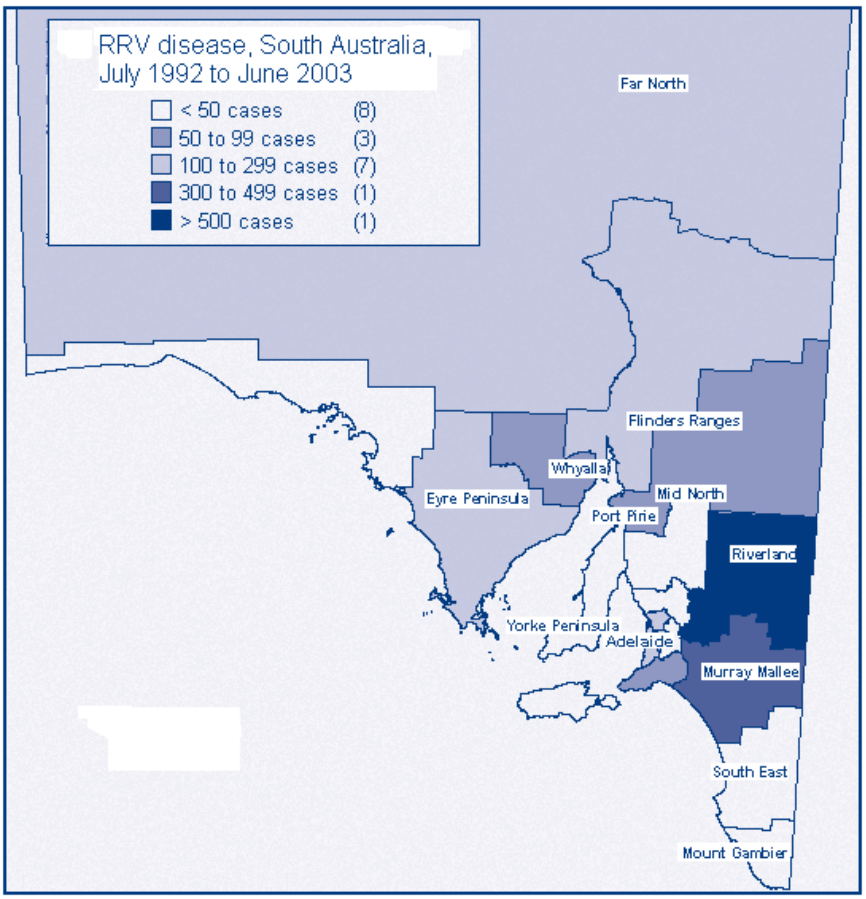


Figure 5. Distribution of Ross River virus cases, South Australia, July 1992 to June 2003, by suspected region of acquisition



Regions along the River Murray were major foci of RRV disease in each of the four epidemic years. In 1992/93, there was also significant activity (at least 10 cases) in coastal regions such as Whyalla, the Lower Yorke Peninsula and the Lower Eyre Peninsula. In 1996/97, the activity tended to be further north, with no cases in either the Lower Yorke Peninsula or the Lower Eyre Peninsula. In 1999/00, activity again tended to be further north, with few cases in the south and again, no cases in either the Lower Yorke Peninsula or the Lower Eyre Peninsula. However, the following year, there were few cases in the northern part of South Australia, many more cases in the southern part, and cases re-appeared in the Yorke Peninsula and the Eyre Peninsula.

Over the study period, cases were acquired from every region in rural South Australia. Compared to the 1992/93 epidemic, the proportion of cases in the Riverland and Murray Mallee regions post-1992/93 dropped from 56 per cent to 42 per cent; in contrast, there was a greater proportion of cases post-1992/93 arising from the Eyre Peninsula, the Flinders Ranges and the Far North (Table).

Table. Percentage of Ross River virus cases, South Australia, 1992/93 and post-1992/93, by suspected region of acquisition

Region	1992/93	1993/94 to 2002/03
Riverland	37	30
Murray Mallee	19	12
Adelaide	12	11
Far North	1	9
Lincoln	5	8
Flinders Ranges	3	7
Whyalla	2	4
Pirie	4	3
Lower South East	1	3
Fleurieu	5	3
West Coast	1	2
Upper South East	3	2
Lower North	3	2
Barossa	2	1
Yorke	3	1
Kangaroo Island	0	1
Onkaparinga	1	0

Discussion

There were three epidemics of RRV disease in South Australia in the decade following the record-level outbreak of 1992/93. The mean number of notifications for these three epidemics was over 400 cases per year, compared to over 800 cases in 1992/93. For the seven non-epidemic years, the average number of cases was less than 40 per year. While the study period was relatively short, the notification data suggest that the incidence of RRV disease in South Australia is not rising. This may not be the case for other parts of Australia, as a number of authors have recently stated that the incidence of RRV disease in Australia is increasing.[3,14]

The number of notified cases is generally considered an under-estimate of the true incidence of RRV disease, i.e. the notification fraction is less than one.[8] The fraction itself is very difficult to precisely estimate, but some researchers have estimated it to be less than 50 per cent.[15] It is therefore always difficult to meaningfully interpret incidence rates which are derived from notification data. Furthermore, the national notification dataset for RRV disease only covers the period since 1991, and so the time-frame of available data may not be sufficient to reveal underlying trends. Prior to 1991, the methods for diagnosis and reporting of RRV disease were less standardised, and so meaningful comparisons would be difficult to make.[8]

Selden and Cameron concluded that in the 1992/93 South Australian epidemic the virus was being acquired in regions well away from the traditional areas along the River Murray, suggesting that the virus was spreading to regions in which activity of RRV had not been previously recognised.[5] The data in this study also suggest that RRV disease has spread into more regions of South Australia over the study period, particularly the northern parts of South Australia.

Of the 68 rural and 56 metropolitan SLAs in South Australia, 53 and 34 of them, respectively, reported cases during the epidemic of 1992/93. Four rural SLAs reported cases for the first time in the epidemic of 1996/97 and four more SLAs reported cases for the first time during the following three years. Of these eight SLAs, three were in the south around Mount Gambier and three were around Port Pirie in the mid-north. Such a finding might be due not only to spread of the virus, but also the result of increased awareness and recognition by medical practitioners, improved laboratory diagnostic methods, and increased encroachment by humans into areas conducive to mosquito breeding, such as wetlands.[7]

In order to study the spread of the virus, it is important that the suspected region of acquisition be collected for all cases. One of the limitations of the national dataset is that the suspected region of acquisition is not routinely collected, and so studies which have utilised these data have generally been required to use the place of residence as a proxy for the region of acquisition. Such approximations may not be very problematic in the endemic, northern regions of Australia, where the region of acquisition is often likely to be the same as the place of residence,

but may be more problematic for a region such as South Australia, where most of the population lives in metropolitan Adelaide where the disease is not endemic, and so for many cases the disease is acquired as a result of travel to an endemic region. It is therefore recommended that the routine collection of suspected region of acquisition be carried out in all Australian states and territories, and that this information then also be recorded at the national level. More precise and complete data regarding the suspected region of acquisition will enable researchers to better understand the geographical distribution of RRV disease.

Epidemics occurred approximately every three to four years, with a large proportion of cases occurring along the River Murray. The distribution of cases away from the River Murray varied in each epidemic, with two epidemics affecting mainly the northern parts of South Australia, and the other two affecting the southern regions. The size of the epidemics, in terms of the number of notified cases, decreased over the study period. This reduction may reflect, at least in part, increasing levels of immunity in the South Australian population, particularly in endemic regions along the River Murray. While it is generally considered that RRV infection confers lifelong immunity, immunity to RRV is not well understood. A general practitioner in Berri (one of the major towns in the Riverland), noted that some patients reported symptoms of RRV disease during epidemics in both 1971 and 1974, suggesting that infection with RRV may lead to only partial immunity in some people.[16,17]

This study showed that the age and sex distribution of RRV disease in South Australia during the study period was similar to that for Australia as a whole. It appears to be a disease primarily of young to middle-aged adults (30–50 years), and the male to female ratio is essentially one to one. The relatively low rates of disease in children and teenagers are thought to be due to a combination of reduced exposure to mosquitoes and a tendency for children to experience either sub-clinical or mild infections. The relatively low rates in those aged 70 or over are thought to be due to a combination of reduced exposure to mosquitoes and increased immunity due to previous infection.[16]

Much remains to be learnt about the incidence and distribution of RRV disease across Australia. More detailed data collection, particularly with regard to the suspected region of acquisition, will assist in the development of interventions aimed at reducing the impact of this significant public health issue.

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Aedes (*Stegomyia*) *albopictus* – a dengue threat for southern Australia?

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Abstract

***Aedes albopictus*, the so-called 'Asian tiger mosquito,' which has invaded areas of the Pacific, the Americas, Africa and Europe, and been intercepted in various Australian seaports in recent years, has now become established on a number of Torres Strait islands in northern Queensland and threatens to invade mainland Australia. As well as being a significant pest with day-biting tendencies, *Ae. albopictus* is a vector of dengue viruses and is capable of transmitting a number of other arboviruses. The species colonises domestic and peri-domestic containers, and can establish in temperate areas with cold winters. According to predictions made using the CSIRO climate matching software CLIMEX,® *Ae. albopictus* could become established elsewhere in Australia, including southern Australia, and lead to these areas becoming receptive to dengue infections—a condition that currently does not exist because the vector *Aedes aegypti* is confined to Queensland and no species in southern Australia is known to be capable of transmitting dengue. *Commun Dis Intell* 2005;29:296–298.**

Keywords: dengue, Aedes albopictus, *Australia, Torres Strait*

Aedes albopictus, the so-called 'Asian tiger mosquito', is indigenous to South East Asia and some islands of the western Pacific and Indian Ocean, but in recent decades has invaded and become established in the eastern Pacific, North and South America, Africa, Europe and the Middle East. Australia has been at risk of invasion as well; between 1997 and 2005 there were at least 28 interceptions of *Ae. albopictus* by the Australian Quarantine Inspection Service and other authorities at Australian international seaports (including Darwin, Cairns, Townsville, Brisbane, Sydney and Melbourne), but diligent surveillance and border control activities have prevented its introduction to, and establishment on, mainland Australia.

The species has been known to be in mainland Papua New Guinea[1] and its southern island of Daru[2] for some years, thus posing a threat to the Torres Strait islands and to mainland Australia through the frequent sea and air travel that occurs in the region, but until 2005 no *Ae. albopictus* activity had been detected in the Australian region. However, in April 2005, mosquito collections on Yorke Island in the eastern Torres Strait were found to include adults of *Ae. albopictus*, although the species had not been recorded on that island in a 2001 survey, or on any other island during surveys associated with dengue activity in the Torres Strait in recent years. Following the Yorke Island discovery, a delimiting survey during April/May 2005 to determine the geographic extent of the infestation in the region revealed the species was established on 10 of the 17 inhabited islands in the Torres Strait but not in any of five communities surveyed on the adjacent mainland (Cape York Peninsula) of Australia.

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Aedes albopictus is an aggressive day-biting pest, able to colonise domestic and peri-domestic natural and artificial container habitats in both tropical and temperate environments, and with desiccation resistant eggs that facilitate its dispersal in portable containers.[3] It has been reported often to be associated with dengue viruses, and it is a capable laboratory vector although its susceptibility to dengue viruses varies among populations.[4,5] It may serve as a maintenance vector of dengue viruses in rural areas of South East Asia, but it has generally been thought to be not an important urban vector when compared with the principal vector species *Aedes aegypti* throughout the range of dengue activity.[6]

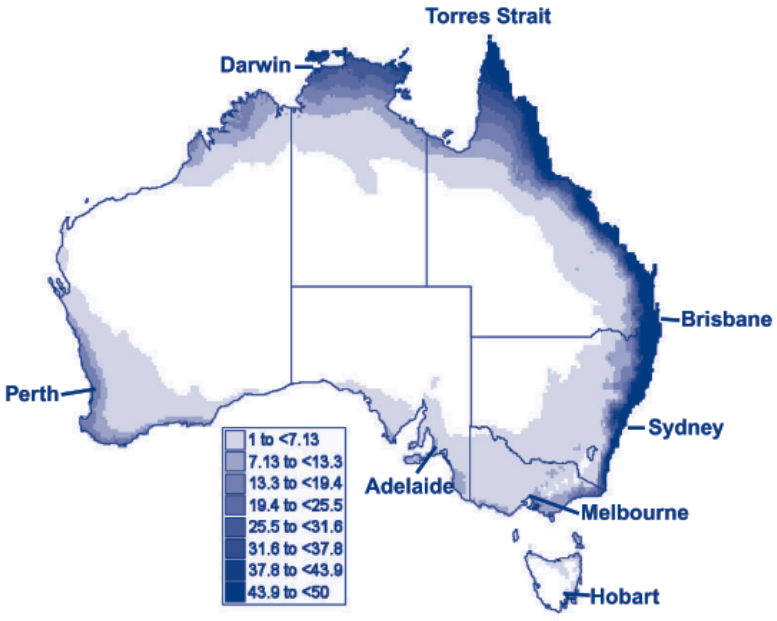
However, historically, *Ae. albopictus* has been responsible for dengue transmission in countries where *Ae. aegypti* was absent, e.g. Japan and parts of China.[6,7,8] More recently, it has been responsible for dengue transmission in an extensive outbreak in the Seychelles in 1976–1977,[9] and reports of significant local dengue infection in Macao in 2001[10] and in Hawaii in 2001[11] have been associated with *Ae. albopictus* on the grounds that it was abundant whereas *Ae. aegypti* appeared to be absent. Also of concern is the fact that as well as the dengue viruses, *Ae. albopictus* is a competent vector under experimental conditions for at least 22 arboviruses, including the internationally important yellow fever virus, the recently introduced to Australia Japanese encephalitis virus, and the Australian local Ross River virus.[8]

In Australia, local transmission of dengue viruses is restricted to Queensland (particularly coastal northern Queensland) where *Ae. aegypti* is found, and other regions currently have no known vectors of dengue. The widespread establishment of *Ae. albopictus* in the Torres Strait provides many and various opportunities for the introduction of the species into mainland Australia. The international distribution of *Ae. albopictus* is limited by factors such as daylength, temperature, rainfall and humidity. Nonetheless, there is evidence that tropical strains of *Ae. albopictus* have adapted to cooler temperate regions when introduced into South America,[12] and temperate strains arguably could persist in southern cool zones of Australia as they have in northern cold regions of North America when introduced from Japan.[3]

The potential distribution of *Ae. albopictus* in mainland Australia was determined using the CLIMEX® model.[13] CLIMEX® infers the response of a species to climate from its known distribution elsewhere and describes the potential for permanent establishment with an Ecoclimatic Index (EI) for each location. The CLIMEX® model of *Ae. albopictus* used by Sutherst[14] was modified to predict the incursion by a tropical strain. The value of the higher optimal temperature parameter, DV2, was increased from 27° C to 30° C and that of the lethal high temperature, DV3, was increased from 30° C to 37° C. The model accurately reproduced the range limits of the species in North America (http://www.cdc.gov/ncidod/dvbid/arbor/albopic_97_sm.htm).

The Figure shows the potential aggregate distribution of both tropical and temperate biotypes of *Ae. albopictus* in Australia, as estimated using CLIMEX.® All of the north, east and south-east coasts as far south as Victoria are suitable for *Ae. albopictus*

Figure. The potential distribution of *Ae. albopictus* as determined by climate in Australia, estimated by the CLIMEX Ecoclimatic Index with a scale from zero for failure to establish up to 100 for optimal for growth throughout the year



The dark shading shows the areas that are most climatically suitable for the mosquito.

establishment, as is the south-west of Western Australia. These areas include most of Australia's major population centres. Marginal populations of *Ae. albopictus* may persist in parts of South Australia, central and western Victoria and Tasmania. The arid interior is estimated to be unsuitable but could support populations in domestic environments where artificial water containers exist. Cold stress was a limiting factor only in Tasmania and the alpine regions of New South Wales and Victoria. Of note is the strong suitability along the eastern coast of Cape York, which could serve as a corridor for the introduction of *Ae. albopictus* from the Torres Strait into the eastern mainland of Australia.

While these coastal locations should be deemed to be receptive to dengue transmission once *Ae. albopictus* became established, their vulnerability will depend upon the likelihood of introduction of dengue viruses with incoming tourists and returning travellers. However, in this respect, all states and most major cities throughout Australia in recent years have reported dengue infections acquired internationally (see Annual Reports of the National Notifiable Diseases Surveillance System), and the arrival of a viraemic traveller in an Australian area with *Ae. albopictus* should be viewed as a public health concern.

The purpose of this report is to quickly notify the Australian health community of the establishment of *Ae. albopictus* in the Torres Strait, and the potential for the species to be introduced to major populations of Australia and provide for the transmission of dengue viruses where currently no vector exists. Details of the initial mosquito collections of *Ae. albopictus* and the delimiting surveys in the Torres Strait and on Cape York, and the genetic associations of the various populations and their epidemiological implications, are the subject of a separate paper being prepared for publication.

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Genetic diversity of the dengue vector *Aedes aegypti* in Australia and implications for future surveillance and mainland incursion monitoring

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Abstract

In February 2004, the discovery of an incursion of the dengue vector *Aedes aegypti* into the town of Tennant Creek in the Northern Territory caused concern for the Northern Territory health authorities who proceeded to implement a Commonwealth-funded eradication program. To determine the origin of the incursion, we performed a genetic analysis on *Ae. aegypti* from several Queensland and overseas localities. A comparison of DNA sequences from the mitochondrial cytochrome oxidase 1 gene indicated that the incursion was probably from Cairns or Camooweal. This genetic marker was also useful in identifying a separate Townsville haplotype population and another population on Thursday Island in the Torres Strait that was genetically divergent to the mainland populations. The possible use of this marker as a surveillance tool for identifying the origins of local and overseas incursions is discussed. *Commun Dis Intell* 2005;29:299–304.

Keywords: Aedes aegypti, *mtDNA, cytochrome oxidase 1 gene, dengue, surveillance*

Introduction

Aedes aegypti is the primary vector of dengue virus. It is the only dengue vector in mainland Australia and has been responsible for outbreaks of dengue fever that reappeared in northern Queensland in the early 1980s and have continued until the present.[1,2] Historically, the distribution of *Ae. aegypti* included all mainland states and territories except Victoria and South Australia. However, in the 1950s it disappeared from Western Australia, New South Wales and the Northern Territory.[3] It maintains a strong hold in Queensland where its southern limit is Dirranbandi to Roma and west to Cloncurry and Mount Isa.[4] In February 2004, specimens of *Ae*. *aegypti* were identified in Tennant Creek in the Northern Territory.[5] This town is located on the main road links to Queensland (via the Barkly Highway) and Darwin (via the Stuart Highway) and is 670 km from Mount Isa—the nearest previously known source of *Ae*. *aegypti*.

Apart from the potential for this species to spread from Queensland into other states or territories, there is the continual threat of its introduction to Australia from overseas via international ports. Darwin alone had 13 importations of *Ae. aegypti* between 1998–2000,[6] and there have been numerous other detections by the Australian Quarantine Inspection Service (AQIS) since then, including the recent detection of an importation in February 2005 from an Indonesian fishing vessel (Whelan, unpublished data). *Aedes aegypti* is a competent traveller with three attributes that contribute to its dispersal: 1) it has a very close association with humans; 2) it readily breeds in artificial receptacles; and 3) its eggs can withstand desiccation for many months.

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The movement of this species, either within or from outside Australia, is of great concern to public health authorities and AQIS. From a surveillance and control perspective, it would be useful to know if the recent infestation at Tennant Creek originated from Queensland, or from Darwin after being imported from overseas. If it is the former, then inspections of towns along the main road, working back to Mount Isa, as the nearest probable source, will be required. If the latter, then increased surveillance and trapping in the towns from Darwin to Tennant Creek will be required. With incursions from outside of Australia, it would be relevant to know in which country the strain originated, as different geographic strains can have different colonising abilities and different competencies with regards to transmitting the dengue virus.[7–9] This situation is complicated by the fact that vessels coming to Australia may have stopped at several Asian ports where *Ae. aegypti* is endemic.

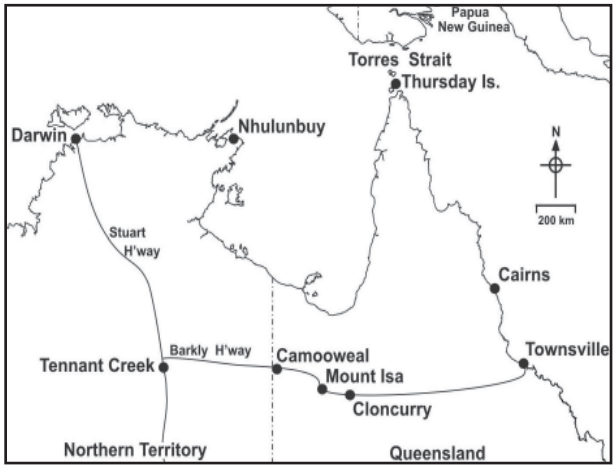
Identifying differences in mosquito strains or populations requires a DNA-based genetic marker that will be informative, will deliver an unambiguous result, will be relatively straightforward to use, and ideally, be useful in later studies of evolution or population genetics. As *Ae. aegypti* is an exotic mosquito that probably arrived in Australia during the mid-19th century,[10] a rapidly evolving genetic marker would be required to identify population variation within this species. Genetic markers based on the mitochondrial DNA (mtDNA) have been to be useful for genetic studies of other species and populations.[11,12]

The aim of this study was to assess the use of the mtDNA cytochrome oxidase 1 (CO1) gene as a genetic marker to evaluate the origin of the *Ae. aegypti* incursion into Tennant Creek. We also evaluated this marker as a potential surveillance tool for identifying populations of *Ae. aegypti* that originated from locations outside of Australia.

Method

Australian specimens of *Aedes aegypti* were collected as larvae from three different breeding sites in Tennant Creek in the Northern Territory, and from breeding sites in Cairns, Townsville and Thursday Island in Queensland. Following the discovery of *Ae. aegypti* in Tennant Creek, a container breeding survey was conducted at Camooweal located on the Barkly Highway at the Queensland-Northern Territory border, 188 km west of Mount Isa. Specimens collected during this survey were also included in this study. Specimens were also obtained from an Indonesian fishing vessel that was intercepted and inspected by AQIS approximately 1.5 km outside Melville Bay near Nhulunbuy on the north-east coast of the Northern Territory in February 2005. The ship contained *Ae. aegypti* larvae and pupal skins categorising it as a risk importation that had a potential for live adults to disperse to shore, had it not been intercepted and appropriately treated. Collection sites from within Australia are indicated in Figure 1. Specimens, collected as immature stages or from established colony material, from outside Australia were obtained from South East Asia and the south-west Pacific: Burma, Viet Nam, Thailand, Timor Leste, Papua New Guinea (PNG) and Vanuatu.

Figure 1. Northern Australia indicating *Aedes aegypti* collection sites



Mosquito DNA extraction, polymerase chain reaction amplification and DNA sequencing

Mosquitoes (partial or whole adults and larvae) were thoroughly ground in a 1.5 ml microfuge tube containing 50 µl of lysis buffer (1.0M NaCl, 0.2M sucrose, 0.1M Tris-HCl (pH 9.0), 0.05M EDTA and 0.5% SDS). Tubes were pulse microfuged to concentrate the homogenate in the bottom of the tube prior to incubation at 65° C for 30 minutes. Then 7 µl of 8.0M KAc was added to each tube; these were mixed, placed on ice for 15–30 minutes and microfuged for 15 minutes at 14,000 rpm. Supernatants were placed in a new tube to which 100 µl of 100 per cent EtOH was added and microfuged at 14,000 rpm for 15 minutes. Supernatants were removed, 100 µl of 70 per cent EtOH was added, and tubes were centrifuged again at 14,000 rpm for 5 minutes. Supernatants were again removed, tubes were air dried and resuspended in 50 µl TE containing RNase (5 µg/ml).

A 5' segment of the mtDNA CO1 gene was amplified in 25 µl volumes using a thermal cycler (DNA Engine, MJ Research Inc.). The forward primer (5'-TAGTTC CTTTAATATTAGGAGC-3') was designed to start approximately 245 bp into the CO1 5' region and the reverse primer (5'-TAATATAGCATAAATTATTCC-3') was designed back from 813 bp into the CO1 gene. The final polymerase chain reaction (PCR) mixture contained 1x *Taq* buffer II (Fisher Biotech Australia), 2.5 mM MgCl, 0.125 mM of each dNTP, 0.4 µM of

each primer, 0.5–1.0 unit of *Taq* polymerase and 5.0–10.0 ng of extracted genomic DNA (1 µl of extraction). The cycling involved an initial denaturation of 94° C for three minutes, then 35 cycles of 94° C for one minute, 50° C for one minute and 72° C for one minute with minimal transition times. The PCR products were separated by agarose gel electrophoresis (1.0%) at 100 V for 40 minutes, then visualised by staining with ethidium bromide (0.3 µg/ml) at 312 nm.

DNA sequencing and genetic analysis

Amplified products were purified using the Qiagen QIAquick PCR purification kit following their set protocol. Sequencing was performed using an ABI Big Dye™ Terminator kit (PE Biosystems) according to the manufacturer's recommendations and the same forward and reverse primers described above were used for sequencing.

The sequence alignment was performed using the PILEUP algorithm in the GCG package using default settings (Genetics Computer Group, Version 8, 1994). Genetic analyses using traditional tree-building phylogenetic methods can be inappropriate for these types of studies because they make assumptions that are invalid at the intraspecific population level.[13] Thus the analysis was performed using the TCS algorithm which estimates genealogical relationships and generates a parsimonious network.[13]

Results

Aedes aegypti genomic DNA was extracted from 46 individual specimens from Australia and various countries of South East Asia and the south-west Pacific. From these, 46 CO1 sequences were derived and aligned together and with two other *Ae. aegypti* sequences (laboratory strains originating from East and West Africa) obtained from Genbank (Table). After editing, the sequence alignment length was 503 bp and showed eight separate sequence haplotypes. All nucleotide changes occur at the third codon position. A summary of the DNA sequence variation for each haplotype (relative to haplotype 1: Tennant Creek and Cairns population, Genbank accession DQ026284) is presented in the Table along with the haplotype distributions and their frequency. Figure 2 shows a minimum parsimony network of the eight haplotypes.

The CO1 haplotypes obtained from the three separate breeding sites in Tennant Creek were the same as those found in Cairns but different to those identified from Townsville. It appears that the Tennant Creek population represents a single haplotype population (H1). The H1 haplotype from Cairns appears well dispersed as it was also found from mosquitoes collected in Viet Nam and Thailand. Haplotype H1 is one mutational step (1 nucleotide) from another well-dispersed haplotype H4, which was found in

Table. Collection sites, haplotype distribution and haplotype diversity of *Aedes aegypti* populations used in this study

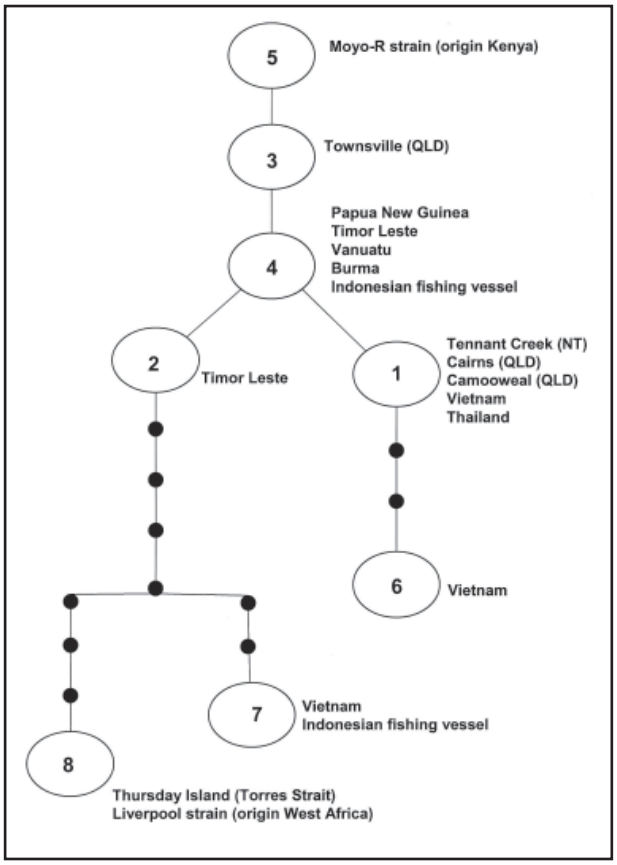
Collection site	n	CO1 haplotype	Haplotype diversity*
Cairns Qld (2 sites)†	5	H1	bp
Townsville Qld†	3	H3	112222333333344
Tennant Creek NT (3 sites)†	9	H1	46890129044568903
Camooweal Qld†	2	H1	56657984625737928
Thursday Is. Torres Strait†	3	H8	H1 GTAAACTAGTTATCACA
Indonesian fishing vessel†	5	3xH7, 2xH4	H2 .....C....
Timor Leste (3 sites)†	7	H2	H3 A.....
Thailand (Bangkok)‡	2	H1	H4 .....T.....
Viet Nam (Hanoi)‡	3	H1, H7, H6	H5 A.....C.....
Burma‡	3	H4	H6 .G...C...C.....T.
Vanuatu†	1	H4	H7 ......C.A.CGCTG.G
Papua New Guinea†	3	H4	H8 ..GGG.C.A..GCT...
MOYO-R strain (Af380835)‡	–	H5	
Liverpool strain (AY056596)‡	–	H8	

* Nucleotide changes relative to H1 (Genbank accession number DQ026284).

† Specimens collected as immature stages from breeding sites.

‡ Specimens from established colonies.

Figure 2. Mitochondrial CO1 haplotype network showing genealogical relationships



Legend: Circles represent the different CO1 sequence haplotypes with geographic regions of specimens listed. Connecting nodes represent single mutational steps between haplotypes and may be unidentified extant haplotypes.

PNG, Timor Leste, Burma, Viet Nam and Vanuatu. Haplotype H3, identified from Townsville, is also a single mutational step from the H4 haplotype, but H3 appears restricted to Townsville. The specimens from Thursday Island were all H8 and the same sequence as the Liverpool laboratory strain that was originally collected from West Africa. This Thursday Island material was considered quite divergent to the Australian mainland material with 10 mutational steps to either H1 or H3. Analysis of five specimens collected in February 2005 from the Indonesian fishing vessel revealed two separate CO1 haplotypes – three H7, and two H4 individuals. The H4 haplotype was found to be widespread, as mentioned above, while the three H7 haplotypes showed the same sequence as one specimen from Viet Nam.

Discussion

The mitochondrial DNA was selected for this study because its genome is maternally inherited through the female egg and very rarely undergoes recombination.[11] Thus it has a more linear or clonal evolution than nuclear DNA and its coding genes also display a more rapid rate of evolution, making it a useful marker for studying intraspecific population genetic variation.[11,12] The CO1 gene has been found useful for intraspecific studies of *Anopheles* and in interspecific studies of *Aedes* mosquitoes,[14,15] and for both *Anopheles* and *Culex* mosquitoes in our laboratory (Beebe, unpublished data).

We reveal for the first time that there are at least two mtDNA CO1 haplotype populations of *Ae. aegypti* on the Australian mainland (H1 from Tennant Creek, Cairns and Camooweal, and H3 from Townsville). This study suggests that the incursion into Tennant Creek was not from the military and industrial centre of Townsville, but from Cairns or Camooweal. The most likely spread was by the carriage of eggs in dry receptacles by vehicle traffic. The presence of *Ae. aegypti* at Camooweal moves the western distribution of *Ae*. *aegypti* in Queensland to the Northern Territory border. However, these conclusions should be viewed with caution as further sampling and analysis of sites within these towns will be required to determine if additional haplotypes are present.

Within Australia, the haplotype population identified on Thursday Island in the Torres Strait (H8), shows considerable genetic distance to the Australian mainland haplotypes (10 mutational steps). It is interesting to note that *Ae. aegypti* populations from Thursday Island have displayed enhanced vector competence to the dengue 2 and 4 serotypes compared to the mainland populations from Cairns and Townsville.[9] The substantial genetic distinction between the Thursday Island H8 population and the mainland Australia H1 and H3 populations may help in the understanding of the observed difference in vector competence between these different populations. It also highlights the need for state authorities and AQIS to prevent the movement of *Ae. aegypti* from the Torres Strait to mainland Australia.

Specimens of *Ae. aegypti* collected from the Indonesian fishing vessel revealed two separate haplotypes (H4 and H7). The maternal inheritance of the mitochondrial genome means that each female mosquito will only produce her own haplotype,[11] and indicates that at least two separate egg batches were laid in the receptacle on this vessel by different CO1 haplotype *Ae. aegypti* females. The origin of these haplotype populations could not be determined, as we have no samples from Indonesia for comparison. However, it is likely that these haplotypes represent Indonesian populations of *Ae*. *aegypti*.

The appearance of a divergent haplotype or lineage in the Torres Strait population may reflect the successful dispersal capabilities of this species. No one has looked at the movement of these haplotypes on a global scale. However such movement appears to be considerable, this small study has revealed, for

example, that haplotypes are shared by populations as widely dispersed as Burma and Vanuatu (H4) and Viet Nam and Australia (H1).

Each node in the network in Figure 2 may represent an extant haplotype sequence, and this study suggests that there could be 11 unidentified haplotypes that exist within this network. If we view this haplotype network, bearing in mind it is a small sampling regime, haplotypes H1 and H4 were found most frequently, were well dispersed geographically and appear embedded within the haplotype network. These factors suggest H1 and H4 may be the original (ancestral) haplotypes introduced into the Asia-Pacific region.[16] It is also interesting that the laboratory strains found in Genbank that had origins in West Africa (H8, Liverpool) and in East Africa (H5, Moyo-R, Kenya) are at the ends or tips of the network. Their positioning may indicate the breadth of genetic diversity of this species within Africa.

The dispersal and colonising ability of this species makes it a continual threat to ports in Australia and highlights the need to prevent the further westward spread from Queensland into the Northern Territory and Western Australia. We suggest it should now be a priority to screen *Ae. aegypti* populations in Australia and around our region to record and monitor the possible spread of the endemic and exotic genetic diversity of this species.

In summary, the partial sequence of the mtDNA CO1 gene from a small number of *Ae. aegypti* has enabled the identification of different genetic populations within Australia, as well as the origin of an incursion into the Northern Territory from Queensland. There was also considerable genetic difference between the mainland Australian and Thursday Island populations, which have been shown to display different vector competencies to dengue viruses.[9] Though further extensive sampling and analysis will be required to verify the robustness of this potentially useful genetic marker, this study suggests that the CO1 gene will be a practical tool to study the genetic diversity and spread of *Ae. aegypti* in Australia, as well as to monitor foreign incursions. It has a potential application in studying other species of quarantine and public health importance in Australasia such as the recent establishment of *Ochlerotatus camptorhynchus* in New Zealand, or the dispersal of *Aedes albopictus* into the Torres Strait and other areas of northern Australia.

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Erratum

The report Invasive pneumococcal disease in Australia, 2003 published in *Communicable Diseases Intelligence* 2004;28:441 contains a number of errors.

The rates shown in the map ‘Notification rates of invasive pneumococcal disease, Australia, 2003 by statistical division of residence’ were incorrect.

Table 16: ‘Details of cases of invasive pneumococcal disease that occurred in those fully vaccinated for age with 23-valent pneumococcal vaccine, by jurisdiction, Australia, 2003’ contains incorrect data for New South Wales and Victoria and the totals are consequently incorrect.

A revised version of the report with correct map and Table 16 are available on the CDA website in HTML and PDF formats.

Septicaemia secondary to *Vibrio vulnificus* cellulitis

Peter R Lewis,[1] Lucy Cook,[2] Janet Drewitt-Smith,[3] Adam D McEwen,[4] Linda V Granger[5]

Abstract

Vibrio vulnificus is a naturally occurring, salt-water bacteria found in estuarine and coastal waters worldwide. It prefers low salinity and warm water temperatures for optimum growth. Infection from _Vibrio vulnificus_ is uncommon, although it has been reported from many locations (e.g. southern United States of America, Israel, Republic of Korea, Japan, Taiwan, Spain, Turkey). It can be serious and life threatening, causing septicaemia and wound infections. This paper reports a case of septicaemia secondary to _Vibrio vulnificus_ cellulitis in an elderly woman. The infection was acquired after wading in a coastal lagoon with a pre-existing superficial leg wound. _Commun Dis Intell_ 2005;29:305–307.

Keywords: Vibrio vulnificus, wound infection, water-borne infection, secondary septicaemia

Introduction

Vibrio vulnificus is a gram-negative bacillus and part of normal marine flora in estuarine and coastal waters worldwide.[1] It has been isolated in waters of low to moderate salinity i.e. 5–25 parts per thousands (ppt)[2,3] and in water temperatures of 9–31° C. *Vibrio vulnificus* is also found in sediment, and filter feeding shellfish such as oysters, mussels, clams, and scallops, and fish that inhabit coastal oyster reefs.[4]

V. vulnificus illness has been reported worldwide and usually occurs in the warmer months. Gastroenteritis associated with ingestion of uncooked seafood (particularly oysters) contaminated with *V. vulnificus* is rarely reported. However, primary septicaemia may occur in those with chronic liver disease, haemochromatosis, or immune disorders. The case fatality rate is 50 per cent, increasing to 90 per cent in those with hypotension.[1] This clinical syndrome includes fever, chills, hypotension, shock, and metastatic necrotizing cutaneous lesions. Thrombocytopaenia and disseminated intravascular coagulation are common complications. In otherwise healthy people, exposure of superficial wounds to water where the organism is present can result in local wound infection that may progress to cellulitis, necrotizing fasciitis and secondary septicaemia.[1,5] The case fatality rate ranges from 20–30 per cent for *V. vulnificus* wound infections.[4]

Case report

An 83-year-old female had been wading in a coastal lagoon with a pre-existing abrasion on her left lower leg. Two days later she presented to her general practitioner with fever (axillary temperature 39.6° C), low abdominal pain, and extreme pain in her left lower leg. The area of abrasion had a motley dark appearance. She was subsequently referred to hospital. Prior to this illness the patient was well, active and independent with no major health issues other than asthma, for which she used a Budesonide inhaler. She had no known history of liver disease or immunosuppression. She did not eat any fresh oysters or seafood leading up to her illness.

On admission to the emergency department the patient was febrile, with a history of rigors, nausea, vomiting, malaise and abdominal pain. She was alert, orientated, and normotensive. Initial treatment for cellulitis included intravenous fluids, penicillin, flucloxicillin, and analgesia. Biochemistry and haematology results were normal (white cell count 10.7 x 10^9/L; normal range 4.0 – 11.0 x 10^9/L) except for neutrophil count 9.6 x 10^9/L (2.0 – 8.0 x 10^9/L); lymphocytes 0.1 x 10^9/L (1.0 – 4.0 x 10^9/L); monocytes 0.9 x 10^9/L (0.2 – 0.8 x 10^9/L) and C-reactive protein 8.5 mg/L (< 5.0 mg/L).

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The following morning the patient's condition deteriorated to septic shock and acute renal failure, requiring intensive care admission and inotropic support. She was profoundly hypotensive, tachycardic and oliguric, despite overnight administration of intravenous fluids and frusemide. Her left calf was warm, swollen and erythematous. Abnormal biochemistry and haematology at this time included: urea 10.6 mmol/L (2.5–6.4 mmol/L); creatinine 0.24 mmol/L (0.06–0.12 mmol/L); bicarbonate 14 mmol/L (21–31 mmol/L); protein 55 g/L (64–82 g/L); albumin 25 g/L (34–50 g/L); alkaline phosphatase 37 IU/L (50–136 IU/L); white cell count 11.1 x 10^9/L; neutrophils 8.2 x 10^9/L; troponin 0.22 ng/mL (0.0–0.05 ng/mL). Coagulation studies were also slightly raised; prothrombin time 20 seconds (normal range; 9–13 seconds); activated partial thromboplastin time; 36 seconds (25–35 seconds); International normalised ratio (INR) 1.8 ratio (1.0–1.3). Hypoxaemia and metabolic acidosis had also developed, and subsequent pathology results deteriorated further. Penicillin was ceased; ceftriaxone and gentamycin added. Debridement of the patient's leg wound was performed on days 2 and 3, after which she required inotropic and ventilatory support for several days.

The patient's antibiotic regime was reviewed as microbiology results emerged. Methicillin-resistant staphylococcus (not an endemic hospital strain) and gram-negative bacilli were cultured from the wound site requiring a change from flucloxicillin to vancomycin. *V. vulnificus* was later identified from admission blood cultures and gentamycin was then swapped for doxycycline.

Her renal function and haemodynamic status gradually improved, and a split skin graft was performed on day 17. The following day she was transferred to a ward. Wound swabs were clear and antibiotics were ceased. Four weeks after admission she was transferred to a private hospital where she continued to convalesce with very restricted mobility. She was eventually discharged after six weeks hospitalisation.

Environmental investigation

The lagoon where the patient went wading (lagoon 1) is one of three distinct coastal lagoons, and is separated from the ocean by a sandbar. Water samples were taken from each of the lagoons and adjacent beaches for bacterial analysis and salinity testing (Table). *V. vulnificus* was isolated from two lagoons, but was not detected in any of the beach samples. All of the beach samples revealed a salinity level of 36.1 parts per thousand (ppt), normal for seawater. Water temperatures recorded for lagoon one fluctuated between 24–28º C at the time of the patient's exposure.

Other *Vibrio* infections

The patient's general practitioner also diagnosed a number of other otitis externa infections around the same time. It is possible that these infections were as a result of swimming in the same lagoon. In one case, *Vibrio* species was cultured from a swab taken (species not identified) when a 14-year-old male presented with an ear infection. Treatment with Augmentin forté and Ciproxin ear drops resulted in a complete recovery.

Communicable disease control significance

This case study highlights the need to consider *V. vulnificus* infection in a differential diagnosis for wound infection, particularly when recreational water activities coincide with growth of the bacteria during the summer months. *V. vulnificus* infection is also potentially life-threatening for people with pre-existing liver disease and immune disorders. This group may benefit from preventative advice regarding consumption of raw seafood and contact with seawater in the summer months. Rapid progression and severity of disease makes early diagnosis and treatment of *V. vulnificus* infection crucial for a positive outcome. This infection is not consistently

Table. Chemical and microbiological analysis

Lagoon source	Salinity (parts per thousand)	Sampling point	Faecal coliforms (per 100 ml)	*Escherichia coli* (per 100 ml)	*Vibrio vulnificus* (per 200 ml)
*1	12.6	shallow	24	24	not detected
		deep	22	18	detected
2	16.8	shallow	150	150	detected
		deep	180	180	detected
3	18.2	shallow	96	96	not detected
		deep	8	8	not detected

Source: NSW Health – Division of Analytical Laboratories.

* Patient's wading lagoon.

susceptible to aminoglycosides as are other more common aerobic gram-negative bacilli.[5] Appropriate antimicrobials include doxycycline, cefotaxime, ceftriaxone, ciprofloxacin or minocycline if *V. vulnificus* infection is suspected.[6]

The environmental investigation confirmed the presence of *V. vulnificus* in local recreational waters with low salinity. It is likely that this bacterium is present during most summers with high water temperatures. It is difficult to quantify the health risk posed by these findings. There is no specific ICD –10 code (International Classification of Diseases – 10th Revision) to allow rapid searching of health databases (in-patient statistics; mortality data). Our local pathology provider upgraded their information system three years ago; there were no other isolates of *V. vulnificus* in the last three years. Intensive care clinical staff recalled a similar case about 10 years ago. It is equally challenging to communicate a life threatening health risk that is a rare event to a local community that generates income and pleasure from its environment.

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OzFoodNet: enhancing foodborne disease surveillance across Australia: Quarterly report, April to June 2005

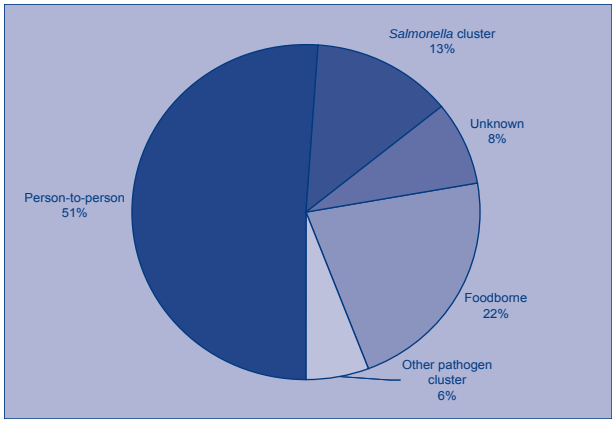
Introduction

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 report summarises the occurrence of foodborne disease outbreaks and cluster investigations between 1 April and 30 June 2005. Data were received from OzFoodNet representatives in all Australian states and territories and a sentinel site in the Hunter/New England region of New South Wales. The data in this report are provisional and subject to change, as results of outbreak investigations can take months to finalise. We would like to thank the investigators in the public health units and state and territory departments of health as well as public health laboratories and local government environmental health officers who collected data used in this report.

During the second quarter of 2005, OzFoodNet sites reported 123 outbreaks of foodborne or enteric illness. Outbreaks of gastroenteritis are often not reported to health agencies or the reports are delayed, meaning that these figures significantly under-represent the true burden of these infections. In total, these outbreaks affected more than 1,661 people and resulted in 64 persons being admitted to hospital. No deaths were reported. As has been the case in previous reports, the majority (51%, n=63) of outbreaks resulted from infections suspected to be spread by person-to-person transmission (Figure). Twenty-seven per cent of these person-to-person outbreaks occurred in aged care facilities, 21 per cent in child care centres and 16 per cent in the community.

Figure. Mode of transmission for outbreaks of gastrointestinal illness reported by OzFoodNet sites, 1 April to 30 June 2005



Foodborne disease outbreaks

There were 27 outbreaks of illness where consumption of contaminated food was suspected or proven to be the primary mode of transmission. These outbreaks affected 327 people. This compares with 37 outbreaks for the second quarter of 2004 and 31 outbreaks in the first quarter of 2005.

Salmonella Typhimurium was responsible for six outbreaks and *Campylobacter* for two outbreaks. *Staphylococcus aureus* was confirmed as responsible for one outbreak and suspected to be the cause of another. The remaining two outbreaks, where an agent was identified, were caused by ciguatoxin and Norovirus. No aetiological agent was identified for the remaining 58 per cent (15/26) of outbreaks.

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

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

Nine of the outbreaks reported in the quarter were associated with meals served in restaurants, five with food prepared in private homes and five with food prepared by commercial caterers. Of the outbreaks caused by food prepared by commercial caterers, two occurred at functions and single outbreaks occurred at a camp, a hospital and in a private home. Food from bakeries and nationally franchised fast food restaurants were responsible for two outbreaks each. Single outbreaks were associated with food prepared at a hospital, a takeaway store, a delicatessen and an unknown setting. Nine of the outbreaks occurred in April, eleven in May and seven in June.

To investigate these outbreaks, sites conducted nine cohort studies and two case control studies. For 16 outbreaks, only descriptive data were collected. Investigators obtained microbiological evidence linking a food vehicle to illness in three outbreaks and analytical epidemiological evidence in six outbreaks. For the remaining outbreaks, investigators obtained descriptive epidemiological evidence implicating the food vehicle or suggesting foodborne transmission.

In New South Wales there were 12 outbreaks of foodborne illness reported during the quarter. Two outbreaks of *Salmonella* Typhimurium 9 were associated with a larger cluster investigated in May and June. One outbreak involved 24 cases who ate Vietnamese pork and chicken rolls from three different bakeries in Sydney. The second outbreak was associated with a meal at a restaurant where three unrelated groups dined on the same evening, resulting in five microbiologically confirmed and three epidemiologically linked cases. No food vehicle was identified for this outbreak, although the restaurant used an egg supplier that was common to two of the bakeries in the first outbreak. The New South Wales Food Authority traced the supply of eggs back to a single farm and tested samples of egg washings and chicken litter, which were positive for *Salmonella* Typhimurium 9.

In the other 10 outbreaks reported by New South Wales, no aetiological agent was identified although three outbreaks were suspected to be caused by viruses. These were likely to be due to person-to-food-to-person transmission caused by ill food handlers or patrons. Four of these involved restaurants. Three of these followed the consumption of meals of chicken salad, lamb and beef dishes and chicken schnitzel. The food vehicle was unknown in the other one. These restaurant-related outbreaks affected between two and 12 people each. Two of the six remaining outbreaks of unknown aetiology occurred following the consumption of hamburgers from a nationally franchised fast food outlet (2 cases each). A further two outbreaks involved food prepared by commercial caterers. In one of these the food was provided to a hospital (11 cases) but the responsible food vehicle was not identified. In the other (28 cases), the food was consumed at a private residence and a potato bake was suspected to be the cause of illness. The remaining two outbreaks occurred in private residences and both involved chicken dishes, one prepared in the home (2 cases) and the other prepared elsewhere (3 cases).

Victoria reported two outbreaks of foodborne disease for the quarter. An aetiological agent was not identified for either outbreak. One outbreak affecting 17 people was associated with a meal of pork and gravy prepared by a commercial caterer. Cases showed symptoms consistent with Norovirus. The second outbreak affected 11 people in an aged care facility who showed symptoms consistent with *Clostridium perfringens* infection. Three faecal specimens tested showed heavy growth of *C. perfringens* while a fourth showed medium growth. Food for the facility was prepared by a hospital but the responsible food vehicle could not be identified.

Queensland reported six outbreaks of foodborne illness for the second quarter. One outbreak was due to ciguatera fish poisoning caused by Spanish mackerel caught off Hervey Bay in Northern Queensland. The fish were distributed to five retailers. Seventeen people, in five unrelated groups, were affected after preparing and consuming the fish at private residences and two people were hospitalised.

Queensland also reported two outbreaks of *Salmonella* Typhimurium. Fourteen people were ill with *S.* Typhimurium 197 after consuming egg based products purchased at a range of outlets but prepared by a single bakery. As part of a *S.* Typhimurium 108/170 cluster investigation, two people reported ill after eating takeaway chicken traced back to a New South Wales poultry supplier linked to another *S.* Typhimurium 108/170 outbreak in New South Wales.

In other Queensland outbreaks, two people became ill after eating custard filled dumplings purchased from a grocery store. *Staphylococcus aureus* was isolated from the dumplings and from faecal samples. One faecal specimen was positive for staphylococcal enterotoxin. No food vehicle was identified in the remaining two outbreak investigations where *Campylobacter jejuni* infected five people after a common meal at a private residence and an undetermined pathogen infected 11 people following consumption of food from a commercial caterer.

South Australia reported that 81 people were infected with *Salmonella* Typhimurium 64 after eating bread, rolls and baguettes with various fillings. A café prepared the rolls over a five day period for six different functions. People eating chicken rolls and hamburgers purchased directly from the café also became ill. Both a chicken roll and raw chicken obtained from the café tested positive for *S.* Typhimurium 64. Trace back identified that a Victorian chicken processor supplied the chicken.

South Australia also reported an outbreak of *Salmonella* Typhimurium 108/170 in which nine people became ill after eating at the same restaurant over a three day period. A case control study identified that the food vehicle responsible for the illness to be marinated chicken roll and the chicken meat was traced to a Victorian chicken processor.

There were two foodborne outbreaks reported by Western Australia for the quarter. Neither the aetiological agent nor the food vehicle responsible were identified in these outbreaks. A commercial caterer supplied food to a camp, where 20 people became ill with gastroenteritis. In the second outbreak 17 people at a work function became ill after consuming food prepared in private homes.

The Northern Territory reported an outbreak following the consumption of Vietnamese pork rolls associated with a stall at a market. Environmental investigations suggest a possible hygiene break-down during the food preparation at a private residence prior to market or inadequate heating of the pork in the bain-marie at the market stall as potential causes. The causative agent was not identified, although five cases presented to hospital emergency departments with symptoms consistent with *Staphylococcus aureus* intoxication.

The Australian Capital Territory reported two foodborne outbreaks. One outbreak affecting 11 people was due to *Campylobacter* infection following the consumption of warm chicken salad and chicken and mushroom pasta served at a restaurant. In the second outbreak, at least 35 people became ill with norovirus infection following a function catered for by a restaurant. Those ill were more likely to have eaten duck and quince tartlets or roast pork on bruschetta.

Tasmania did not report any foodborne outbreaks during the quarter.

Comments

During the second quarter of 2005, contaminated eggs were suspected as the cause of three outbreaks. Two of these outbreaks were due to *S.* Typhimurium 9, which was the same pathogen causing two egg-related outbreaks in the first quarter of 2005. *S.* Typhimurium 9 was isolated from egg washings on the farm that supplied eggs used raw in mayonnaise for Vietnamese pork and chicken rolls. Vietnamese pork rolls also caused an outbreak of suspected staphylococcal intoxication in the Northern Territory. These rolls are a high-risk food due to the ingredients, and intensive handling required to prepare them. In the past they have caused very large outbreaks of salmonellosis that have involved fatalities.[1] Food safety agencies and Vietnamese communities need to consider new ways to make these foods safer.

There were three outbreaks of salmonellosis and an outbreak of campylobacteriosis associated with chicken meat during the quarter. Raw chicken meat is commonly contaminated with *Salmonella* and *Campylobacter*, which regularly results in outbreaks where the meat is inadequately cooked or cross contamination occurs. The outbreak of *S.* Typhimurium 64 in South Australia was unusual in that human infections with this phage type have become very rare in recent years. *S.* Typhimurium 64 was one of the most common salmonella types infecting humans in the late 1990s.

One outbreak in Victoria this quarter was suspected to be due to *Clostridium perfringens*. This outbreak was unable to be confirmed microbiologically, as the Australian Quarantine Inspection Service has restricted the importation of toxin-based test kits. Traditionally, case definitions for a *C. perfringens* outbreak use a consistent clinical picture, along with either $\geq 10^5$ organisms per gram of stool from ≥2 or more ill persons, or demonstration of enterotoxin in stool of ≥2 or more ill persons, or isolation of $\geq 10^5$ organisms per gram of epidemiologically implicated food.[2] There is considerable variation as to how different jurisdictions attribute an outbreak to this pathogen. OzFoodNet has sought the assistance of the Public Health Laboratory Network to develop a practical case definition for outbreaks of clostridial toxin poisoning for health agencies.

In June, Victoria identified an increase in cases of a rare *Salmonella* serotype—Hvittingfoss. Other eastern Australian jurisdictions also reported cases. Normally *S.* Hvittingfoss infects young children in Far North Queensland. In this instance cases occurred from southern Queensland down to Victoria and affected all age groups. The National Notifiable Diseases Surveillance System recorded 79 cases of *S.* Hvittingfoss across Australia in the second quarter of 2005, compared to 39 and 24 in 2004 and 2003 respectively (data as at 5 August 2005). OzFoodNet convened an outbreak investigation team on behalf of the Communicable Diseases Network Australia to conduct intensive hypothesis generating interviews and a case control study. The results of the investigation are not yet finalised.

Jurisdictions conducted 15 other investigations into time, place, and person clustering of *Salmonella* infections, including serotypes Birkenhead, Infantis, Liverpool, London, Mbandaka, Mississippi, Reading, Typhimurium 12, Typhimurium 135, Typhimurium 186, Virchow 8, Virchow 25 var 1, Weltevreden, and Zanzibar. There was also a considerable increase in cryptosporidiosis during the quarter, with several jurisdictions reporting cases of infection associated with community swimming pools.

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Table. Outbreaks of foodborne disease reported by OzFoodNet sites,* 1 April to 30 June 2005

State	Month of outbreak	Setting prepared	Infection	Number affected	Evidence	Responsible vehicles
ACT	April	Restaurant	*Campylobacter*	11	A	Warm chicken salad, chicken mushroom pasta,
	June	Restaurant	Norovirus	Unknown	A	Duck and quince tartlets, roast pork on bruschetta
NSW	April	Restaurant	Unknown	2	D	Suspected chicken salad
	April	Takeaway	Unknown	2	D	Suspected hamburger
	April	Restaurant	Unknown	5	D	Suspected lamb & beef dishes
	April	Restaurant	Unknown	5	D	Suspect chicken schnitzel
	May	Home	Unknown	2	D	Suspected chicken kebab
	May	Restaurant	Unknown	12	D	Unknown
	May	Restaurant	*S.* Typhimurium 9	9	M	Unknown vehicle, eggs likely source
	May	Bakery	*S.* Typhimurium 9	24	M	Vietnamese chicken & pork rolls
	May	Other	Unknown	3	D	Suspect chicken schnitzel
	May	Takeaway	Unknown	2	D	Suspect hamburger
	June	Caterer	Unknown	28	A	Suspect potato bake
	June	Caterer	Unknown	11	D	Unknown
NT	May	Home	Unknown	5	D	Vietnamese pork rolls
Qld	April	Store/deli	*Staphylococcus aureus*	2	M	Custard filled dumplings
	April	Caterer	Unknown	11	D	Unknown
	April	Home	Ciguatoxin	17	D	Spanish mackerel
	May	Home	*Campylobacter jejuni*	5	D	Unknown
	May	Takeaway	*S.* Typhimurium 108/170	2	D	Chicken meat
	May	Bakery	*S.* Typhimurium 197	14	D	Egg based bakery products
SA	May	Restaurant	*S.* Typhimurium 108/170	9	A	Marinated chicken roll
	June	Restaurant	*S.* Typhimurium 64	81	A	Bread roll with fillings
Vic	June	Hospital	Unknown	11	D	Unknown
	June	Caterer	Unknown	17	A	Pork & gravy
WA	April	Caterer	Unknown	20	A	Salad rolls suspected
	June	Home	Unknown	17	D	Unknown

* No foodborne outbreaks reported in Tasmania during the quarter.

D = Descriptive evidence implicating the suspected vehicle or suggesting foodborne transmission.

A = Analytical epidemiological association between illness and one or more foods.

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

Meningococcal disease – probable transmission during an international flight

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Abstract

Two cases of meningococcal disease were identified in passengers who travelled on the same international flight. Both cases were serogroup B with the same allelic profile. The public health action involved chemoprophylaxis for persons seated adjacent to, and in the rows in front and behind, each case. The most likely scenario is that transmission of *N. meningitidis* occurred on board a long distance flight, either from one case to the other or from an asymptomatic carrier to both cases. This scenario and the absence of reports of similar cases in the literature, indicate the risk to other passengers in this setting is low. This investigation reinforces the need for, and the distribution of, good national and international surveillance information to better inform public health decision making. *Commun Dis Intell* 2005;29:312–314.

Keywords: aeroplane, chemoprophylaxis, disease surveillance, meningococcal disease, transmission

Introduction

There have been occasional reports of probable transmission of diseases such as tuberculosis, severe acute respiratory syndrome, influenza, measles and foodborne illness during air travel.[1–4] Although a number of cases of meningococcal disease have been identified as having flown on aircraft while symptomatic or during their incubation period, no secondary cases have been reported in the literature.[5]

The aircraft cabin has been investigated as a potential setting for infectious disease transmission. Factors such as equal mixing of conditioned and recirculated air, efficient filtering and frequent air exchanges suggest there is little increased risk of disease transmission due to air quality in this setting.[6] The grouping of persons within a confined space such as the aeroplane setting still poses a risk for transmission of organisms that are easily spread from person-to-person such as measles and influenza.[7]

Specific factors that have been found to affect the risk of transmission of particular infectious diseases such as tuberculosis during air travel include proximity to the case (within two rows), duration of flight (longer than 8 hours) and infectiousness of the index case.[4,8]

We report on two cases of meningococcal disease who travelled on the same international flight during their incubation period and discuss the likely mode of transmission, the public health response and issues that emerged in response to this cluster.

Case reports

Case A was a 68-year-old female with a history of respiratory illness for three weeks prior to becoming acutely unwell in early May 2003 when she presented to hospital with signs of meningitis. The next day her condition deteriorated, petechial rash had developed and she was admitted to an intensive care unit of a Sydney hospital. The diagnosis of meningococcal disease was confirmed by polymerase chain reaction (PCR) of cerebrospinal fluid (CSF).

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Case B was a 86-year-old female who presented to hospital three days later with fever, diarrhoea and vomiting. Her respiratory status deteriorated and she was intubated and admitted to an intensive care unit of a second Sydney hospital. A petechial rash developed and diagnosis of meningococcal disease was confirmed by PCR of CSF. Both cases were non-smokers and had no medical condition predisposing them to meningococcal disease. Both recovered with antibiotic treatment.

During their incubation period both cases had been on board the same international flight from Los Angeles to Sydney, a 14.5 hour flight arriving into Sydney two days before the onset of illness in case A. The cases were both seated on the left-hand side of the aeroplane in economy class; they were situated 12 rows apart with a galley between their two sections. The investigation at the time could not identify direct contact between the two cases either before, during or after the flight.

Case A was seated in a window seat next to her husband and reported regularly walking laps of the aircraft but denied having any direct contact with other passengers on board. Case B travelled with a family member from Central America to Los Angeles and on to Sydney. On the flight from Los Angeles to Sydney, she was seated in an aisle seat and did not report walking through the aeroplane other than visiting the toilet situated at the back of the aeroplane.

Laboratory results

The CSF and blood of both cases were culture-negative for *Neisseria meningitidis.* The diagnoses of both cases were confirmed as meningococcus serogroup B by PCR of CSF specimens. The genotyping results confirmed that *N. meningitidis* detected in these cases was indistinguishable with the same allelic profile, B:19,7,1:P1.22,14 by *siaD*, *porB* and *porA* sequencing and *abcZ*–4, *adK*–10, *aroE*–15, *fumC*–9, *gdh*–8, *pdhC*–11, *pgm*–9 (ST–269 complex) by multilocus sequence typing (MLST). Both PCR and MLST techniques were performed using standard methodologies.[9–13] There were no serogroup B meningococci with the same subtype and serosubtype detected amongst invasive isolates of *N. meningitidis* identified during the same year in Australia.[14]

Public health intervention

An expert panel was convened to discuss the cases with reference to the Australian guidelines.[15] The panel recommended that chemoprophylaxis be provided to persons seated adjacent to, and in the rows in front and behind, each case. The provision of chemoprophylaxis was facilitated by the airline providing a passenger manifest and contact was established via the public health network of New South Wales. A media release was also issued. Routine surveillance for cases of meningococcal disease in Australia, which includes a thorough travel history, did not reveal any further cases of meningococcal disease in persons from the flight.

Discussion

The two cases of meningococcal disease were linked by probable transmission occurring on board a long distance flight. The probable scenario is that case B was infected by case A during inadvertent contact at some point during their travel. Case A's movements around the aeroplane and case B being seated on an aisle may be important factors in explaining the possible contact within the aircraft. Alternatively, an asymptomatic carrier on board may have transmitted *N. meningitidis* to case A and B during the period of the flight and cases A and B had different incubation periods.

Both Australian and United States of America guidelines currently recommend chemoprophylaxis for those persons seated immediately adjacent to the case for flights longer than eight hours duration.[5,15,16] The Australian guidelines, at the time these cases were notified, also suggest that persons in the rows in front and behind should be considered for chemoprophylaxis depending on their type of contact.[15] The guidelines from the United Kingdom for sporadic cases do not include chemoprophylaxis for persons travelling in the next seat on the same aeroplane unless that person has had prior prolonged close contact in a household type setting.[17]

The risk of transmission of meningococcal disease in this setting appears low. However, given the variations that exist between national guidelines, it is important that high quality surveillance information is collected to inform the public health response. Air travel allows people to cross many regions within an incubation period which emphasises the need for disseminating national and international surveillance data to accurately monitor the risk of communicable disease transmission in this setting. Therefore, Australian states and territories should notify each other of single cases of meningococcal disease in passengers who have travelled on flights longer than eight hours during their incubation period.

Our understanding of the mechanism of transmission within clusters of meningococcal disease is limited and the evidence for chemoprophylaxis in this setting is not strong.[18] This investigation did not identify significant contact between the cases or a common contact but suggests that transmission can occur on long distance flights.

Using PCR methodology to confirm the diagnosis in these cases meant the serogroup information was timely. It is important to note that the serogroup information guided the public health management of these cases, while the genotyping, which can take several weeks, was able to confirm the epidemiological link.

The passenger manifest was easily obtained in this instance. However anecdotal evidence suggests this is not often the case and a standardised procedure for this process would facilitate contact tracing exercises involving airline passengers should they be required.

This report provides evidence of probable transmission of meningococcal disease occurring on board a long distance flight. The limited number of cases in this instance and the absence of reports of similar cases in the literature, indicate the risk to other passengers in this setting is low. Factors that assisted in the public health management of this situation were having timely laboratory confirmation of cases using PCR methodology, an expert public health network available and a cooperative airline company.

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A report from the Communicable Diseases Network Australia, 1 April to 30 June 2005

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

Influenza information kits for aged care facilities

CDNA provided advice to the Ageing and Aged Care Division on the development of influenza information management kits, which were distributed to aged care facilities throughout Australia in April 2005.

Interim Guidelines for Pre-departure Health Screening and Post Arrival Health Management of Refugees from Africa

Following a request from the Department of Immigration and Multicultural and Indigenous Affairs (DIMIA) in mid-April 2005, CDNA developed the interim guidelines for screening of refugees from Africa. CDNA are currently providing further advice to DIMIA on the development of long-term related protocols.

Airline Contact Tracing Workshop

The Airline Contact Tracing Workshop was convened by CDNA on 15 April 2005 to consider ways of improving current processes of contact tracing people exposed to communicable diseases on airlines. Representatives from DIMIA, the Australian Quarantine Inspection Service, Qantas and airline associations also attended the workshop. Protocols will be developed as an outcome of the meeting.

National HIV/AIDS Strategy and National Sexually Transmitted Infections Strategy

In April 2005, CDNA considered the changes to the Strategies proposed by the Inter-Governmental Committee on HIV/AIDS, Hepatitis C and Related Diseases (IGCAHRD), including the inclusion of implementation plans. CDNA Jurisdictional Executive members will provide input to the Strategy Implementation forums, which will commence in early August 2005.

National Hepatitis C Strategy 2005–2008

In April 2005, CDNA considered and endorsed the *National Hepatitis C Strategy 2005–2008,* which was released on 1 July 2005.

National Australian and Torres Strait Islander Sexual Health and Bloodborne Virus Strategy 2005–2008

CDNA endorsed the *National Australian and Torres Strait Islander Sexual Health and Bloodborne Virus Strategy 2005–2008* on 18 May 2005, following consultation with and input from IGCAHRD. Following CDNA endorsement, the Strategy has been referred to the National Public Health Partnership for consideration.

Communicable Disease Control Conference 2005

The CDNA sponsored Conference was held on 2nd and 3rd May 2005 and was well attended by national and international representatives from the communicable disease management and control sector. Key themes covered by the conference included: threats posed by avian influenza, public health issues arising from the Asian tsunami, disease outbreaks, vaccine preventable diseases and the current and future challenges and opportunities for communicable disease control in Australia.

NAMAC proposed eradication program for *Aedes Albopictus* mosquito (associated with dengue fever) in northern Queensland and the Torres Strait

CDNA considered and endorsed the eradication program on 29 June 2005 prior to submitting it to the National Public Health Partnership for consideration. The proposed program will emphasise environmental vector control and the need to prevent the potential spread of the mosquito and dengue fever to mainland Australia.

Introduction of national varicella surveillance

Funding was provided by the Australian Government Department of Health and Ageing for varicella surveillance to complement the rollout of the National Varicella Immunisation Program. CDNA agreed to commence varicella surveillance through the National Notifiable Diseases Surveillance System.

Communicable diseases surveillance

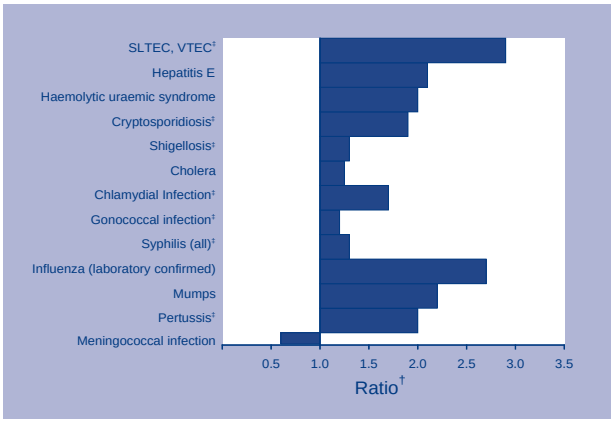
Highlights for 2nd quarter, 2005

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

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

Figure 1 shows the changes in select disease notifications with an onset in the second quarter of 2005 compared with a five-year mean for the same period. The number of notifications received in the quarter was above the five-year mean for haemolytic uraemic syndrome (HUS), cholera, influenza (laboratory-confirmed) and mumps. The following diseases were above the five-year mean for the same period and exceeded two standard deviations from the five-year mean: Shiga-like toxin producing *Escherichia coli*/verotoxin producing *E. coli* (SLTEC/VTEC), hepatitis E, cryptosporidiosis, shigellosis, chlamydial infection, gonococcal infection, syphilis (all categories) and pertussis. The number of notifications received was below the five-year mean for meningococcal infection (Figure 1).

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



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

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

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

Gastrointestinal illnesses

Botulism

One case of infant botulism in a four-month-old female was reported in this quarter from rural Victoria. The source of the botulism was unknown.

Cryptosporidiosis

There were 828 notifications of cryptosporidiosis during the quarter which is 1.9 times the five-year mean for the same period. The majority of cases were reported by Queensland (283/828), New South Wales (269/828) and Victoria (143/828).

Five hundred and nineteen of the notifications (63%) were identified as *Cryptospiridium parvum* infection; there was no species information provided for the remaining 37 per cent. Children aged under five years accounted for 46 per cent (383/828) of the total number of notifications.

Queensland reported an outbreak in March 2005 in a child care facility where 20 children and eight adult staff were affected.

In New South Wales, 105 of the 269 cases were reported in May 2005 and the notification rates for *Cryptospiridium* spp have been higher than usual since March 2005, with a peak in late April of more than 35 cases per week. Of the cases reported since May, 162 cases have been investigated for risk factors. The most common risk factor during the exposure period was found to be swimming in a public pool. The increased notification rates of cryptospiridia also appear to relate to increased testing of stools for *Cryptospiridium* spp by private laboratories since late 2004.

Haemolytic uraemic syndrome

There were four notifications of HUS during this reporting period, which is two times the five-year mean for the same period. Three of the notifications were reported from Victoria, while the fourth case was reported from South Australia.

Hepatitis E

Eight notifications of hepatitis E were received for the quarter, which is two times the five-year mean for the same period. Six of the eight cases were acquired overseas and the place of acquisition in the other cases was unknown.

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

Twenty-nine notifications of SLTEC/VTEC were received during the quarter, which is almost three times the five-year mean for the same period. Nineteen of the 29 notifications were reported from South Australia.

A case of HUS was notified from South Australia in late April. An *E. coli* serotype O111 was isolated. This case attended the same church as another SLTEC/VTEC case (also serotype O111), although the two cases did not report attending the church at the same time or eating common food. A third SLTEC/VTEC case occurred in a sibling of the HUS case and transmission was thought to be person-to-person. Both church cases had the same pulsed field gel electrophoresis pattern. Information on SLTEC/VTEC disease transmission and prevention was provided and discussed with the Elders of the church and the family of cases.

From 3–13 May, the Institute of Medical and Veterinary Science expanded the screening of bloody stools to include diarrhoeal stools. Ten SLTEC/VTEC cases were notified during this period including the sibling of the HUS case.

Shigellosis

There were 177 notifications of shigellosis during the quarter, which is 1.3 times the five-year mean for the same period. The notifications were mainly from the Northern Territory (48), New South Wales (35), and Victoria and Western Australia (32 each).

Fourteen per cent were reported as imported from overseas, 16 per cent were locally acquired and the places of acquisition of the rest were unknown.

Sixty-four per cent (114/177) of the cases had species recorded. The most frequently notified species was *Shigella sonnei* biotype A, with a further 25 notifications of *Shigella sonnei* of unknown biotype (Table 1).

Previously published work has shown that the prevalent species of shigellae in New South Wales over a four month period in 2000, was *Shigella sonnei* biotype G.[1] *Shigella sonnei* biotype G has also been associated with an outbreak in a child care centre in Victoria in 2000.[2]

Quarantinable diseases

Cholera

There was one notification of cholera from Western Australia in a 49-year-old female returning from Indonesia. The isolate was identified as *Vibrio cholerae O1* Ogawa, a toxin-producing strain, as confirmed by polymerase chain reaction for the presence of the ctx A gene.

Table 1. Notifications of shigellosis, 1 April to 30 June 2005, by species and type

Shigella species	Subtype/biotype	Number of notifications	Per cent of notifications (%)
Shigella boydii	Not typed	2	2
Shigella flexneri	1	2	2
Shigella flexneri	2	1	1
Shigella flexneri	2A	13	11
Shigella flexneri	4	9	8
Shigella flexneri	4a	1	1
Shigella flexneri	4a mannitol neg	7	6
Shigella flexneri	4b	6	5
Shigella flexneri	6	4	4
Shigella flexneri	Not typed	8	7
Shigella sonnei	biotype A	29	25
Shigella sonnei	biotype F	1	1
Shigella sonnei	biotype G	6	5
Shigella sonnei	Not typed	25	22
Total		114	100

Sexually transmissible infections

Chlamydial infection

During the quarter there were 10,856 notifications of chlamydial infection received from all jurisdictions, which is 1.7 times the five-year mean for the same period. The majority of these notifications were reported by New South Wales (2,823), Queensland (2,746) and Victoria (2,339).

Seventy-eight per cent of the notifications were reported from the 15–29 year age group. Sixty per cent of the chlamydial infection notifications were reported from females.

Vaccine preventable diseases

Influenza (laboratory-confirmed)

There were 740 cases of laboratory-confirmed influenza in the second quarter of 2005. This was nearly three times the average number of notifications for this time of year. New South Wales, Queensland and Victoria each contributed 31 per cent toward the total number of notifications. Seventy-seven per cent of the national laboratory-confirmed influenza notifications were type A, 21 per cent type B and two per cent were of unknown type.

Mumps

There were 73 notifications of mumps in the quarter, which is 2.2 times the five-year mean for the same period. The majority of cases were reported from New South Wales (31) and Queensland (32). Of the 73 cases, 51 cases (70%) were reported from the 20–34 year age group.

Pertussis

For the second quarter, 2,370 pertussis notifications were received, from which 1,395 (60%) were reported by New South Wales. Three per cent of the notifications were reported in infants aged less than one year. Pertussis activity in the quarter was two times the average number of notifications for this time of year.

Other bacterial infections

Meningococcal infections

There were 75 notifications of meningococcal infection during the quarter, which was two-thirds the average number reported in the quarter over the previous five years. Of the 75 cases, meningococcal serogroup data were available for 62 cases. There were 47 cases of serogroup B (62%), eight cases of serogroup C (11%), four cases of serogroup Y and two cases of serogroup W135 (Table 2). Thirteen cases were not typed (17%).

One case of serogroup A received from Victoria was in an Ethiopian refugee. This was only the third notification of serogroup A received over the past five years, the last one occurring in 2004.

Table 2 shows that during the quarter, there were three deaths from meningococcal infections, two from serogroup B and one from serogroup Y. There were no reported deaths during the quarter from *Neisseria meningiditis* serogroup C, for which a vaccine is currently available as part of the Australian Standard Vaccination Schedule.[3]

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Table 2. Notifications and deaths due to meningococcal infection, 1 April to 30 June 2005, by State and serogroup

Jurisdiction	Notification by serogroup							Death(s) by serogroup						
	A	B	C	W135	Y	NT	Total	A	B	C	W135	Y	NT	Total
ACT	0	1	1	0	0	0	2	0	0	0	0	0	0	0
NSW	0	17	2	2	2	10	33	0	1	0	0	1	0	2
NT	0	1	1	0	0	2	4	0	0	0	0	0	0	0
Qld	0	10	2	0	0	1	13	0	1	0	0	0	0	1
SA	0	3	0	0	0	0	3	0	0	0	0	0	0	0
Tas	0	3	0	0	0	0	3	0	0	0	0	0	0	0
Vic	1	8	2	0	1	0	12	0	0	0	0	0	0	0
WA	0	4	0	0	1	0	5	0	0	0	0	0	0	0
Total	1	47	8	2	4	14	75	0	2	0	0	1	0	3

NT Not typed.

Tables

A summary of diseases currently being reported by each jurisdiction is provided in Table 3. There were 31,148 notifications to the National Notifiable Diseases Surveillance System (NNDSS) with a notification date between 1 April and 30 June 2005 (Table 4). The notification rate of diseases per 100,000 population for each State or Territory is presented in Table 5.

There were 2,494 reports received by the Virology and Serology Laboratory Reporting Scheme (LabVISE) in the reporting period, 1 April and 30 June 2005 (Tables 6 and 7).

Table 3. Reporting of notifiable diseases by jurisdiction

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

Disease	Data received from:
Vaccine preventable diseases	
Diphtheria	All jurisdictions
Haemophilus influenzae type b	All jurisdictions
Influenza (laboratory confirmed)*	All jurisdictions
Measles	All jurisdictions
Mumps	All jurisdictions
Pertussis	All jurisdictions
Pneumococcal disease (invasive)	All jurisdictions
Poliomyelitis	All jurisdictions
Rubella	All jurisdictions
Rubella - congenital	All jurisdictions
Tetanus	All jurisdictions
Vectorborne diseases	
Barmah Forest virus infection	All jurisdictions
Flavivirus infection (NEC)†	All jurisdictions
Dengue	All jurisdictions
Japanese encephalitis virus	All jurisdictions
Kunjin virus‡	All jurisdictions except ACT
Malaria	All jurisdictions
Murray Valley encephalitis virus‡	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 unspecified	All jurisdictions
Ornithosis	All jurisdictions
Q fever	All jurisdictions
Other bacterial infections	
Legionellosis	All jurisdictions
Leprosy	All jurisdictions
Meningococcal infection	All jurisdictions
Tuberculosis	All jurisdictions

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

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

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

Table 4. Notifications of diseases received by State and Territory health authorities in the period 1 April to 30 June 2005, by date of onset*

Disease	State or territory								Total 2nd quarter 2005†	Total 1st quarter 2005	Total 2nd quarter 2004	Last 5 years mean 2nd quarter	Year to date 2005	Last 5 years YTD mean	Ratio‡
	ACT	NSW	NT	Qld	SA	Tas	Vic	WA							
Bloodborne diseases															
Hepatitis B (incident)	1	12	4	18	1	1	14	7	58	73	72	100.0	131	190.2	0.6
Hepatitis B (unspecified)	20	942	49	206	88	13	446	114	1,878	1,880	1,413	1,788.0	3,758	3,457.6	1.0
Hepatitis C (incident)	5	11	0	NN	12	10	11	28	77	64	74	118.0	141	254.4	0.6
Hepatitis C (unspecified)	42	1,693	64	673	121	56	754	277	3,680	3,576	3,386	4,164.2	7,256	8,632.8	0.9
Hepatitis D	0	2	0	2	0	0	0	0	4	4	9	7.2	8	13.0	0.6
Gastrointestinal diseases															
Botulism	0	0	0	0	0	0	1	0	1	1	0	0.0	2	0.3	0.0
Campylobacteriosis§	84	NN	75	907	420	166	1,353	515	3,520	4,155	3,006	3,338.4	7,675	7,171.8	1.1
Cryptosporidiosis‖	14	269	19	283	40	4	143	56	828	1,247	432	447.3	2,075	1,364.0	1.9
Haemolytic uraemic syndrome	0	0	0	0	1	0	3	0	4	4	3	2.0	8	5.0	2.0
Hepatitis A	0	19	14	18	1	1	8	25	86	81	79	125.2	167	280.8	0.7
Hepatitis E	1	1	0	4	0	0	2	0	8	17	7	3.8	25	9.4	2.1
Listeriosis	0	6	0	2	0	0	4	1	13	13	21	17.6	26	37.4	0.7
Salmonellosis (NEC)	27	528	98	658	177	37	241	186	1,952	2,691	1,972	1,761.0	4,643	4,332.8	1.1
Shigellosis	0	35	48	19	9	2	32	32	177	226	147	133.0	400	285.4	1.3
SLTEC, VTEC¶	0	2	0	2	19	0	2	4	29	13	8	9.8	41	27.0	2.9
Typhoid	0	3	0	0	1	0	3	3	10	23	15	10.4	33	37.6	1.0
Quarantinable diseases															
Cholera	0	0	0	0	0	0	0	1	1	2	2	0.8	3	1.8	1.3
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 4. Notifications of diseases received by State and Territory health authorities in the period 1 April to 30 June 2005, by date of onset,* *continued*

Disease	State or territory								Total 2nd quarter 2005[†]	Total 1st quarter 2005	Total 2nd quarter 2004	Last 5 years mean 2nd quarter	Year to date 2005	Last 5 years YTD mean	Ratio[‡]
	ACT	NSW	NT	Qld	SA	Tas	Vic	WA							
Sexually transmissible infections															
Chlamydial infection	183	2,823	477	2,746	719	255	2,339	1,317	10,859	10,293	8,995	6,351.8	21,152	12,707.0	1.7
Donovanosis	0	0	1	1	0	0	0	0	2	5	1	5.0	7	11.0	0.4
Gonococcal infection	1	372	558	318	138	6	267	377	2,037	2,042	1,937	1,710.0	4,079	3,394.2	1.2
Syphilis (all)	6	248	61	77	2	6	130	25	555	581	554	523.8	1,136	1,028.8	1.3
Syphilis < two years duration	2	46	32	29	0	2	30	1	142	144	136	NA	286	244.3	NA
Syphilis > two years or unspecified duration	4	200	28	47	2	4	100	24	409	432	414	NA	841	662.0	NA
Syphilis - congenital	0	2	1	1	0	0	0	0	4	5	4	4.0	9	6.6	1.0
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	0	0	0	0	0	0	1	7	3	8.6	8	13.0	0.1
Influenza (laboratory confirmed)[‖]	4	225	17	220	3	1	227	43	740	372	166	274.0	1,112	370.0	2.7
Measles	0	2	0	0	0	0	0	0	2	5	6	18.0	7	46.6	0.1
Mumps	0	31	2	32	0	0	5	3	73	49	21	32.6	122	61.8	2.2
Pertussis	53	1,395	14	297	317	13	190	91	2,370	2,222	1,229	1,200.0	4,592	2,437.8	2.0
Pneumococcal disease (invasive)[‖]	6	159	21	87	35	10	86	29	433	257	621	565.8	690	856.8	0.8
Poliomyelitis	0	0	0	0	0	0	0	0	0	0	0	0.0	0	0.0	0.0
Rubella	0	5	0	2	0	0	2	5	14	6	11	36.2	20	76.6	0.4
Rubella - congenital	0	0	0	0	0	0	0	0	0	0	0	0.2	0	0.6	0.0
Tetanus	0	0	0	0	0	0	0	0	0	0	2	0.6	0	3.0	0.0
Vectorborne diseases															
Barmah Forest virus infection	0	144	19	249	4	0	6	12	434	359	319	415.4	793	704.4	1.0
Dengue	0	8	5	25	2	0	1	2	43	95	64	93.0	138	258.8	0.5
Flavivirus infection (NEC)	0	1	0	7	0	0	0	0	8	9	9	16.4	17	42.2	0.5
Japanese encephalitis virus[‖]	0	0	0	0	0	0	0	0	0	0	0	0.2	0	0.4	0.0
Kunjin virus[‖]	0	0	0	0	0	0	0	0	0	1	0	1.8	1	7.8	0.0
Malaria	1	21	19	60	4	6	36	20	167	352	150	173.2	519	366.6	1.0
Murray Valley encephalitis virus[‖]	0	0	0	0	0	0	0	0	0	2	1	0.3	2	2.0	0.0
Ross River virus infection	1	174	36	281	15	4	15	27	553	916	1,376	1,375.0	1,469	2,865.0	0.4

Table 4. Notifications of diseases received by State and Territory health authorities in the period 1 April to 30 June 2005, by date of onset,* *continued*

Disease	State or territory								Total 2nd quarter 2005†	Total 1st quarter 2005	Total 2nd quarter 2004	Last 5 years mean 2nd quarter	Year to date 2005	Last 5 years YTD mean	Ratio‡
	ACT	NSW	NT	Qld	SA	Tas	Vic	WA							
Zoonoses															
Anthrax‖	0	0	0	0	0	0	0	0	0	0	0	0.0	0	0.0	0.0
Australian bat lyssavirus‖	0	0	0	0	0	0	0	0	0	0	0	0.0	0	0.0	0.0
Brucellosis	0	0	0	3	0	0	0	0	3	12	6	5.6	15	12.6	0.5
Leptospirosis	0	3	1	26	1	0	0	2	33	40	55	56.0	73	124.6	0.6
Lyssavirus unspecified‖	0	0	0	0	0	0	0	0	0	0	0	0.0	0	0.0	0.0
Ornithosis	0	38	0	0	1	0	6	0	45	37	58	43.6	82	81.0	1.0
Q fever	0	35	2	41	11	0	13	1	103	79	106	152.6	182	326.6	0.7
Other bacterial infections															
Legionellosis	0	17	1	14	12	0	8	16	68	86	93	120.2	154	196.6	0.6
Leprosy	0	0	0	1	0	0	0	0	1	4	1	1.4	5	3.6	0.7
Meningococcal infection	2	33	4	13	3	3	12	5	75	73	117	137.0	148	244.2	0.6
Tuberculosis	0	40	5	40	10	3	93	19	210	197	154	209.4	407	434.0	0.8
Total	451	9,298	1,614	7,332	2,167	597	6,453	3,243	31,155	32,171	26,701	25,601.2	63,322	52,906.8	1.2

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

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

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

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

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

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

NN Not notifiable.

NEC Not elsewhere classified.

Table 5. Notification rates of diseases by state or territory, 1 April to 30 June 2005. (Rate per 100,000 population)

Disease*	State or territory								Australia
	ACT	NSW	NT	Qld	SA	Tas	Vic	WA	
Bloodborne diseases									
Hepatitis B (incident)	1.2	0.7	8.0	1.9	0.3	2.5	1.1	1.4	1.2
Hepatitis B (unspecified)	24.7	56.7	100.0	21.2	23.7	9.1	35.9	22.88	37.1
Hepatitis C (incident)	6.2	0.7	0.0	NN	3.4	7.5	1.0	5.9	2.0
Hepatitis C (unspecified)	51.8	100.8	128.1	69.3	33.9	46.5	60.7	55.7	73.4
Hepatitis D	0.0	0.1	0.0	0.2	0.0	0.0	0.0	0.0	0.1
Gastrointestinal diseases									
Botulism	0.0	0.0	0.0	0.0	0.0	0.0	0.1	0.0	0.0
Campylobacteriosis†	103.7	NN	150.1	93.6	109.8	134.4	108.8	103.9	105.2
Cryptosporidiosis	17.3	16.0	38.0	29.2	10.4	3.3	11.5	11.3	16.5
Haemolytic uraemic syndrome	0.0	0.0	0.0	0.0	0.3	0.0	0.2	0.0	0.1
Hepatitis A	0.0	1.1	28.0	1.9	0.3	0.8	0.6	5.0	1.7
Hepatitis E	1.2	0.1	0.0	0.4	0.0	0.0	0.2	0.0	0.2
Listeriosis	0.0	0.4	0.0	0.2	0.0	0.0	0.3	0.2	0.3
Salmonellosis (NEC)	33.3	31.4	196.1	67.8	46.1	30.7	19.4	37.7	38.8
Shigellosis	0.0	2.1	96.0	2.0	2.1	1.7	2.6	6.5	3.5
SLTEC, VTEC‡	0.0	0.1	0.0	0.2	4.7	0.0	0.2	1.0	0.6
Typhoid	0.0	0.2	0.0	0.0	0.3	0.0	0.2	0.6	0.2
Quarantinable diseases									
Cholera	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.2	0.0
Plague	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
Rabies	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
Smallpox	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
Tularemia	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
Viral haemorrhagic fever	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
Yellow fever	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
Sexually transmissible infections									
Chlamydial infection	225.9	167.0	954.4	283.0	188.5	208.2	188.3	267.2	216.0
Donovanosis	0.0	0.0	2.0	0.1	0.0	0.0	0.0	0.0	0.0
Gonococcal infection	1.2	22.2	1,116.5	32.8	38.1	4.1	21.5	76.1	40.7
Syphilis (all)	1.9	4.4	30.4	2.0	0.1	1.2	2.6	1.3	3.0
Syphilis < 2 years duration	0.6	0.5	15.9	0.7	0.0	0.4	0.6	0.1	0.7
Syphilis > 2 years or unspecified duration	1.2	3.8	13.9	1.2	0.1	0.8	2.0	1.2	2.3
Syphilis - congenital	0.0	0.0	0.5	0.0	0.0	0.0	0.0	0.0	0.0

Table 5. Notification rates of diseases by state or territory, 1 April to 30 June 2005. (Rate per 100,000 population) , *continued*

Disease*	State or territory								
	ACT	NSW	NT	Qld	SA	Tas	Vic	WA	Australia
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	0.0	0.0	0.0	0.0	0.0	0.0	0.0
Influenza (laboratory confirmed)	4.9	13.4	34.0	22.8	0.8	0.8	18.4	8.7	14.8
Measles	0.0	0.1	0.0	0.0	0.0	0.0	0.0	0.0	0.0
Mumps	0.0	1.8	4.0	3.3	0.0	0.0	0.4	0.6	1.5
Pertussis	65.4	85.6	28.0	30.6	82.6	11.6	15.4	19.4	48.2
Pneumococcal disease (invasive)	8.6	9.6	42.0	9.1	9.1	8.3	7.0	5.7	8.7
Poliomyelitis	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
Rubella	0.0	0.3	0.0	0.2	0.0	0.0	0.2	1.0	0.3
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	0.0	0.0	0.0	0.0	0.0	0.0
Vectorborne diseases									
Barmah Forest virus infection	0.0	8.6	38.0	25.7	1.0	0.0	0.5	2.4	8.6
Dengue	0.0	0.5	10.0	2.6	0.5	0.0	0.1	0.4	0.9
Flavivirus infection (NEC)	0.0	0.1	0.0	0.7	0.0	0.0	0.0	0.0	0.2
Japanese encephalitis virus	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
Kunjin virus	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
Malaria	1.2	1.2	38.0	6.2	1.0	5.0	2.9	4.0	3.3
Murray Valley encephalitis virus	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
Ross River virus infection	1.2	10.4	72.0	29.0	3.9	3.3	1.3	5.4	11.0
Zoonoses									
Anthrax	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
Australian bat lyssavirus	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
Brucellosis	0.0	0.1	0.0	0.3	0.0	0.0	0.0	0.0	0.1
Leptospirosis	0.0	0.2	2.0	2.8	0.0	0.0	0.0	0.4	0.7
Lyssavirus unspecified	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
Ornithosis	0.0	2.3	0.0	0.0	0.3	0.0	0.5	0.0	0.9
Q fever	0.0	2.1	4.0	4.3	2.9	0.0	1.2	0.2	2.1
Other bacterial infections									
Legionellosis	0.0	1.0	2.0	1.5	3.4	0.0	0.6	3.2	1.4
Leprosy	0.0	0.0	0.0	0.1	0.0	0.0	0.0	0.0	0.0
Meningococcal infection	2.5	2.0	8.0	1.3	0.8	2.5	1.0	1.0	1.6
Tuberculosis	0.0	4.0	10.0	4.1	2.6	3.3	7.5	3.8	4.8

* Rates are subject to retrospective revision.

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

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

NN Not notifiable.

NEC Not elsewhere classified.

Table 6. Virology and serology laboratory reports by state or territory* for the reporting period 1 April to 30 June 2005, and total reports for the year†

	State or territory								This period 2005	This period 2004	Year to date 2005	Year to date 2004
	ACT	NSW	NT	Qld	SA	Tas	Vic	WA				
Measles, mumps, rubella												
Measles virus	–	1	–	–	–	–	–	–	1	6	3	13
Mumps virus	–	–	–	6	1	–	4	–	11	1	17	3
Rubella virus	–	–	–	2	–	–	1	–	3	2	7	8
Hepatitis viruses												
Hepatitis A virus	–	–	1	4	5	–	–	–	10	9	16	19
Hepatitis D virus	–	–	–	–	3	–	1	–	4		6	3
Hepatitis E virus	–	–	–	–	1	–	2	–	3	6	9	11
Arboviruses												
Ross River virus	–	2	3	55	9	2	3	–	74	250	282	681
Barmah Forest virus	–	3	–	66	5	–	–	–	74	41	128	126
Flavivirus (unspecified)	–	–	–	7	–	–	–	–	7	29	20	78
Adenoviruses												
Adenovirus not typed/pending	–	22	–	17	67	–	48	–	154	278	262	449
Herpesviruses												
Cytomegalovirus	5	86	4	27	69	3	24	1	219	191	377	399
Varicella-zoster virus	1	34	7	187	107	4	10	–	350	403	711	853
Epstein-Barr virus	–	4	7	160	166	–	13	34	384	539	936	1,168
Other DNA viruses												
Poxvirus group not typed	–	1	–	–	–	–	–	–	1	1	1	2
Parvovirus	1	3	–	10	5	–	5	–	24	57	78	118
Picornavirus family												
Coxsackievirus A9	–	1	–	–	–	–	–	–	1	1	2	1
Coxsackievirus A16	1	2	–	–	–	–	–	–	3	3	3	5
Echovirus type 6	–	1	–	–	–	–	–	–	1		2	
Echovirus type 7	–	2	–	–	–	–	–	–	2	1	5	1
Echovirus type 9	–	1	–	–	–	–	–	–	1	2	1	2
Echovirus type 11	–	2	–	–	–	–	–	–	2	4	3	6
Echovirus type 13	–	1	–	–	–	–	–	–	1		1	
Echovirus type 18	–	2	–	–	–	–	–	–	2		9	3
Echovirus type 22	–	1	–	–	–	–	–	–	1	1	1	2
Echovirus type 30	–	8	–	–	–	–	–	–	8	2	17	4
Poliovirus type 1 (uncharacterised)	–	2	–	–	–	–	–	–	2	4	4	6
Poliovirus type 2 (uncharacterised)	–	1	–	–	–	–	–	–	1	6	5	8
Poliovirus type 3 (uncharacterised)	–	1	–	–	–	–	–	–	1	1	2	1
Rhinovirus (all types)	–	65	–	–	14	1	–	–	80	106	155	187
Enterovirus type 71 (BCR)	–	1	–	–	–	–	–	–	1		2	2
Enterovirus not typed/pending	4	24	–	4	2	–	5	–	39	45	63	87
Picornavirus not typed	–	–	–	–	–	1	–	–	1	2	1	4

Table 6. Virology and serology laboratory reports by state or territory* for the reporting period 1 April to 30 June 2005, and total reports for the year,† *continued*

	State or territory								This period 2005	This period 2004	Year to date 2005	Year to date 2004
	ACT	NSW	NT	Qld	SA	Tas	Vic	WA				
Ortho/ paramyoviruses												
Influenza A virus	–	5	1	9	42	–	6	–	63	31	86	71
Influenza B virus	–	2	–	4	23	–	9	–	38	25	70	37
Parainfluenza virus type 1	–	4	–	–	2	–	9	–	15	57	24	97
Parainfluenza virus type 2	–	13	–	4	6	–	4	–	27	4	33	6
Parainfluenza virus type 3	–	9	–	1	19	–	9	–	38	109	80	197
Respiratory syncytial virus	–	205	–	82	52	20	121	2	482	1,193	583	1,393
Paramyxovirus (unspecified)	–	–	–	–	–	–	9	–	9		9	
Other RNA viruses												
HTLV-1	–	–	–	–	1	–	–	–	1	4	3	6
Rotavirus	–	16	–	–	77	3	51	–	147	92	202	169
Norwalk agent	–	–	–	–	–	–	78	–	78	114	93	197
Other												
Chlamydia trachomatis not typed	–	219	3	581	471	20	6	1	1,301	1,247	2,460	2,432
Chlamydia psittaci	–	–	–	–	–	–	16	–	16	47	30	109
Mycoplasma pneumoniae	–	4	3	88	65	3	49	5	217	305	453	660
Mycoplasma hominis	–	1	–	–	–	–	–	–	1		2	1
Coxiella burnetii (Q fever)	–	4	–	13	28	–	6	–	51	32	85	80
Rickettsia prowazeki	–	–	–	–	29	–	–	–	29		51	
Rickettsia tsutsugamushi	–	–	–	–	7	–	–	–	7	1	18	1
Rickettsia - spotted fever group	–	–	–	–	44	1	–	–	45		94	
Streptococcus group A	–	–	–	93	–	1	44	–	138	98	242	223
Yersinia enterocolitica	–	2	–	–	–	–	–	–	2	1	6	2
Brucella species	–	–	–	1	–	–	–	–	1	3	3	3
Bordetella pertussis	–	23	–	35	251	–	44	–	353	112	734	268
Legionella pneumophila	–	3	–	–	4	–	–	–	7	33	14	53
Legionella longbeachae	–	1	–	–	4	–	1	–	6	22	18	38
Cryptococcus species	–	–	–	2	13	–	–	–	15	10	25	23
Leptospira species	–	–	–	9	4	–	–	–	13	3	16	16
Treponema pallidum	–	13	1	132	134	–	–	–	280	287	503	615
Entamoeba histolytica	–	–	–	3	–	–	1	–	4	1	8	5
Toxoplasma gondii	–	2	–	–	5	–	1	–	8	3	16	17
Total	0	272	7	957	1,059	25	168	6	2,494	2,205	4,778	4,546

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

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

– No data received this period.

Table 7. Virology and serology reports by laboratories for the reporting period 1 April to 30 June 2005*

State or territory	Laboratory	April 2005	May 2005	June 2005	Total this period
Australian Capital Territory	The Canberra Hospital	–	–	–	–
New South Wales	Institute of Clinical Pathology and Medical Research, Westmead	145	171	156	472
	New Children's Hospital, Westmead	69	96	127	292
	Repatriation General Hospital, Concord	–	–	–	–
	Royal Prince Alfred Hospital, Camperdown	–	–	–	–
	South West Area Pathology Service, Liverpool	–	–	–	–
Queensland	Queensland Medical Laboratory, West End	296	593	794	1,683
	Townsville General Hospital	–	–	–	–
South Australia	Institute of Medical and Veterinary Science, Adelaide	613	567	550	1,730
Tasmania	Northern Tasmanian Pathology Service, Launceston	13	17	24	54
	Royal Hobart Hospital, Hobart	–	–	–	–
Victoria	Monash Medical Centre, Melbourne	17	51	43	111
	Royal Children's Hospital, Melbourne	78	118	94	290
	Victorian Infectious Diseases Reference Laboratory, Fairfield	58	43	74	175
Western Australia	PathCentre Virology, Perth	–	–	–	–
	Princess Margaret Hospital, Perth	–	–	–	–
	Western Diagnostic Pathology	–	–	51	51
Total		1,289	1,656	1,913	4,858

* The complete list of laboratories reporting for the 12 months, January to December 2005, will appear in every report regardless of whether reports were received in this reporting period. Reports are not always received from all laboratories.

– No data received this period.

Additional reports

Australian Sentinel Practice Research Network

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

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

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

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

Data from 1 January to 30 June 2005 compared with 2004 are shown as the rate per 1,000 consultations in Figures 2 and 3.

Figure 2. Consultation rates for influenza-like illness, ASPREN, 1 January to 30 June 2005, by week of report

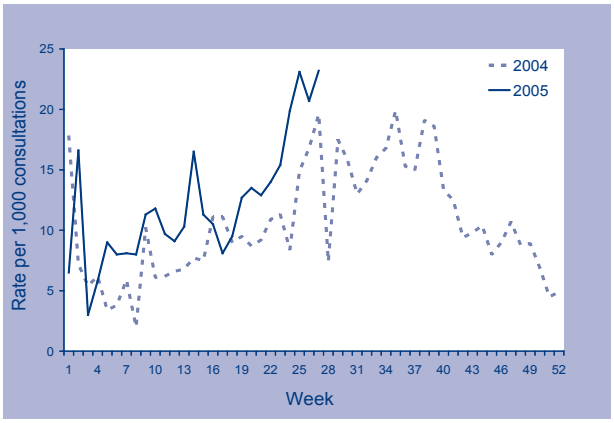
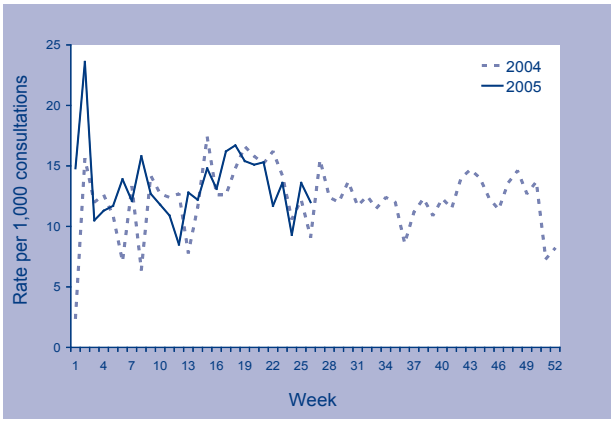


Figure 3. Consultation rates for gastroenteritis, ASPREN, 1 January to 30 June 2005, by week of report



Childhood immunisation coverage

Tables 8, 9 and 10 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 2004, at 24 months of age for the cohort born between 1 January and 31 March 2003, and at 6 years of age for the cohort born between 1 January and 31 March 1999 according to the Australian Standard Vaccination Schedule.

For information about the Australian Childhood Immunisation Register see Surveillance systems reported in CDI, published in Commun Dis Intell 2005;29:90 and for a full description of the methodology used by the Register see Commun Dis Intell 1998;22:36-37.

Commentary on the trends in ACIR data is provided by the National Centre for Immunisation Research and Surveillance of Vaccine Preventable Diseases (NCIRS). For further information please contact the NCIRS on telephone +61 2 9845 1435 or email: brynleyh@chw.edu.au.

Immunisation coverage for children 'fully immunised' at 12 months of age for Australia increased marginally from the last quarter by 0.3 percentage points to 91.0 per cent (Table 8). There was a substantial increase in 'fully immunised' coverage by State and Territory in only one jurisdiction, the Australian Capital Territory, with an increase of 3.0 percentage points. As expected, the Australian Capital Territory also had increases in coverage for individual vaccines.

There was a 0.1 per cent increase in coverage for children 'fully immunised' at 24 months of age for Australia, to 91.8 per cent (Table 9). Coverage for individual vaccines remained largely unchanged in most jurisdictions and was greater than 95 per cent in almost all jurisdictions for all vaccines, except *Haemophilus influenzae* type b and measles-mumps-rubella.

Table 10 shows immunisation coverage estimates for children 'fully immunised' at 6 years of age and for individual vaccines for Australia by state and territory. Coverage was largely unchanged in most jurisdictions, apart from decreases in Tasmania and the Australian Capital Territory. This was also reflected in individual vaccines. Coverage for

Table 8. 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 2004; assessment date 30 June 2005

Vaccine	State or territory								
	ACT	NSW	NT	Qld	SA	Tas	Vic	WA	Australia
Number of children	1,012	21,604	903	13,187	4,445	1,353	15,600	6,485	64,589
Diphtheria, tetanus, pertussis (%)	96.4	92.1	92.8	91.9	91.7	92.9	93.1	91.0	92.3
Poliomyelitis (%)	96.3	92.0	92.5	91.8	91.7	93.1	93.0	90.9	92.2
Haemophilus influenzae type b (%)	96.9	93.9	96.3	93.8	94.6	94.9	94.8	93.8	94.3
Hepatitis B (%)	97.2	94.8	96.9	94.4	94.7	94.8	94.7	93.5	94.6
Fully immunised (%)	95.7	90.6	91.9	90.8	91.0	91.2	91.8	90.0	91.0
Change in fully immunised since last quarter (%)	+2.9	-0.1	-0.3	-0.0	-0.1	-1.7	+1.0	+0.7	+0.3

Table 9. 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 2003; assessment date 30 June 2005*

Vaccine	State or territory								
	ACT	NSW	NT	Qld	SA	Tas	Vic	WA	Australia
Total number of children	981	21,109	926	12,740	4,237	1,391	15,052	6,191	62,627
Diphtheria, tetanus, pertussis (%)	94.6	94.8	96.3	94.8	94.7	97.0	95.8	93.8	95.0
Poliomyelitis (%)	94.5	94.7	96.5	94.8	94.7	97.2	95.7	93.7	94.9
Haemophilus influenzae type b (%)	93.0	92.7	94.3	93.5	93.4	95.7	94.3	91.3	93.3
Measles, mumps, rubella (%)	93.7	92.9	95.6	93.4	93.6	95.5	94.4	92.0	93.4
Hepatitis B(%)	95.0	95.4	97.8	95.6	95.5	97.6	96.5	94.2	95.7
Fully immunised (%)	91.6	91.2	93.6	91.6	92.1	94.6	92.9	90.0	91.8
Change in fully immunised since last quarter (%)	-2.0	+0.3	-1.3	-0.3	-0.7	+0.6	+0.7	-0.6	+0.1

* The 12 months age data for this cohort was published in *Commun Dis Intell* 2004;28:422.

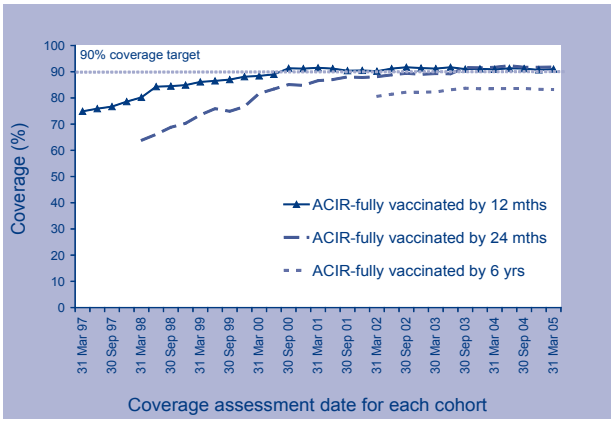
Table 10. Percentage of children immunised at 6 years of age, preliminary results by disease and state or territory for the birth cohort 1 January to 31 March 1999; assessment date 30 June 2005

Vaccine	State or territory								
	ACT	NSW	NT	Qld	SA	Tas	Vic	WA	Australia
Total number of children	1,032	22,320	852	13,611	4,749	1,618	15,956	6,745	66,883
Diphtheria, tetanus, pertussis (%)	88.3	85.1	85.3	81.3	83.8	83.1	87.0	82.3	84.4
Poliomyelitis (%)	88.4	85.0	86.7	81.6	84.0	83.3	87.3	82.5	84.5
Measles, mumps, rubella (%)	88.1	84.9	87.0	81.5	84.0	82.8	87.0	82.4	84.4
Fully immunised (%)	87.9	83.8	84.3	79.9	82.8	81.9	86.2	80.8	83.2
Change in fully immunised since last quarter (%)	-1.9	+0.1	-0.8	-0.7	+0.0	-2.3	+0.4	+1.0	-0.1

vaccines assessed at 6 years is at or near 85 per cent in most jurisdictions, but Western Australia, Tasmania and Queensland still remain below this.

Figure 4 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 6 years, although the rate of increase has slowed over the past two years for all age groups. The figure shows that there have now been seven consecutive quarters where 'fully immunised' coverage at 24 months of age has been greater than 'fully immunised' coverage at 12 months of age, following the removal of the requirement for the 18 month DTPa vaccine. However, both measures have been above 90 per cent for this 21-month period and show levels of high coverage being maintained over a significant period of time.

Figure 4. Trends in vaccination coverage, Australia, 1997 to 2005, by age cohorts



Gonococcal surveillance

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

The Australian Gonococcal Surveillance Programme (AGSP) reference laboratories in the various States and Territories report data on sensitivity to an agreed 'core' group of antimicrobial agents quarterly. The antibiotics currently routinely surveyed are penicillin, ceftriaxone, ciprofloxacin and spectinomycin, all of which are administered as single dose regimens and currently used in Australia to treat gonorrhoea. When in vitro resistance to a recommended agent is demonstrated in 5 per cent or more of isolates from a general population, it is usual to remove that agent from the list of recommended treatment.[1] Additional data are also provided on other antibiotics from time to time. At present all laboratories also test isolates for the presence of high level (plasmid-mediated) resistance to the tetracyclines, known as TRNG. Tetracyclines are however, not a recommended therapy for gonorrhoea in Australia. Comparability of data is achieved by means of a standardised system of testing and a program-specific quality assurance process. Because of the substantial geographic differences in susceptibility patterns in Australia, regional as well as aggregated data are presented. For more information see Commun Dis Intell *2005;29:92-93.*

Reporting period 1 January to 31 March 2005

The AGSP laboratories received a total of 985 isolates in this quarter of which 952 underwent susceptibility testing. This represents a slight decrease from the 1,001 reported for the same period in 2004 and 1,051 seen in 2003. About 33 per cent of this total was from New South Wales, 20 per cent from Queensland, 16 per cent from Victoria, 15 per cent from the Northern Territory, 11 per cent from Western Australia and four per cent from South Australia. Small numbers of isolates were also received from Tasmania and the Australian Capital Territory.

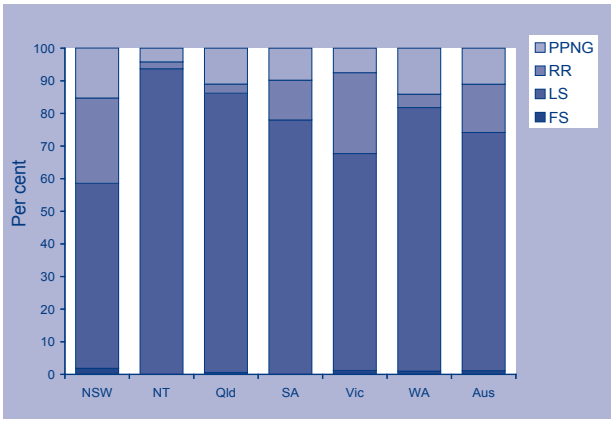
Penicillins

In this quarter 246 (25.8%) of all isolates examined were penicillin resistant by one or more mechanisms. One hundred and five (11%) were penicillinase producing *Neisseria gonorrhoeae* (PPNG) and 141 (14.8%) resistant by chromosomal mechanisms, (CMRNG). The proportion of all strains resistant to the penicillins by any mechanism ranged from 6.2 per cent in the Northern Territory to 41 per cent in New South Wales.

Figure 5 shows the proportions of gonococci fully sensitive (MIC ≤0.03 mg/L), less sensitive (MIC 0.06–0.5 mg/L), relatively resistant (MIC ≥1 mg/L) or else PPNG aggregated for Australia and by state 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.

The highest number and proportion of PPNG was found in New South Wales where the 49 PPNG were 15.3 per cent of all isolates. Fourteen PPNG representing 14.1 per cent of all isolates were found in Western Australia, 12 (7.5%) in Victoria and 20 (11%) in Queensland. Six PPNG (4.2%) were found in the Northern Territory, all from Darwin. Increases in PPNG numbers (compared with the first quarter of 2004) were noted in Queensland (from 6 to 20) and New South Wales. More isolates were resistant to the penicillins by separate chromosomal mechanisms and CMRNG were especially prominent in Victoria (40 isolates, 25 per cent of all gonococci

Figure 5. Categorisation of gonococci isolated in Australia, 1 January to 31 March 2005, by penicillin susceptibility and region



FS Fully sensitive to penicillin, MIC ≤0.03 mg/L.

LS Less sensitive to penicillin, MIC 0.06–0.5 mg/L.

RR Relatively resistant to penicillin, MIC ≥1 mg/L.

PPNG Penicillinase producing *Neisseria gonorrhoeae.*

tested) and New South Wales (84 CMRNG, 26%). Five CMRNG were present in Queensland (2.8% of all Queensland isolates) and Western Australia (4%), four (12%) in South Australia and three (2.1%, again all from Darwin) in the Northern Territory. No PPNG or CMRNG were reported from Tasmania or the Australian Capital Territory.

Ceftriaxone

Fifteen isolates with decreased susceptibility to ceftriaxone (MIC range 0.06–0.12 mg/L) were detected. Thirteen were found in New South Wales and one each in Victoria and Queensland. These strains have been particularly prominent in Japan for quite some time and the decreased susceptibility is associated with the presence of altered *penA* gene resulting in a changed penicillin binding protein 2. All 15 isolates were penicillin resistant, 14 by chromosomal mechanisms and one was a PPNG. Twelve were also quinolone resistant. It is emphasised that no treatment failures have been documented locally when a 250 mg IM dose of ceftriaxone has been used.

Spectinomycin

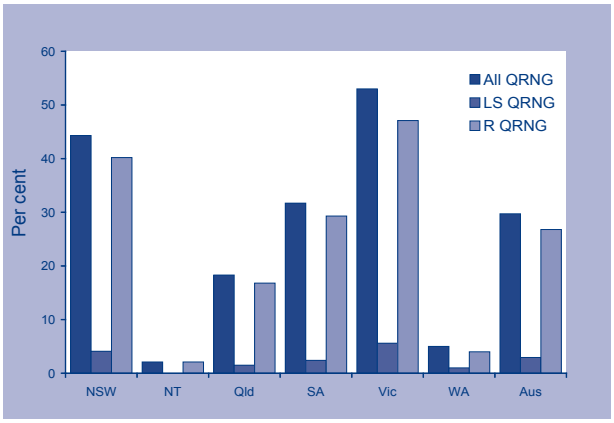
All isolates were susceptible to this injectable agent.

Quinolone antibiotics

The total number (283) and proportion (29.7%) of quinolone resistant *Neisseria gonorrhoeae* (QRNG) were both substantially higher than the corresponding figures in the first quarter of 2004 (188 QRNG, 20.5%) and 2003 (108 isolates, 11.5%). The majority of QRNG (255 of 283, 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 and were detected in all states and territories with the exception of Tasmania (Figure 6). The highest proportion of QRNG was found in Victoria where 85 QRNG represented 53 per cent of all isolates. In New South Wales there were 142 QRNG (44% of isolates), in Queensland 33 (18.3%), in South Australia 13 (32%) and in Western Australia 5 (5%). Two QRNG were detected in the Northern Territory and a single isolate was detected in the Australian Capital Territory.

Figure 6 The distribution of quinolone resistant isolates of *Neisseria gonorrhoeae* in Australia, 1 January to 31 March 2005, 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 (145) and proportion (15.2%) of tetracycline resistance *Neisseria gonorrhoeae* (TRNG) detected also increased when compared with the 2004 (107, 11.7%) figures. TRNG were found in all states and territories and represented between 10 per cent (Queensland) and 23 per cent (South Australia and Victoria) of isolates in mainland states. Six TRNG were present in the Northern Territory, two in Tasmania and one in the Australian Capital Territory.

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. For more information see Commun Dis Intell *2005;29:93.*

Laboratory confirmed cases of invasive meningococcal disease for the period 1 to 30 June 2005, are included in this issue of Communicable Diseases Intelligence *(Table 11).*

Table 11. Number of laboratory confirmed cases of invasive meningococcal disease, Australia, 1 April to 30 June 2005, 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	05			1	2	1	2							2	4
	04			(0)	(4)	(2)	(4)							(2)	(8)
New South Wales	05			17	33	2	9	2	3	3	3	0	1	24	49
	04			(22)	(37)	(5)	(9)	(1)	(2)	(2)	(2)	(5)	(11)	(37)	(61)
Northern Territory	05			2	3	2	2			0	0			4	5
	04			(1)	(6)	(1)	(1)			(1)	(1)			(3)	(8)
Queensland	05	0	0	12	21	2	6	0	0	0	0	0	0	14	27
	04	(1)	(1)	(11)	(23)	(5)	(12)	(1)	(1)	(1)	(1)	(6)	(8)	(19)	(40)
South Australia	05			4	4	0	2							4	6
	04			(5)	(9)	(0)	(0)							(5)	(9)
Tasmania	05			2	2	0	0			0	0	0	0	2	2
	04			(0)	(2)	(0)	(0)			(1)	(1)	(1)	(3)	(2)	(6)
Victoria	05	1	1	8	15	2	3	0	0	0	2	0	1	11	22
	04	(0)	(0)	(18)	(28)	(9)	(9)	(1)	(3)	(0)	(0)	(1)	(2)	(25)	(42)
Western Australia	05			4	9	0	0	1	2					5	11
	04			(8)	(12)	(1)	(3)	(0)	(0)					(9)	(14)
Australia	05	1	1	50	89	9	24	3	5	3	5	0	2	66	126
	04	(1)	(1)	(65)	(121)	(19)	(37)	(4)	(6)	(5)	(5)	(8)	(18)	(102)	(188)

Numbers of laboratory-confirmed diagnoses of invasive meningococcal disease made in the same period in 2004 are shown in parentheses.

Q2 = 2nd quarter.

Ytd = Year to 30 June 2005.

HIV and AIDS surveillance

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

Tabulations of diagnoses of HIV infection and AIDS are based on data available three months after the end of the reporting interval indicated, to allow for reporting delay and to incorporate newly available information. More detailed information on diagnoses of HIV infection and AIDS is published in the quarterly Australian HIV Surveillance Report, and annually in 'HIV/AIDS, viral hepatitis and sexually transmissible infections in Australia, annual surveillance report'. The reports are available from the National Centre in HIV Epidemiology and Clinical Research, 376 Victoria Street, Darlinghurst NSW 2010. Internet: http://www.med.unsw.edu.au/nchecr. Telephone: +61 2 9332 4648. Facsimile: +61 2 9332 1837. For more information see Commun Dis Intell *2005;29:91–92.*

HIV and AIDS diagnoses and deaths following AIDS reported for 1 January to 31 March 2005, as reported to 30 June 2005, are included in this issue of Communicable Diseases Intelligence *(Tables 12 and 13).*

Table 12. New diagnoses of HIV infection, new diagnoses of AIDS, and deaths following AIDS occurring in the period 1 January to 31 March 2005, 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 2005	This period 2004	YTD 2005	YTD 2004
HIV diagnoses	Female	0	5	0	7	0	0	7	5	24	38	24	38
	Male	0	78	0	36	15	0	37	8	174	215	174	215
	Sex not reported	0	0	0	0	0	0	0	0	0	0	0	0
	Total*	0	83	0	43	15	0	44	13	198	254	198	254
AIDS diagnoses	Female	0	1	0	3	0	0	2	0	6	4	6	4
	Male	0	8	0	4	0	0	10	2	24	45	24	45
	Total*	0	9	0	7	0	0	12	2	30	50	30	50
AIDS deaths	Female	0	0	0	0	0	0	0	0	0	1	0	1
	Male	0	5	0	2	0	0	2	1	10	15	10	15
	Total	0	5	0	2	0	0	2	1	10	16	10	16

* Totals include people whose sex was reported as transgender.

Table 13. Cumulative diagnoses of HIV infection, AIDS, and deaths following AIDS since the introduction of HIV antibody testing to 31 March 2005 and reported by 30 June 2005, by sex and state or territory

	Sex	State or territory								
		ACT	NSW	NT	Qld	SA	Tas	Vic	WA	Australia
HIV diagnoses	Female	31	791	18	238	84	8	318	170	1,658
	Male	247	12,779	123	2,474	849	89	4,803	1,111	22,475
	Not reported	0	235	0	0	0	0	22	0	257
	Total*	278	13,833	141	2,721	934	97	5,162	1,288	24,454
AIDS diagnoses	Female	9	225	2	66	31	4	99	35	471
	Male	92	5,181	42	982	387	48	1,883	414	9,029
	Total*	101	5,421	44	1,050	419	52	1,992	451	9,530
AIDS deaths	Female	6	128	1	41	20	2	59	23	280
	Male	71	3,518	26	642	269	32	1,372	289	6,219
	Total*	77	3,655	27	685	289	34	1,439	313	6,519

* Totals include people whose sex was reported as transgender.

National Enteric Pathogens Surveillance System

Since 1980, the National Enteric Pathogens Surveillance System (NEPSS) has collected, analysed and disseminated data on human enteric bacterial infections diagnosed in Australia. These pathogens include Salmonella, E. coli, Vibrio, Yersinia, Plesiomonas, Aeromonas *and* Campylobacter. Communicable Diseases Intelligence *NEPSS quarterly reports include only* Salmonella*.*

Data are based on reports to NEPSS from Australian laboratories of laboratory-confirmed human infection with Salmonella*.* Salmonella *are identified to the level of serovar and, if applicable, phage-type. Infections apparently acquired overseas are included. Multiple isolations of a single* Salmonella *serovar/phage-type from one or more body sites during the same episode of illness are counted once only. The date of the case is the date the primary diagnostic laboratory isolated a* Salmonella *from the clinical sample.*

Note that the historical quarterly mean counts should be interpreted with caution, and are affected by surveillance artefacts such as newly recognised (such as S. Typhimurium 197 and S. Typhimurium U290) and incompletely typed Salmonella.

NEPSS is operated by the Microbiological Diagnostic Unit, Public Health Laboratory, Department of Microbiology and Immunology, University of Melbourne; and is overseen by a Steering Committee of state, territory and commonwealth stakeholders. NEPSS can be contacted at the above address or by telephone: +61 3 8344 5701, facsimile: +61 3 8344 7833 or email joanp@unimelb.edu.au

Scientists, diagnostic and reference laboratories contribute data to NEPSS, which is supported by state and territory health departments and the Australian Government Department of Health and Ageing.

Reports to the National Enteric Pathogens Surveillance System of Salmonella infection for the period 1 April to 30 June 2005 are included in Tables 14 and 15. Data include cases reported and entered by 18 July 2005. Counts are preliminary, and subject to adjustment after completion of typing and reporting of further cases to NEPSS. For more information see Commun Dis Intell *2005;29:93–94.*

Second quarter 2005

The total number of reports to NEPSS of human *Salmonella* infection fell to 1,794 in the second quarter of 2005, 30 per cent less than in the first quarter of 2005. This decline after the summer peak is typical of seasonal trends in the incidence of salmonellosis in Australia. The second quarter count was nine per cent less than the comparable second quarter of 2004 but approximately nine per cent greater than the ten-year historical mean for this period.

During the second quarter of 2005, the 25 most common *Salmonella* types in Australia accounted for 1,168 cases, 65 per cent of all reported human *Salmonella* infections. Nineteen of the 25 most common *Salmonella* infections in the second quarter of 2005 were among the 25 most commonly reported in the first quarter of 2005.

S. Typhimurium 170 (and the related *S*. Typhimurium 108) was the most common serovar/phage type. Two-thirds of cases were from New South Wales. *S*. Typhimurium 197 was less common than in the first quarter (when a large outbreak occurred in Victoria) but counts remain well above historical averages, particularly in Queensland and New South Wales.

Reports of other salmonellae with recent increases and counts that remain above historical averages include *S*. Hvittingfoss (in the eastern States, particularly Queensland), *S*. Aberdeen (in Queensland), and *S*. Corvallis and *S*. Enteritidis 6a (both typically acquired overseas).

S. Typhimurium phage types 135 (widespread) and 9 (south-eastern mainland states) and *S*. Saintpaul (northern states, particularly Queensland) remain very common, each with approximately 100 reports during the quarter.

Acknowledgement: We thank scientists, contributing laboratories, state and territory health departments, and the Australian Government Department of Health and Ageing for their contributions to NEPSS.

Table 14. Reports to the National Enteric Pathogens Surveillance System of *Salmonella* isolated from humans during the period 1 April to 30 June 2005, as reported to 18 July 2005

	State or territory								
	ACT	NSW	NT	Qld	SA	Tas	Vic	WA	Australia
Total all *Salmonella* for quarter	30	483	78	635	124	36	242	166	1,794
Total contributing *Salmonella* types	15	92	37	115	47	11	83	68	213

Table 15. Top 25 *Salmonella* types identified in Australia, 1 April to 30 June 2005, by state or territory

National rank	*Salmonella* type	State or territory								Total 2nd quarter 2005	Last 10 years mean 2nd quarter	Year to date 2005	Year to date 2004
		ACT	NSW	NT	Qld	SA	Tas	Vic	WA				
1	*S.* Typhimurium 170	7	129	0	20	2	1	14	5	178	47	343	379
2	*S.* Typhimurium 135	1	39	0	19	9	12	10	15	105	133	234	349
3	*S.* Saintpaul	1	4	8	69	2	1	6	11	102	87	259	234
4	*S.* Typhimurium 9	5	56	2	4	6	0	26	1	100	115	268	246
5	*S.* Hvittingfoss	5	9	2	43	0	0	16	2	77	24	132	106
6	*S.* Typhimurium 197	0	21	0	37	0	0	8	1	67	11	449	142
7	*S.* Virchow 8	1	10	3	45	1	0	1	0	61	50	163	231
8	*S.* Birkenhead	0	18	0	39	0	0	1	0	58	57	129	173
9	*S.* Aberdeen	0	4	1	41	0	0	1	0	47	30	112	72
10	*S.* Chester	1	5	4	21	5	0	3	6	45	40	118	127
11	*S.* Infantis	0	14	2	2	11	1	9	3	42	30	94	90
12	*S.* Muenchen	0	6	0	13	3	0	4	11	37	33	101	73
13	*S.* Waycross	0	4	0	29	1	0	0	0	34	28	78	89
14	*S.* Typhimurium RDNC	2	5	1	4	4	0	2	4	22	18	54	57
15	*S.* Enteritidis 6a	0	6	0	3	1	0	4	7	21	3.6	43	24
16	*S.* Stanley	0	7	1	3	1	0	2	5	19	10	34	33
17	*S.* Anatum	0	2	2	5	0	0	2	7	18	24	37	61
18	*S.* Typhimurium 12	0	9	0	1	1	0	2	5	18	18	76	188
19	*S.* Mississippi	0	2	0	0	0	13	3	0	18	18	49	53
20	*S.* Typhimurium untypable	0	4	0	3	0	0	6	5	18	16	33	11
21	*S.* Reading	0	10	1	4	0	0	3	0	18	6	29	25
22	*S.* Corvallis	1	5	0	1	4	0	6	0	17	1.2	44	23
23	*S.* Virchow 25 var 1	0	1	0	15	0	0	0	0	16	0	22	0
24	S Agona	0	2	0	7	4	0	1	1	15	16	30	52
25	*S.* Ball	0	0	15	0	0	0	0	0	15	10	28	35

Overseas briefs

World Health Organization Disease Outbreak News

This material has been summarised from information provided by the World health Organization (http://www.who.int/csr/don/en/). A link to this site can be found under the 'Related communicable diseases surveillance sites' on the Communicable Diseases Australia section of the Australian Government Department of Health and Ageing website (www.health.gov.au/cda)

Poliomyelitis in Indonesia

1 July 2005

On 30 June 2005, one new polio case was confirmed in Indonesia, bringing the total number of cases to 66. The new case is the first from Lampung Province on the island of Sumatra. The 3-year-old girl had onset of paralysis on 4 June.

Both this case and a previous case from Central Java are from outside the area where an emergency 'mop-up' campaign was held from 31 May to 2 June, covering the provinces of West Java, Banten and Jakarta, to reach 6.4 million children under the age of five years. A second round of vaccinations was completed on 29 June.

A large outbreak response immunisation targeting 78,000 children aged less than five years was held from 26 June around the case in Central Java. Lampung and Central Java will be included in the next phase of the large-scale immunisation campaigns which will start from August.

Avian influenza – situation in Viet Nam

30 June 2005

At the request of the Ministry of Health, World Health Organization (WHO) sent a team of international experts to Viet Nam last week to assess laboratory and epidemiological data on recent cases and to determine whether the present level of pandemic alert should be increased. Team members were drawn from institutes in Australia, Canada, Hong Kong SAR, Japan, the United Kingdom, and the United States of America having extensive experience in the testing of avian influenza viruses in human clinical specimens.

The team completed its work and submitted its preliminary findings to the government. The team found no laboratory evidence suggesting that human infections are occurring with greater frequency or that the virus is spreading readily among humans. The current level of pandemic alert, which has been in effect since January 2004, remains unchanged.

Some reports now circulating suggest that WHO has downgraded its assessment of the pandemic threat. These reports are unfounded. The experts were specifically asked to search for evidence that could substantiate concerns raised first at a WHO consultation of international experts held in Manila at the beginning of May. That consultation considered suggestive findings, largely based on epidemiological observations, that the H5N1 virus had changed its behaviour in ways consistent with an improved, though not yet efficient, ability to spread directly from one human to another. The specific epidemiological observations considered included milder disease across a broader age spectrum and a growing number of clusters of cases, closely related in time and place.

More recently, testing of clinical specimens by international experts working in Viet Nam provided further suggestive evidence of more widespread infection with the virus, raising the possibility of community-acquired infection. These findings have not been confirmed by the present investigative team.

Firm evidence of improved transmissibility would be grounds for moving to a higher level of pandemic alert. Because of the huge consequences of such a change, WHO is following a cautious approach that combines heightened vigilance for new cases with immediate international verification of any suggestive findings.

Because the detection of H5N1 in clinical specimens is technically challenging and prone to errors, members of the investigative team took sophisticated laboratory equipment with them to Hanoi for on-site testing. Tests were performed using WHO-approved reagents and primers.

While these first results are reassuring, further retesting of clinical specimens will continue over the next few weeks to provide the most reliable possible foundation for risk assessment.

Marburg haemorrhagic fever in Angola

7 June 2005

As of 5 June 2005, the Ministry of Health in Angola has reported 423 cases of Marburg haemorrhagic fever. Of these cases, 357 were fatal. The vast majority of cases have occurred in Uige Province, where 412 cases and 346 deaths have been reported.

The number of new cases being reported in Uige municipality has declined considerably, with only one new confirmed case detected in the past week. This case was a recognised contact who was under follow-up. For comparison, during the peak of the outbreak, which occurred in late March and April, 30 to 40 new cases were being reported weekly.

Alerts to potential cases continue to be received and investigated, indicating that vigilance remains high.

Ebola haemorrhagic fever in the Republic of the Congo

16 June 2005

From 25 April to 16 June 2005, a total of 12 cases of Ebola haemorrhagic fever (1 laboratory-confirmed and 11 epidemiologically linked) including nine deaths has been reported in Etoumbi and Mbomo in Cuvette Ouest Region. The last reported death occurred on 26 May.

Eleven contacts of this last reported death have been followed for 21 days, the maximum incubation period. None of these people have been infected.

The Ministry of Health and the WHO Regional Office for Africa are continuing to strengthen infection control and raise awareness about the disease among the population in the affected districts.

Meningococcal disease in India – update 4

14 June 2005

As of 8 June 2005, the cumulative total is now 405 cases with 48 deaths (CFR=11.9%). Three hundred and fourteen cases have been discharged from hospital.

Control measures are underway including contact tracing, chemoprophylaxis of household contacts, and immunisation of high risk groups. Serogroup A has been confirmed by the National Institute of Communicable Diseases.

Public education, surveillance, vaccination of high risk population and chemoprophylaxis for close contacts within 48 hours of case detection continues. Adjacent districts and states have been alerted on the need to be vigilant for any suspected case and to take appropriate public health actions.

WHO is working closely with the national authorities and providing technical support to the health authorities in the form of guidelines and tools on meningococcal disease. WHO is providing inputs to the technical working group to assist with surveillance, early detection, laboratory testing, case management, prevention and control. WHO is assisting with the epidemiological analysis and in improving preparedness and response.

ProMED-mail

This material has been summarised from information provided by ProMED-mail (www.fas.org/promed/). A link to this site can be found under the 'Related communicable diseases surveillance sites' on the Communicable Diseases Australia section of the Australian Government Department of Health and Ageing website (www.health.gov.au/cda)

Clostridium difficile, *increased virulence – UK*

Source: The Guardian 7 June 2005 [edited]

Public health experts are consulting hospitals in the United States of America and Canada for advice on tackling a virulent strain of bacteria that is thought to be responsible for 12 deaths at Stoke Mandeville hospital over the past two years.

Staff at the Health Protection Agency say the particular strain of *Clostridium difficile* appears to be similar to one found in some hospitals in North America. *C. difficile* is not rare, new or dangerous under most circumstances. It is carried harmlessly in the gut of half of all children under the age of two and substantial numbers of adults. The 12 deaths have to be set in the context of more than 43,000 reported infections in 2004. But the strain persisting at Stoke Mandeville hospital produces toxins which can cause problems for the very elderly and frail.

Unlike the so-called superbug MRSA (methicillin-resistant *Staphylococcus aureus*), *C. difficile* is not resistant to antibiotics. However, Mark Enright, a microbiologist and senior research fellow at Bath University said it is still the use of antibiotics that causes it to become a problem. 'It is a common

component of the gut, in balance with all the other bacteria with it which are helpful—we can't digest food without them. However *C. difficile* forms spores like hardy seeds which are not killed when somebody has a long course of antibiotics and some strains have toxins that they excrete,' he said.

This toxin production results in diarrhoea. In very elderly people who are already weak and frail because of illness, complications from damage to the gut or the dehydration caused by diarrhoea could be a factor in their death.

The particular problem with *C. difficile* is that the spores are very hard to get rid of from the ward. The alcohol wipes now used by doctors and nurses to prevent the spread of most bacteria do not work. Surfaces have to be cleaned with bleach and hands should be washed with soap and water.

Influenza B virus – New Zealand

Source: Public Health Directorate, New Zealand Ministry of Health, 22 June 2005 [edited]

New Zealand is currently experiencing an epidemic of influenza B virus infection. Both influenza B Shanghai-like virus and influenza B Hong Kong-like virus have been isolated. However, influenza B Hong Kong-like virus is currently the predominant strain. Children and young people are predominantly affected with absenteeism rates in schools in some areas of greater than 20 per cent.

Currently, three deaths have been identified in association with this epidemic: a child who developed Reye syndrome, (this child was on aspirin for another condition); an otherwise fit and well adolescent who developed *Staphylococcus aureus* pneumonia and septicaemia; and an otherwise fit and well child who developed *Staphylococcus aureus* pneumonia and septicaemia.

Two of these deaths are under investigation by New Zealand coroners. The viral isolates in all three cases have been identified as an influenza B Hong Kong-like strain. The current Southern Hemisphere vaccine contains an influenza B Shanghai-like strain.

Avian influenza, human – Indonesia

Source: Washington Post Foreign Service, 15 June 2005 [edited]

A farm worker in eastern Indonesia has tested positive for avian influenza virus, making him the country's first human case of the virus infection that has already killed at least 54 people elsewhere in South East Asia, health officials in Indonesia said on 15 June 2005.

The worker from southern Sulawesi is healthy and currently shows no symptoms of illness but two tests at a Hong Kong laboratory confirmed that he had been infected by avian influenza virus, health officials said. The laboratory results make Indonesia the fourth country to register a human case of avian influenza, which international health experts warn could easily undergo genetic change, sparking a global pandemic.

Since 2003, the highly lethal disease has struck chickens, quail and other birds in 18 Indonesian provinces on seven islands, prompting the government to order a massive campaign to vaccinate poultry against the virus. Indonesian health experts, however, have sought to ease public anxiety about the outbreak over the last year by saying the local virus was slightly different from the strain in other Asian countries and had demonstrated no capability to infect people.

The farm worker was initially tested in late March after the epidemic spread to Sulawesi, killing at least 25,000 chickens. That outbreak prompted officials to limit the transfer of poultry off the island and take blood samples from labourers, veterinarians and others exposed to sick chickens. In total, 81 people were tested and all but one of the samples came back negative, officials said.

Efforts to complete a second round of testing in Hong Kong were prolonged in part because the farm worker had left his job and health investigators had to track him back to his home village elsewhere on the island. The second test, finally completed earlier this month, confirmed that the labourer had been infected by bird flu but the concentration of antibodies was relatively low, officials said. That finding meant the worker was no longer carrying the virus but it was impossible to determine how long ago he had been infected.

Since late 2003, more than 100 people have been infected by avian influenza in Viet Nam, Thailand and Cambodia. In Viet Nam, where the outbreak is most serious, government health officials have previously reported at least five cases in which people had the disease but showed no symptoms. Klaus Stohr, head of the World Health Organization's influenza program, said last month it is not unprecedented for otherwise healthy poultry workers to test positive for avian influenza. During a 1997 outbreak in Hong Kong, about 10 per cent of workers in live poultry markets tested positive for the virus, health officials said.

Variant Creutzfeldt-Jakob disease – Portugal and France

Source: Agence France Presse report, 11 June 2005 [edited]

Portugal announced its first suspected case of variant Creutzfeldt-Jakob disease (vCJD), while France said it had identified its 13th case of the degenerative brain ailment.

vCJD is a human form of bovine spongiform encephalopathy (BSE), caused by a rogue protein that proliferates in the brain, turning it spongy. A total of 177 people have died or been diagnosed with the fatal condition, according to official data.

So far 150 people have died of vCJD in Britain, where another six people who have contracted the disease are still alive, according to figures posted on the official British vCJD website.

There have been two cases in Ireland, with single cases reported in Canada, Italy, Japan, the Netherlands and the United States of America. Britain was the epicentre of the BSE outbreak that occurred in the late 1990s. Its suspected source was cattle feed that came from cows with brain disease. Experts believe the pathogen leapt the species barrier to humans through the consumption of contaminated beef.

According to the latest figure compiled by the European Union and the OIE, Portugal ranks third in the world in terms of total number of BSE-affected cattle (949) after the UK (184,138), and Ireland (1,470). France ranks fourth with 946 cases of BSE. In the current year the UK, Ireland and Portugal reported 126, 29, and 17 cases of BSE respectively.