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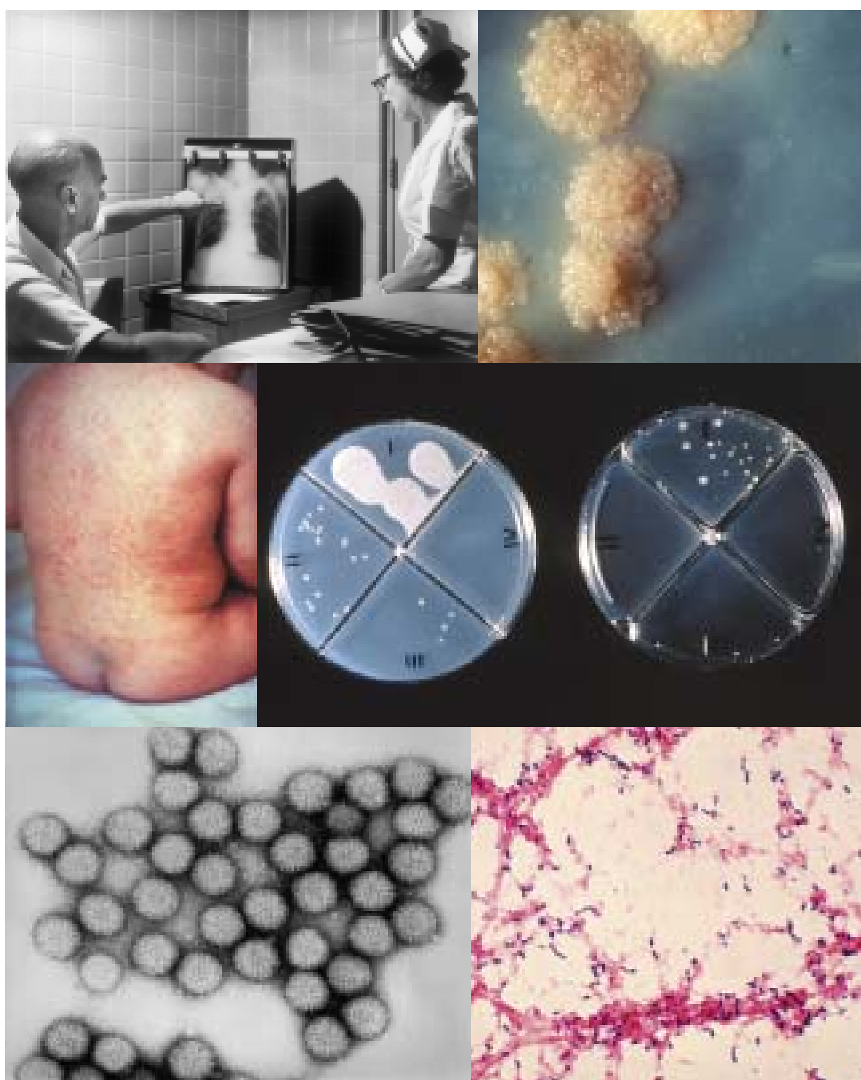
Communicable Diseases Intelligence

Communicable Diseases Network Australia

A national network for communicable diseases surveillance



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Images sourced from the Centers for Disease Control and Prevention Public Health Image Library, courtesy of the Centers for Disease Control and Prevention
Clockwise from top left: Chest x-ray after positive tuberculosis skin test (Supplied by Charles Farmer); *Mycobacterium tuberculosis* culture (provided by Dr George Kubica); *Mycobacterium tuberculosis* drug susceptibility test (CDC); Photomicrograph of *Streptococcus pneumoniae* bacteria (provided by Dr Mike Miller); Transmission electron micrograph of intact rotavirus particle (provided by Dr Erskine Palmer); and Rubella rash on child's back (CDC).

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Editorial: A strategy for the surveillance of antimicrobial resistance in Australia

Rebecca Hundy and Paul Roche
Surveillance and Epidemiology Section, Department of Health and Ageing, Canberra

Antibiotics have underpinned the success of modern infectious disease control for the last half-century. The emergence of resistance in almost all major human pathogenic and many commensal bacteria to antibiotics now presents an enormous challenge to disease control.

Emerging resistance is undermining previously effective treatment regimes worldwide. Gonococcal disease is one example of how emerging resistance has necessitated changes in standard treatment programs from cheap oral antibiotics to more expensive agents. Penicillin, which remains a recommended treatment for gonorrhoea in many countries, is becoming less effective as a treatment due to increasing resistance. In the World Health Organization (WHO) Western Pacific Region in 2000, the proportion of isolates resistant to penicillin varied from region to region, ranging from over 90 per cent in Korea to 7.9 per cent in New Zealand.[1] Of particular concern is the increasing number of gonococcal isolates showing an altered susceptibility to the third-generation cephalosporins. Third-generation cephalosporins have an increasing importance in the treatment of gonococcal disease as the incidence of resistance to penicillins and quinolones increases. As demonstrated in the WHO Western Pacific Gonococcal Antimicrobial Surveillance Programme report in this issue of *Communicable Diseases Intelligence*, *Neisseria gonorrhoeae* isolates with an altered susceptibility to third generation cephalosporins continue to be reported from several countries in the region, including Australia.[2]

The potential economic costs of antimicrobial resistance (AMR) to the healthcare system are substantial, given that resistance necessitates the use of more expensive antibiotics in treatment, the administration of multiple courses of antibiotics, and increases in the length of hospital stay. In the United States of America, the health care costs associated with the treatment of infections caused by antibiotic resistant bacteria have been estimated between 4 and 5 billion dollars annually.[3] The actual costs of resistance can be far greater, given that non-resource costs include increased morbidity and mortality, and overall poorer health outcomes.

A link between the use of antimicrobial agents as growth promotants in food-producing animals and the emergence of antimicrobial resistance in Australia was confirmed by the findings of the Joint Expert Advisory Group on Antibiotic Resistance (JETACAR). The JETACAR report made recommendations for the future management of AMR in Australia, the intent of which were strongly supported by the Australian Government.

Several of the recommendations made by the JETACAR identified the need for a national program for the surveillance of antimicrobial resistance in Australia in animals, food and humans. While there are a number of AMR surveillance systems in operation in various jurisdictions in Australia, at the national level, these activities are fragmented and not comprehensive. *The Strategy for AMR Surveillance in Australia* (the Strategy) was developed to coordinate current surveillance activities, to address current gaps in surveillance, and to provide a centralised point for the collation and reporting of national AMR data.

As an initial step, a Central Coordinating Unit (CCU) for the implementation of the Strategy has been established in the Surveillance and Epidemiology Section at the Australian Government Department of Health and Ageing in Canberra. Over the next two years, in consultation with existing AMR surveillance networks, the CCU will collect, collate, evaluate and report on trends in antimicrobial resistance in Australia. Emerging resistance threats, populations and settings of risk and gaps in surveillance will by identified. A plan for an on-going comprehensive national surveillance system for AMR in animals, food and humans will be developed.

Nationally collected surveillance data is essential to improve the understanding and impact of antimicrobial resistance. In Australia, AMR surveillance data will be used to more accurately inform revisions of the *Therapeutic Guidelines: Antibiotic*. Accurate resistance information guides disease control be ensuring that diseases, like gonorrhoea, are treated using antibiotics that are more likely to be effective.

Through a greater understanding of trends in resistance, steps to contain AMR in Australia may be achieved.

Further details about the Strategy can be obtained from:

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An abbreviated version of the Strategy is included in this issue of Communicable Diseases Intelligence. Full copies of the Strategy can be obtained from: http://www.health.gov.au/pubhlth/strateg/jetacar/reports.htm

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National Arboviral and Malaria Surveillance Website

A new website for arbovirus surveillance has been established under the auspices of the Australian Government Department of Health and Ageing. The National Arboviral and Malaria Surveillance website was launched in early November 2003 and can be accessed at: http://www.health.gov.au/arbovirus.

The website is a joint Commonwealth and State initiative and has been designed to provide information on arboviral disease in Australia to assist in the control of arboviral disease and malaria.

The website aims to increase public awareness of the risks of mosquito-borne disease, and to facilitate the dissemination of related surveillance data.

Strategy for antimicrobial resistance surveillance in Australia

Introduction

The importance of surveillance* in combating and managing antimicrobial† resistance (AMR) is recognised as an important component of the *World Health Organization (WHO) Global Strategy for Containment of Antimicrobial Resistance*, 2001. There is good evidence that surveillance is a cost-effective infection control strategy.[1,2] Appropriate surveillance provides vital information for the targeting of interventions, and measures success or failure of these interventions. Surveillance enables early detection and intervention, and can therefore reduce the extent and severity of outbreaks. This in turn should reduce infection-related costs, making funds available for other healthcare activities. Short-term investment, therefore, leads to longer-term gains and overall savings.

Many healthcare facilities, professional groups, networks and surveillance programs already have extensive experience from which other parties can learn. Sharing of experiences and knowledge of successful interventions (e.g. improved infection control practices; antibiotic restriction policies; new methods and techniques; and surveillance findings) allows institutions to build upon the successes of others and avoid duplication. Better mechanisms are needed for reporting surveillance information at local, state/territory and national levels that will increase awareness and access to information. It is also vital that surveillance information is incorporated into updates of best practice guidelines and adopted by medical and veterinary prescribers.

A national surveillance strategy provides an opportunity for consolidating and building upon existing, high quality, surveillance activities in Australia. This requires strengthening of existing networks and systems, and a re-focusing of priorities towards data for action at the local, state/territory and national levels.

While controlling antimicrobial resistance impacts human health, control requires a cross-sectoral approach, engaging human and animal health, industry and a range of other stakeholders. This Strategy provides a framework for how these diverse groups can provide evidence for action to control AMR.

Background

Joint Expert Technical Advisory Committee on Antibiotic Resistance

The Joint Expert Technical Advisory Committee on Antibiotic Resistance (JETACAR) was established in April 1998 to provide independent expert scientific advice on the threat posed by antimicrobial resistant bacteria. The JETACAR released its report in September 1999, making 22 recommendations for an antimicrobial resistance management program covering:

- regulatory controls;
- monitoring and surveillance;
- infection prevention strategies;
- education; and
- research.

The Australian Government released its response to recommendations of the JETACAR report in August 2000. The government response strongly supported the intent of the JETACAR report and outlined the mechanisms for implementing the recommendations.

Coordination and implementation

To facilitate the implementation of the JETACAR recommendations the Commonwealth Government established the Commonwealth Interdepartmental JETACAR Implementation Group (CIJIG) comprising technical experts and senior representatives from:

* Surveillance in a broad sense, and for the purpose of this document, is defined as the ongoing and systematic collection, analysis and interpretation of outcome-specific data essential to the planning, implementation, and evaluation of public health practice, closely integrated with the timely dissemination of these data to those who need to know. The final link of the surveillance chain is the application of these data to the control and prevention of human disease and injury.[3]

† For the purpose of the surveillance strategy, the term antimicrobial resistance (AMR) will be used however it will specifically refer to resistance to antibiotics. Antibiotics are defined as antibacterial agents (including ionophores) but not including antiprotozoals, antifungals, antiseptics, disinfectants, antineoplastic agents, antivirals, immunologicals, direct-fed microbials or enzyme substances.[4] Notwithstanding this, similar considerations about monitoring utilisation and surveying resistance may be applied appropriately to other antimicrobial agents.

- the Australian Government Department of Health and Ageing (DoHA);
- the Australian Government Department of Agriculture, Fisheries and Forestry;
- the Australian Pesticides and Veterinary Medicines Authority (APVMA, formerly known as the National Registration Authority for Agricultural and Veterinary Chemicals);
- the Therapeutic Goods Administration (TGA);
- Food Standards Australia New Zealand; and
- the National Health and Medical Research Council (NHMRC).

The Australian Health Ministers' Conference (AHMC) and the Primary Industries Standing Committee each appointed a taskforce to facilitate and monitor the implementation of the JETACAR recommendations and to provide policy advice to CIJIG.

The AHMC JETACAR Taskforce released its final report in November 2000. In summary, it recommended that:

- the Expert Advisory Group on Antimicrobial Resistance continue to provide scientific and policy advice on antimicrobial resistance issues;
- an AMR surveillance network implements a national surveillance strategy; and
- ongoing implementation of all JETACAR recommendations, including those related to surveillance, to be coordinated by CIJIG.

Expert Advisory Group

The Expert Advisory Group on Antimicrobial Resistance (EAGAR) was established under the auspices of the NHMRC to provide independent scientific and policy advice on antimicrobial resistance and related matters to national, state and territory governments and regulatory authorities.

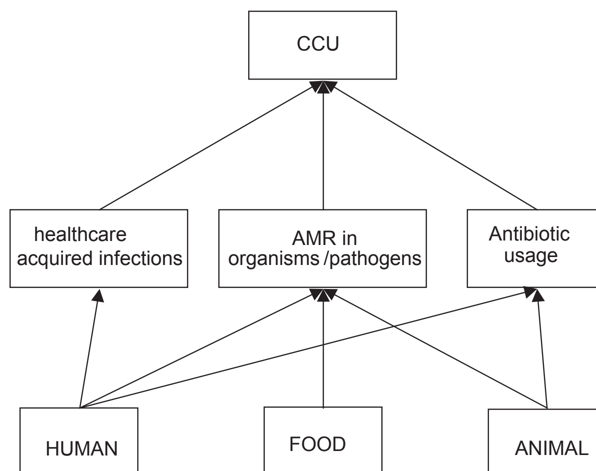
A strategy for AMR surveillance in Australia

This document is a national Surveillance Strategy to address both JETACAR recommendations relating to monitoring and surveillance and an additional recommendation relating to surveillance of antibiotic usage (Table 1).

A key component in the development of a cross-disciplinary coordinated approach to antimicrobial resistance in Australia is the development of a central coordinating unit (CCU), at DoHA. Project officer(s) will work as part of the Surveillance and Epidemiology Section, DoHA on the CCU development project. Following the development project, the CCU functions will be absorbed into the normal business of the Surveillance and Epidemiology Section, Australian Government Department of Health and Ageing.

The CCU will act as a central site for the collation of national surveillance data (Figure). A variety of agencies will engage in the development and implementation of specific action plans. It is the responsibility of each sector and agency to examine the Strategy and decide how best to meet the national objectives by building upon current initiatives and refining these to allow best possible utilisation of data and information.

Figure. Areas of surveillance collated by the Central Coordinating Unit



A number of overarching principles will guide the strategic approach taken when establishing a national network of surveillance activities. The CCU will:

1. recognise and build on existing local, state and territory, and national surveillance and monitoring systems rather than establish new ones;

Table 1. JETACAR recommendations addressed in the Strategy

No.	Recommendation
10	Development of a comprehensive surveillance system for antimicrobial resistant bacteria and resistance genes in humans and animals. The surveillance system should include medical (including nosocomial), food-producing animal and veterinary areas with particular emphasis on the establishment of food-chain and environmental connections.
11	Monitoring and audit of antibiotic usage
14	Surveillance of hospital acquired infections

Table 2. Agencies involved in surveillance activities

Area of surveillance activity	Primary agency
Antimicrobial resistance in community-acquired infections (humans)	Australian Government Department of Health and Ageing (Communicable Diseases Branch).
Antimicrobial resistance in animals	Australian Government Department of Agriculture, Fisheries and Forestry — Australia
Antimicrobial surveillance in animal-derived foods	Australian Government Department of Health and Ageing (Food and Environmental Health Branch) through OzFoodNet
Surveillance of antimicrobial resistance in healthcare acquired infections	Australian Government Department of Health and Ageing (Communicable Diseases Branch) with the Safety and Quality Council.
Surveillance of antibiotic usage in humans and animals	Australian Government Department of Health and Ageing through the Therapeutic Goods Administration and Australian Pesticides and Veterinary Medicines Authority.

2. work towards nationally consistent standards for national AMR surveillance data; and
3. ensure security and confidentiality of data, and not identify individuals, companies or healthcare establishments. This will be guided by the Australian Health Ministers' Advisory Council National Health Privacy Code.

Aim

This Strategy aims to address recommendations 10, 11 and 14 of the JETACAR report. More specifically, the Strategy aims to:

1. identify priorities for action that will strengthen surveillance at the local, state/territory and national levels;
2. outline surveillance needs;
3. strengthen communication and reporting mechanisms to ensure maximum utilisation of information; and
4. raise awareness of surveillance and antimicrobial resistance.

Strategy outcomes

The surveillance data generated will provide evidence to evaluate policies and set priorities to manage antimicrobial resistance, including:

- antibiotic therapeutic and prudent use guidelines and treatment regimes to improve public health outcomes;
- assessment of risks to public health to form the basis of risk management policy;
- infection control guidelines, hygiene measures and antibiotic restriction policies; and
- the cost-effectiveness of these interventions.

The surveillance data generated will provide data to inform:

- education strategies for medical and veterinary prescribers, hospital staff, food-animal producers, food handlers, industry and consumers;
- risk assessments of new and existing antibiotics for use in humans and animals; and
- prudent use of antimicrobials by medical practitioners, veterinarians and industry, to prolong the efficacy of these products.

Implementation of the Strategy

Introduction: a staged approach

Establishing a comprehensive surveillance system across the human and animal sectors is not a task that can be fully implemented immediately. The plan therefore sets out a staged approach for implementation. Stage 1 and 2 will incorporate the CCU development project.

Stage one – Consultation, planning and implementation

Stage one is intended to acknowledge and draw upon the work that is taking place at the state/ territory and local level. Stage one will include:

- implementation of a central coordinating unit that will be responsible for collection and consolidation of nationally consistent information encompassing the areas of antimicrobial resistance in humans, animals and food, including healthcare acquired infections and antimicrobial usage;

- consultation with key stakeholders to identify and evaluate existing antimicrobial resistance surveillance systems and making recommendations for developing longer term mechanisms to acquire national data on antimicrobial resistance;
- reporting on existing systems;
- identifying local and state/territory activities that can serve as models or developmental projects for possible linking and/or adoption nationally; and
- development and implementation of pilot surveillance programs for antimicrobial resistance, as appropriate.

Stage two – Correlation and improving data systems

It is proposed that stage two focus on:

- correlation of data from the diverse surveillance systems;
- assessment of the surveillance activities to identify gaps and make recommendations for improvement;
- achieving national consensus on national minimum datasets and consistent standards, definitions and methodology; and
- collecting antimicrobial usage data at the state/territory and national level.

Stage three – Evaluation and future planning

It is expected that stage three will involve:

- full implementation and establishment of an ongoing, national surveillance program incorporating local and state/territory surveillance data and data from established surveillance schemes;
- integration of human and animal surveillance systems;
- evaluation of the integrated national surveillance program;
- production and supply of data and information to users to provide a clearer national view of national antimicrobial resistance trends; and
- using the data collected to inform future policy and strategic development and interventions.

Aims

The overall aim is to develop a national surveillance system utilising, where available, existing programs to provide reliable information on antimicrobial resistance in Australia. Antimicrobial resistance surveillance will provide information on the magnitude and distribution of resistant organisms in Australia to identify changing trends and emerging resistance.

Goals

1. Bring together national data collected by existing systems to meet immediate data needs.
2. Develop appropriate pilot programs where systems do not exist.
3. Develop nationally consistent standards of data quality.
4. Develop and implement protocols for appropriate data collection for national antimicrobial resistance surveillance.
5. Report on national prevalence of and trends in antimicrobial resistance.

Processes

The CCU will facilitate the establishment of both a Human Health and an Animal Health Reference Network, and articulate the role of these networks across the four areas of surveillance.

Membership of the Human Health Reference Network may include:

- federal, state and territory health department representatives;
- Communicable Diseases Network Australia;
- Public Health Laboratory Network; and
- representatives from existing surveillance networks.

Membership of the Animal Health Reference Network may include:

- federal, states and territories Primary industry representatives;
- food-animal producer representatives;
- veterinary testing laboratories; and
- individuals with specific expertise.

The CCU development project will proceed in implementing stages 1 and 2 of the Strategy by:

Stage one of Strategy: Consultation, planning and implementation

- developing relationships with key stakeholders in AMR surveillance;
- developing draft surveillance action plan for AMR in humans, and working with other agencies to further develop plans for AMR in animals, healthcare acquired infections, and antimicrobial usage;
- identifying, strengthening and working with existing surveillance networks;
- identifying data gaps to be filled by pilot surveillance programs; and
- collating and reporting on existing antimicrobial resistance data (as appropriate).

Stage two of Strategy: Correlation and improving data systems

- developing consistency in data formats;
- developing consistent case definitions;
- developing consistent laboratory methods and diagnoses;
- developing protocols for data reporting, analysis and interpretation;
- evaluating existing AMR surveillance systems; and
- evaluating the need for a comprehensive surveillance system for AMR in humans and animals.

Surveillance of antimicrobial resistance in community acquired infections

Background

Surveillance of antimicrobial resistant organisms in community-acquired infections is essential to provide insight into the levels of resistant bacteria and their trends. Australia already has many systems in place at the local, state, and national level for the surveillance of antimicrobial resistant organisms in humans. The data collected via these systems are collected for many different purposes, however, much of the data remains at the local level. There is no coordination of these programs, but they form a solid basis for an integrated national program for surveillance of antimicrobial resistance in bacteria of medical importance.

Some of the more well established surveillance systems currently collecting antimicrobial resistance data nationally from hospitals and the community include: the Australian Group on Antimicrobial Resistance (AGAR), the Australian Gonococcal Surveillance Programme, the Australian Meningococcal Surveillance Programme, the Australian Mycobacterium Reference Laboratory Network (AMRLN) and the National Enteric Pathogen Surveillance Scheme (NEPSS).

Aim

To measure the prevalence of and trends in antimicrobial resistance in community-acquired organisms causing significant human diseases in Australia.

Primary agency

Communicable Diseases Branch, Population Health Division, Department of Health and Ageing

Stakeholders

State and territory health department representatives and Communicable Diseases Network Australia

Australian Group on Antimicrobial Resistance

National Enteric Pathogens Surveillance Scheme

National Centre for Immunisation Research and Surveillance of Vaccine Preventable Diseases

National Neisseria Network (NNN), incorporating the Australian Meningococcal Surveillance Programme and the Australian Gonococcal Surveillance Programme

OzFoodNet

Australian Mycobacterium Reference Laboratory Network

National Notifiable Diseases Surveillance System

Goals

1. Create links with key data collections of antimicrobial resistance in human pathogens.
2. Evaluate data quality in national data collections and identify deficiencies.
3. Estimate the prevalence of resistant bacteria in human pathogens and detect the emergence of new antimicrobial resistance patterns with assistance of surveillance networks and EAGAR.
4. Report national data on the prevalence of antimicrobial resistance in humans.

Processes

Table 3. Processes for reaching goals of AMR surveillance in human pathogens

Goal	Process
1. Establish links with data collections	Identify stakeholders; CCU coordinator to visit stakeholders and attend meetings, where appropriate.
2. Improve data quality	Evaluate data quality; identify gaps and deficiencies and work with stakeholders to improve data quality.
3. Estimate AMR prevalence	Summarise AMR prevalence for key diseases and antimicrobials from existing data.
4. Reporting	Produce reports in consultation with stakeholders on trends in AMR in pathogens of interest and establish a reporting cycle and format.

Organisms and antimicrobials for surveillance

Table 4. Organisms and antimicrobials proposed as national priorities for human antimicrobial resistance surveillance in human pathogens

Organism	Passive surveillance	Targeted surveillance (current group)	Minimum antibiotics or classes*
Streptococcus pneumoniae	Yes	Invasive only i.e. blood/ cerebrospinal fluids (none)	Benzylpenicillin, 3rd generation cephalosporins, erythromycin, tetracycline, cotrimoxazole/ trimethoprim
Haemophilus influenzae	Yes	Invasive type b only i.e. blood/ cerebrospinal fluids (none)	Ampicillin, cefaclor, tetracycline, cotrimoxazole
Moraxella catarrhalis	Yes	No	Ampicillin, erythromycin, tetracycline, cotrimoxazole
Staphylococcus aureus	Yes	Yes (AGAR)	Benzylpenicillin, methi/oxacillin, erythromycin, tetracycline, gentamicin, cotrimoxazole/ trimethoprim (plus vancomycin, rifampicin, fusidic acid if methi/oxacillin resistant)
Streptococcus pyogenes	Yes	No	Benzylpenicillin, erythromycin
Escherichia coli	Yes	Yes (AGAR)	Ampicillin, 1st generation cephalosporins, 3rd generation cephalosporins, amoxycillin-clavulanate, gentamicin, fluoroquinolones, cotrimoxazole/trimethoprim
Salmonella species	No	Yes (NEPSS)	Ampicillin, 3rd generation cephalosporins, fluoroquinolones, cotrimoxazole/trimethoprim, chloramphenicol
Campylobacter species	No	Yes (NEPSS)	Fluoroquinolones, gentamicin
Neisseria gonorrhoeae	No	Yes (NNN)	Benzylpenicillin, 3rd generation cephalosporins, fluoroquinolones, tetracycline, spectinomycin
Neisseria meningitidis	No	Yes (NNN)	Benzylpenicillin, 3rd generation cephalosporins, fluoroquinolones, rifampicin
Mycobacterium tuberculosis	No	Yes (AMRLN)	Isoniazid, rifampicin, ethambutol, pyrazinamide

Outcomes

- Estimation of the prevalence and detection of trends of AMR in invasive pneumococcal disease, *Haemophilus influenzae* type b, *Salmonella* causing gastroenteritis, meningococcal disease, gonorrhoea, *Campylobacter* and tuberculosis.
- Improvement in data quality on AMR infections in humans and the development of a national data set on the prevalence of AMR in humans.
- Regular reporting of data on AMR infections in humans.
- To have a national data set on the prevalence of AMR in humans. These data can be used:
  - in risk analysis to determine the risk to human health;
  - to detect the emergence of particular phenotypes of AMR;
  - to detect trends in AMR;
  - to identify the need for particular interventions and to assess the impact of interventions; and
  - to provide a basis for policy recommendations for public health.

Surveillance of antimicrobial resistance in healthcare acquired infections

Background

The incidence of healthcare acquired infections (HAIs) in Australia is high, estimated to be 150,000 per year. These infections cause significant mortality, possibly contributing to as many as 7,000 deaths per year.[5] It is recognised that HAIs compromise patient safety and the quality of care provided, and add a significant resource burden to the health system. A proportion of HAIs involve antimicrobial resistant organisms, and these increase costs even further and often result in increased morbidity and mortality and even higher hospital and post-hospital care costs. HAI surveillance provides important information to hospital staff. It increases awareness of: the organisms present within the hospital and those entering the hospital; the risk of infection associated with a particular procedure; and appropriate and successful interventions. HAI surveillance can highlight deficiencies in infection control or faults in procedures and can assist in changing attitudes. HAI surveillance, including surveillance of antimicrobial resistant infections, better positions hospital staff to implement appropriate population health action to ensure improved patient outcomes.

The Strategy supports the development of a national minimum dataset and consistent case definitions for antimicrobial resistant HAIs, advocates wide participation, and aims to promote timely reporting and communication of successful interventions.

Aims

To measure the prevalence of and trends in antimicrobial resistance in HAIs (both inpatient and outpatient) in Australia.

Primary agency

Communicable Diseases Branch, Australian Government Department of Health and Ageing (with Safety and Quality Council)

Stakeholders

State and territory health departments

Australian Infection Control Association

Australian Group on Antimicrobial Resistance

Health care facilities and laboratories

Goals

1. Identify national and state-based HAI surveillance systems, which collect AMR data as well as laboratory networks, which report AMR data on key pathogens and seek cooperation in the development of a national surveillance system.
2. Evaluate data quality in available data collections and identify deficiencies.
3. Estimate the prevalence of AMR in HAI and emerging issues.
4. Report national data on the prevalence of AMR HAI.

Processes

Table 5. Processes for reaching goals of surveillance of AMR in healthcare acquired infections

Goals	Processes
Identify national and state-based surveillance systems	Establish contacts with existing surveillance systems; CCU to visit and/or attend meetings of surveillance groups as appropriate
Evaluate data quality	In discussion with established surveillance systems, identify gaps and deficiencies and work with stakeholders to improve data quality
Estimate AMR prevalence	In collaboration with existing groups, estimate the prevalence of AMR in HAI in a variety of settings from existing data
Reporting	In collaboration with stakeholders produce reports on prevalence and trends in AMR HAI

Organisms and antimicrobials for surveillance

Table 6. Organisms and antimicrobials for surveillance priority in the healthcare setting

Organism	Passive surveillance	Targeted surveillance (current group)	
Staphylococcus aureus	Yes	Yes –multi-resistant *Staphylococcus aureus* (AGAR)	Benzylpenicillin, methi/oxacillin, erythromycin, tetracycline, gentamicin, cotrimoxazole/trimethoprim, vancomycin, rifampicin, fusidic acid
Enterococcus species	Yes	Yes –vancomycin resistant enterococci (AGAR)	Ampicillin or benzylpenicillin, vancomycin (plus quinupristin-dalfopristin and linezolid if vancomycin resistant)
Escherichia coli	Yes	Yes (AGAR)	Ampicillin, 1st generation cephalosporins, 3rd generation cephalosporins, amoxycillin-clavulanate, gentamicin, fluoroquinolones, cotrimoxazole/trimethoprim
Klebsiella species	Yes	Yes (AGAR)	Ampicillin, 1st generation cephalosporins, 3rd generation cephalosporins, amoxycillin-clavulanate, gentamicin, fluoroquinolones, cotrimoxazole/trimethoprim
Enterobacter species	Yes	Yes (AGAR)	Ampicillin, 1st generation cephalosporins, 3rd generation cephalosporins, amoxycillin-clavulanate, gentamicin, fluoroquinolones, cotrimoxazole/trimethoprim, carbapenems
Acinetobacter species	Yes	Yes –multi-resistant (SA only at present)	Ampicillin, 1st generation cephalosporins, 3rd generation cephalosporins, amoxycillin-clavulanate, gentamicin, fluoroquinolones, cotrimoxazole/trimethoprim, carbapenems (plus amikacin if gentamicin-resistant)
Pseudomonas aeruginosa	No	Yes –multi-resistant (SA only at present)	Ticarcillin/piperacillin, gentamicin/tobramycin, fluoroquinolones, carbapenems
Clostridium difficile	Yes	No	Nil

Outcomes

To obtain an accurate estimate of the problem of antimicrobial resistance in Australian hospitals and healthcare facilities and to establish a network of surveillance systems which can provide on-going nationally representative data to inform those responsible for management of HAI in Australia.

Surveillance of antimicrobial resistance in animals

Background

There is general agreement in the international literature with the JETACAR report finding that there is qualitative evidence that antimicrobials fed to animals leads to resistant bacteria and that these bacteria or their resistance genes can be passed to humans, principally via the food chain. There is little systematic surveillance of antimicrobial resistance in animals in Australia that is relevant and accessible to public health. A national system of surveillance is needed to monitor antimicrobial resistance in animals. Currently, most data are derived from individual veterinary investigations and are not collected in a routine and specified manner nor aggregated and analysed further. Some molecular studies of antimicrobial resistance genes are currently underway in Australia but these are generally conducted as research projects rather than surveillance programs.

Monitoring and surveillance of antimicrobial resistance derived from the veterinary and agricultural use of antimicrobials in Australia will require a new approach. The most relevant guide in the development of a program for Australia is the international standard developed by the world organisation for animal health, the Organization International des Epizooties (OIE).[6] The OIE is the international standards setting organisation recognised by the World Trade Organization.

Aim

To measure the prevalence of and trends in antimicrobial resistance (that is of public health significance) in bacteria from animals.

Primary agency

Product Integrity Animal and Plant Health, Australian Government Department of Agriculture, Fisheries and Forestry.

Stakeholders

Federal, state and territory primary industry departments

Australian Veterinary Association and veterinarians in food animal practice

Livestock industries

Animal Health Laboratories

National Enteric Pathogen Surveillance Scheme

Australian Salmonella Reference Centre

OzFoodNet

Commercial companies and industry

Processes

Table 7. Processes for reaching goals of AMR surveillance in animals

Goals	Processes
Prevalence of AMR in animals – public health focus	Develop and implement an active surveillance program for commensals that has a public health focus. Summarise data on AMR prevalence for commensal bacteria and antimicrobials from the active surveillance program.
Prevalence of AMR in bacteria in animals	Identify sources of passive surveillance data for zoonotic bacteria. Summarise data on AMR prevalence for zoonotic bacteria and antimicrobials from existing data.
Prevalence of AMR in animals – animal health focus	Identify stakeholders. Evaluate data quality from passive surveillance. Identify gaps and deficiencies and work with stakeholders to improve data quality. Summarise AMR prevalence for key diseases and antimicrobials from existing data.
Data analysis	Identify any trends in AMR prevalence. Identify any associations, if any, between AMR and use patterns of antimicrobials. Identify associations, if any, between AMR in animals and AMR in humans.
Reporting	Establish a reporting cycle and format in conjunction with stakeholders. Produce reports in consultation with stakeholders on trends in AMR in commensals, zoonotic agents and animal pathogens.

Goals

1. Determine the prevalence of antimicrobial resistant bacteria in animals and their environment and detect the emergence of new antimicrobial resistance patterns.

2. Investigate any association there might be between emergence of resistance and the pattern of use of antimicrobials in animals.

3. Identify circumstance where antimicrobial resistance in animals is related to resistance patterns and trends in humans.

4. Report national data on the prevalence of antimicrobial resistance in animals.

Organisms and antimicrobials for surveillance

Table 8. Organisms and antimicrobials proposed as national priorities for animal antimicrobial resistance surveillance

Antimicrobial class	Animal pathogens Gram –ve	Animal pathogens Gram +ve	*Salmonella/ E. coli*	*Campylobacter*	*Enterococcus*
Aminoglycosides					
Apramycin	√	√	√		√
Gentamicin	√	√	√		
Neomycin	√		√		
Streptomycin			√		
Amphenicols					
Chloramphenicol*	√	√	√		
Florfenicol			√		
Beta-lactams					
Ampicillin	√	√	√		√
Oxacillin/cloxacillin		√			
Penicillin					
Cephalosporins					
Ceftiofur	√		√		
Glycopeptides					
Vancomycin*		√			√
Lincosamides					
Lincomycin		√			
Macrolides					
Erythromycin	√	√		√	√
Tylosin					√
Quinolones					
Enrofloxacin*	√	√	√	√	√
Ciprofloxacin*			√		
Streptogramins					
Virginiamycin					√
Sulfonamides					
Trimethoprim/ sulphamethoxazole	√	√	√		
Tetracyclines					
Tetracycline	√	√	√	√	√

* Not registered for use in food animals in Australia.

Outcomes

To obtain objective data on the prevalence of AMR in bacteria of animal origin that have the potential to transfer to humans and cause public health concerns. These data can be used:

- in risk analysis to determine the risk to human and animal health;
- to detect the emergence of particular phenotypes of AMR;
- to detect trends in AMR;
- to identify the need for particular interventions and to assess the impact of interventions; and
- to provide a basis for policy recommendations for public and animal health.

Surveillance of antimicrobial resistance in foods

Background

There is sufficient evidence in the literature to suggest a link between the use of antimicrobials in food producing animals and the emergence of antimicrobial resistant organisms and their spread to humans. Therefore surveillance for antimicrobial resistant organisms of public health importance in foods needs to be addressed. In Australia, there is little systematic surveillance of antimicrobial resistance in bacteria contaminating foods, including animal derived foods. Some data are collected by individuals states, however there is no integration of this data to determine trends in AMR at a national level. Data collected from existing food surveillance activities should form part of the overall resistance surveillance system. A possible future initiative includes the collection of resistance data on *Salmonella, E. coli* and *Campylobacter* in foods through OzFoodNet and Australian Quarantine Inspection Service Imported Foods Inspection Scheme (formerly the Imported Foods Program).

Aim

To measure the prevalence of and trends in antimicrobial resistance in bacteria recovered from food.

Primary agency

Food and Environmental Health Branch, Australian Government Department of Health and Ageing, with OzFoodNet

Stakeholders

Federal, state and territory primary industry and health departments

Food Standards Australia New Zealand

Australian Quarantine Inspection Service Imported Food Inspection Scheme

Livestock industries

Veterinarians in food animal practice

Animal Health Laboratories

National Enteric Pathogen Surveillance Scheme

Australian Salmonella Reference Centre

Goals

1. To evaluate the prevalence of antimicrobial resistant bacteria in food at various stages of production and processing.
2. To investigate any association there might be between emergence of resistance and the pattern of antimicrobial use in food production.
3. To identify circumstances where antimicrobial resistance in foods is related to resistance patterns and trends in animals and humans.
4. To report national data on the prevalence of antimicrobial resistance in bacteria recovered from food.

Processes

Table 9. Processes for reaching goals of AMR surveillance in foods

Prevalence of AMR in animal-derived food	Identify stakeholders. Evaluate data quality from passive surveillance, if any. Identify gaps and deficiencies and work with stakeholders to improve data collection and quality. Summarise AMR prevalence for key organisms and antimicrobials from existing data. Develop and implement a program of active surveillance in foods that has a public health focus.
Data analysis	Identify any trends in AMR prevalence. Identify any associations, if any, between AMR and use patterns of antimicrobials. Identify associations, if any, between AMR in foods and AMR in humans.
Reporting	Establish a reporting cycle and format in conjunction with stakeholders. Produce reports in consultation with stakeholders on trends in AMR.

Organisms and antimicrobials for surveillance of AMR in foods

Table 10. Organisms and antimicrobials proposed as priorities for AMR surveillance in food

Organism	Antimicrobials for surveillance.
Salmonella	Multi-resistance, including fluoroquinolones and 3G cephalosporins.
Campylobacter	Ciprofloxacin, erythromycin, tetracycline.
Enterococcus	Ampicillin, vancomycin, erythromycin, ciprofloxacin, tetracycline, streptogramins.
Escherichia coli	Gentamicin, ampicillin, co-trimoxazole.

Outcomes

1. Systematic surveillance system that generates high quality data on the presence of AMR in food.
2. Effective mechanisms for collating, interpreting and reporting data at a national level.
3. Standardised documentation of trends in food AMR.
4. Data to inform risk assessment and that can be used to develop risk management measures for AMR in food.

Monitoring of antimicrobials used in people and animals

Background

Over the past 20 years there has been increasing concern about the overuse or inappropriate use of antimicrobials in human medicine and a general recognition that over use of antimicrobials can lead to rapid establishment of large pools of antimicrobial resistant pathogens. Surveillance of antimicrobials from the time they enter Australia to the time of their consumption in either animals or humans has to become an accepted component of the total surveillance system. Action is being undertaken through CIJIG and EAGAR to improve usage data. National data on antimicrobial use needs to include total antimicrobials imported, how much is used in humans (including community and hospital use) and animals, and what these antimicrobials are used for. The two regulatory bodies that approve antimicrobials for humans and animals, TGA and APVMA respectively, are already working towards enhancing the quality of antimicrobial import and supply data. Antimicrobial import data are currently collected by TGA, and this includes antimicrobials for human, veterinary and stockfeed use.

The use of antimicrobials in humans is monitored through the Pharmaceutical Benefits Scheme (PBS) and pharmacies. The Drug Utilisation Sub-committee collates and reports on usage data collected by these two bodies. Few hospitals have usage surveillance in place, although a statewide antimicrobial use surveillance system across 15 public and private hospitals has recently commenced in South Australia. There is no comparable system for collecting data on antimicrobial use in the agricultural sector.

Aim

To provide reliable data on the volume of antimicrobials consumed by humans and animals in Australia and their patterns of use.

Primary agency

Australian Government Department of Health and Ageing through the Therapeutic Goods Administration and Australian Pesticides and Veterinary Medicines Authority

Stakeholders

Health care facilities

Australian Infection Control Association

Australian Customs Service

Pharmaceutical Benefits Scheme and Pharmacy Guild

Therapeutic Goods Administration

Australian Pesticides and Veterinary Medicines Authority

State and territory health and primary industries departments

Australian Veterinary Association

Drug Utilisation Sub-committee

Goals

1. Reliable and accurate antimicrobial import, supply and end-use data collected and provided nationally, involving the national collation of human antimicrobial use data from community and hospital pharmacies, PBS and veterinary sources.

2. Aggregated state/territory antimicrobial utilisation information provided to the CCU for inclusion in the national annual publication.

3. Human and Animal Health Reference Networks, the Australian Infection Control Association and other relevant groups, to refine the definition for antimicrobial-use in the context of national surveillance.

Processes

Table 11. Processes for reaching goals of monitoring antimicrobial use in humans and animals

Goals	Processes
Collation of data on antimicrobials imported for use in humans, animals and animal-derived food.	Conduct evaluation sessions to determine that importers define who has requested import and that import request tallies with registered product registers. Evaluate existing sources of antimicrobial import data (e.g. TGA import data) and make recommendations for improvement.
Collation of data on regulatory authority supply of antimicrobials for use in humans, animals and animal derived foods.	Evaluate existing sources of regulatory supply data and make recommendations for improvement, including improving the review and follow-up of antimicrobial supply data. Obtain cooperation from the pharmaceutical companies to facilitate collection of regulatory authority supply data.
Collation of end-use data on antimicrobials used in humans, animals, and animal derived foods.	Evaluate existing sources of end use data and make recommendations, including methods for development of better data on antimicrobials consumption in food producing animals. Determine optimal mechanism for obtaining end use data in animal health and human health sectors. Identify financial infrastructure for collection of end-use data.
Data analysis	Reconciliation of import, regulatory authority and end use antimicrobial usage data. Identify trends in antimicrobial usage among humans and animals, for example in humans, by age group, setting, and indication, and in animals, by species and setting.
Reporting	Establish a reporting format in conjunction with stakeholders. Produce a report on findings and trends in antimicrobial usage in humans, animals, and animal derived food.

Antimicrobials for surveillance in humans and animals

Table 12. Antimicrobials proposed as priorities for surveillance of usage

Settings	Antimicrobials to be considered for surveillance.
Community	Third-generation cephalosporins, first-generation cephalosporins, penicillins, tetracycline, fluoroquinolones, rifamycins, spectinomycin, erythromycin, tetracyclines, co-trimoxazole/trimethoprim, gentamicin, vancomycin, fusidic acid, fluoroquinolones, chloramphenicol, isoniazid, spectinomycin, ethambutol, pyrazinamide.
Hospitals	Penicillins, erythromycin, tetracycline, gentamicin, cotrimoxazole/trimethoprim, glycopeptides, First and third generation cephalosporins, rifampicin, fusidic acid, fluoroquinolones, carbapenems, amikacin, macrolides, linezolid, quinupristin-dalfopristin.
Animals	Aminoglycosides, amphenicols, bambermycins, cephalosporins, lincosamides, macrolides, orthosomycins, penicillins, polyethers, polypeptides, quinolones, streptogramins, sulfonamides, tetracycline.

Outcomes

- To improve existing collection of antimicrobial usage data, including the provision of import supply and end-use data for national surveillance reporting. The data collected will:
  - facilitate risk analysis for registration applications, extensions of use application, and Pharmaceutical Benefits listing.
  - be used in formal reviews of antimicrobials by regulatory authorities;
  - enable evaluation of the effectiveness of prudent use efforts and mitigation strategies;
  - enable trends in antimicrobial usage to be studied; and
  - enable international reporting and comparisons.
- To facilitate the development of prescribing and regulatory interventions that would improve the prudent use of antimicrobials.

Monitoring and evaluation of the Strategy

An ongoing evaluation process should monitor progress against the Strategy. The performance of the strategy will be monitored against performance indicators that will be outlined in the detailed action plans for each of the four surveillance areas. Progress against the Strategy will be regularly reported by CIJIG on the Implementing JETACAR website, to the Communicable Diseases Network Australia, the Public Health Laboratory Network and other relevant bodies including the Australian Health Ministers' Conference and the Primary Industries Standing Committee, as required.

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Tuberculosis notifications in Australia, 2002

Gina Samaan,[1,2] Paul Roche,[1] Jenean Spencer[1] and the National Tuberculosis Advisory Committee (Ral Antic – Chair, Ivan Bastian, Amanda Christensen, Mark Hurwitz, Anastasios Konstantinos, Vicki Krause, Avner Misrachi, Graham Tallis, Justin Waring, Moira McKinnon) for the Communicable Diseases Network Australia

Abstract

In 2002, there were 1,028 cases of tuberculosis (TB) reported to the National Notifiable Disease Surveillance System, of which 997 were new cases, 30 were relapses and 1 unknown. The incidence rate of TB in Australia in 2002 was 5.2 cases per 100,000 population. The highest incidence of TB was reported in people born overseas (20.2 cases per 100,000 population), followed by Indigenous Australians (8.5 cases per 100,000 population). By contrast, the incidence rate of TB in the non-Indigenous Australian-born population was 1.1 cases per 100,000 population. This pattern of TB incidence rates amongst the sub-populations of Australia has been observed for over 10 years. The rates were evaluated against the performance indicators set by the National Tuberculosis Advisory Committee to ensure that Australia's record of TB control is maintained and improved. *Commun Dis Intell* 2003;27:449–458.

Keywords: Mycobacterium tuberculosis, *tuberculosis, disease surveillance*

Introduction

Tuberculosis (TB) represents one of the most significant public health threats to the global population. In 2001, 183 countries notified 3.8 million cases of TB to the World Health Organization (WHO) Global Surveillance Programme,[1] of which, 42 per cent were sputum smear-positive. However, the rates are likely to be underestimates of the global TB burden. WHO reports that the global incidence rate of TB is increasing at approximately 0.4 per cent per year, but that the increase is faster in sub-Saharan Africa and in countries of the former Soviet Union. The Western Pacific Region, of which Australia is a member, accounted for 24 per cent of all cases notified to WHO in 2001. Four countries from the Western Pacific Region were among the top 22 countries with a high TB burden. In contrast, Australia has one of the lowest incidence rates for TB in the world. There remain two sub-populations within Australia who have high incidence rates of TB: Indigenous Australians and people born overseas.

The targets for global TB control, set by the WHO, are to successfully treat 85 per cent of detected sputum smear-positive TB cases and to detect 70 per cent of all active TB cases. To meet the treatment target, the WHO has recommended the Directly Observed Treatment – Short-course (DOTS) program. The five major components of the DOTS program are political commitment and resources, the use of microscopy to diagnose TB, standardised observed treatment for all patients with active TB, uninterrupted supplies of anti-TB drugs, and a standardised reporting system for monitoring treatment and progress of TB patients (WHO, 2002). The major principles that underpin the DOTS program guide the treatment of TB patients throughout Australia.

In order to address the burden and human impact of TB on the Australian population, the National TB Advisory Committee implemented a *National Strategic Plan for TB Control in Australia Beyond 2000*, which was endorsed by the Communicable Diseases Network Australia (2002). This is the second annual report to match the national surveillance data to the Performance Indicators set by the strategic plan.

In the past, TB notifications in Australia were reported to the National Mycobacterial Surveillance System. Enhanced surveillance for TB notifications was commenced in 2001 as a part of the National Notifiable Diseases Surveillance System. Information about drug susceptibility is published by the Australian Mycobacterium Laboratory Reference Network in the accompanying report.

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Methods

Data collection

Each jurisdiction in Australia has legislation that requires medical practitioners, public health laboratories and other health professionals to report cases of TB to the state and territory health authority. Notifications of TB for 2002 were collated by jurisdictions and sent electronically to the Commonwealth Department of Health and Ageing. Confidentiality was ensured as all records were forwarded in a de-identified format. Data fields in the enhanced TB dataset that are analysed in this report are listed in Table 1 with a brief description of each variable.

The National Tuberculosis Advisory Committee, as a sub-committee of the Communicable Diseases Australia Network, was responsible for determining the dataset collected in 2002. The dataset collected was the same as in 2001.

Data processing and quality control

Data on all TB notifications reported in 2002 were received by September 2003. Each variable was examined for data completeness. Data were checked for validity, whereby any invalid or missing entries were returned to the jurisdictions for review and correction.

Most cases of TB in Australia are reported to the surveillance system.[2] Reasons for the high level of reporting include the presence of an effective TB screening program, a high standard of health care for all TB patients, and specialised and multi-disciplinary TB services in each jurisdiction. The terms 'notification rate' and 'incidence' are therefore used interchangeably in this report.

Case definition

In 2002, cases were classified as either new or relapsed. A new case required a diagnosis accepted by the Director of TB Control (or equivalent) in the relevant jurisdiction, based on laboratory or clinical evidence. Laboratory evidence includes either the isolation of *Mycobacterium tuberculosis* complex (*M. tuberculosis, M. bovis or M. africanum)* from a clinical specimen by culture; or nucleic acid testing indicating *M. tuberculosis* complex except where it is likely to be due to previously treated or inactive disease.

Clinical evidence is a diagnosis made by a clinician experienced in tuberculosis and includes clinical follow-up assessment.

A relapsed TB case was defined as a case of active tuberculosis diagnosed bacteriologically, radiologically or clinically, having been considered inactive or quiescent following previous treatment

Table 1. Description of some of the data fields in the enhanced tuberculosis dataset of the National Notifiable Disease Surveillance System*

Data field	Description
Country of birth	Country in which patient was born.
Extrapulmonary site	Details of extrapulmonary site involved.
New or relapse case	Whether the case was a new case (without previous treatment), relapse following full treatment in Australia, relapse following partial treatment in Australia, relapse following full treatment overseas or relapse following partial treatment overseas.
TB outcomes	Whether the case was cured (bacteriologically confirmed), completed treatment, interrupted treatment (but still completed), died of TB, died of other cause, defaulter (failed to complete treatment), failure (completed treatment but failed to be cured), transferred out of Australia and still under treatment.
Age	Age of patient at diagnosis
Indigenous status	Whether patient is self-identified Indigenous (Aboriginal and/or Torres Strait Islander) Australian or not.
Selected risk factors	Selected risk factors including close contact with a TB patient, residing in a correctional facility, residing in an aged care facility, employed in an institution, employed in the health industry, HIV status or past residence in a high risk country.
Sex	Male or female.

* Other data collected on each case included diagnosis details, therapy and susceptibility. These were analysed in the accompanying TB lab report.

(as deemed by the State or Territory Director of Tuberculosis). Relapses refer to retreatment cases and some of these may be reinfections rather than a true relapse of prior disease.

Population estimates for 2002

The rates presented in this report were calculated using population data produced by the Australian Bureau of Statistics (ABS). The estimated resident population (ABS, 2002) in each state and territory and in Australia as a whole, as at 30 June 2002, was used as the denominator in crude rate calculations.

Estimates of the Indigenous Australian population were based on projections from the 2001 census estimate of the Indigenous population in Australia (ABS, 2001). The ABS calculated the projections based on assumptions about future births, deaths and migrations in the Indigenous population and a 'low' and 'high' estimate provided. For the purpose of this report, the 'low' estimate has been used, which is consistent with previous annual reports for TB notifications in Australia.

The 2001 census data were used to calculate incidence rates of TB in people born overseas. The estimated resident population of overseas-born people (total and by country of birth) in 2001 was used as the denominator in calculating rates.

To estimate the non-Indigenous Australian-born population, the Indigenous population estimate and the overseas-born population estimate were subtracted from the total Australian population. Since some of the TB notifications in the report may include non-permanent residents of Australia in 2002, the rates may be overestimated.

Results

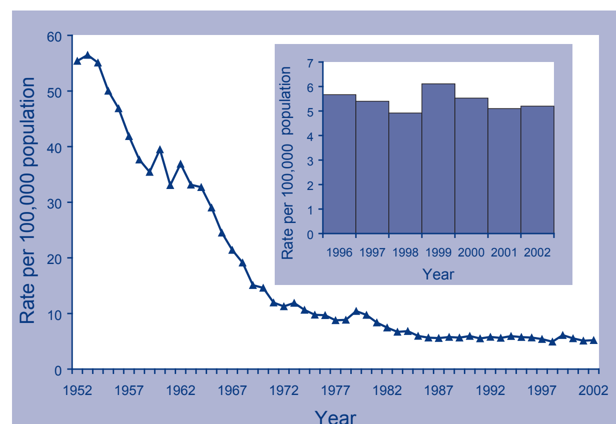
Data quality

Information on age and sex for all notifications were reported. Indigenous status was reported for 198 of the 204 (97.1%) cases born in Australia and country of birth was recorded for 1,023 (99.5%) of the total TB notifications. The site(s) of TB disease were reported for 1,025 cases and whether the case was new or relapse was reported for 1,027 cases. The outcome from treatment was reported for 802 (78%) cases. One data field that was not well reported in 2002 was risk factors for TB (48.7% complete), and it was unclear whether the lack of reporting indicate no underlying risk factors or missing data. Similarly, HIV status was only provided for 27.3 per cent of cases.

Tuberculosis notification rates

The number of cases of TB reported in Australia in 2002 was 1,028 (5.2 cases per 100,000 population). The notification rate of TB in 2002 was similar to that in 2001 (5.1 cases per 100,000 population) (Figure 1). The national notification rate of TB has remained relatively stable since 1985 except for an increase in 1999, due to the number of TB cases amongst the East Timorese refugees evacuated to Australia in that year.

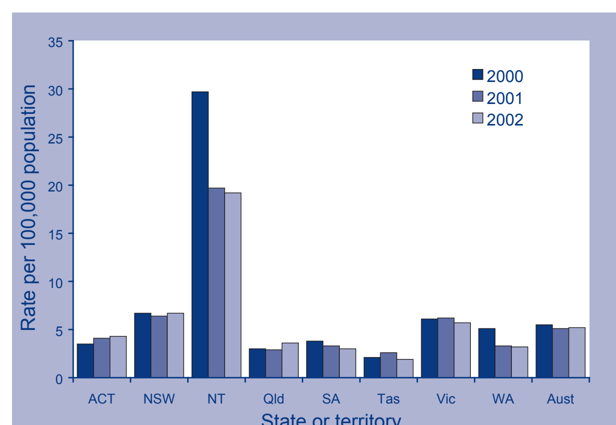
Figure 1. Incidence rates per 100,00 population for tuberculosis notifications, Australia, 1952 to 2002



Tuberculosis notifications by jurisdiction

New South Wales reported the most notifications (447 cases) of TB in 2002, however, the highest rate was recorded in the Northern Territory (19.2 cases per 100,000 population) (Table 2). This rate was lower than the rate for the Northern Territory reported in 2001 (19.7 cases per 100,000 population) and in 2000 (29.7 cases per 100,000 population) (Figure 2). The lowest notification rates in 2002 were reported in Tasmania (1.9 cases per 100,000 population) and South Australia (3 cases per 100,000 population).

Figure 2. Notification rates for tuberculosis, Australia, 2000 to 2002, by state or territory



Of the 1,028 cases reported in 2002, 997 (97%) were new cases of TB, 30 (3%) were relapsed cases and one where relapse status was unknown (Table 2). Of the 30 relapsed cases, 23 relapsed following full treatment in Australia, one relapsed after partial treatment in Australia, four relapsed after full treatment overseas and two relapsed after partial treatment overseas.

Tuberculosis notifications in the Australian-born population

In 2002, 204 (19.8%) cases of TB occurred in the Australian-born population, of whom 167 (82%) were non-Indigenous Australian-born, 37 (18.1%) were Indigenous Australians and 6 (2.9%) were of unknown Indigenous status. For the six cases where Indigenous status was unknown, the numbers were added to the non-Indigenous Australian-born category for the calculations of rates (Table 3).

The incidence of TB in Indigenous Australians for 2002 was 8.5 cases per 100,000 population, which was the second lowest rate reported for this population since 1991. The rate of TB incidence in the non-Indigenous Australian-born population (1.1 cases per 100,000 population) has remained stable over the past 11 years.

The highest notification rate of TB among Australian-born individuals was in the Northern Territory (14.0 cases per 100,000 population) and the lowest was in Western Australia (0.6 cases per 100,000 population) (Table 3). The majority of cases in Indigenous Australians (24/37 cases; 64.9%) were reported in the Northern Territory (41.9 cases per 100,000 population), a jurisdiction where 28 per cent of the population are Indigenous Australians as compared to two per cent nationwide. Queensland reported nine cases of TB in the Indigenous population (7.4 cases per 100,000 population), which was similar to the number of cases reported in the previous year.

Table 2. Notifications of new and relapsed cases of tuberculosis and rates per 100,000 population, Australia, 2002, by state or territory*

State	New cases		Relapsed cases		Total	
	n	%	n	%	n	%
Australian Capital Territory	14	4.3	0	0.0	14	4.3
New South Wales	441	6.6	6	0.1	447	6.7
Northern Territory	37	18.7	1	0.5	38	19.2
Queensland	124	3.3	9	0.2	133	3.6
South Australia	43	2.8	3	0.2	46	3.0
Tasmania	9	1.9	0	0.0	9	1.9
Victoria	272	5.6	7	0.1	279	5.7
Western Australia	57	3.0	4	0.2	61	3.2
Australia	997	5.1	30	0.2	1,027	5.2

* There was one case where relapse status was unknown.

Table 3. Notifications of tuberculosis and incidence rates in Indigenous and non-Indigenous people born in Australia, 2002, by state or territory

State/territory	Indigenous Australian-born		Non-Indigenous Australian-born		Total Australian-born	
	n	%	n	%	n	%
Australian Capital Territory	0	0.0	1	0.4	1	0.4
New South Wales	2	1.6	60	1.2	62	1.2
Northern Territory	24	41.9	2	1.8	26	14.0
Queensland	9	7.4	34	1.1	43	1.4
South Australia	1	4.0	10	0.8	11	0.9
Tasmania	0	0.0	5	1.2	5	1.2
Victoria	1	4.0	47	1.2	48	1.3
Western Australia	0	0.0	8	0.6	8	0.6
Australia	37	8.5	167	1.1	204	1.3

The more populous states of New South Wales, Victoria and Queensland reported 60, 47 and 34 cases of TB, respectively, in the non-Indigenous population, while the Northern Territory only reported two cases but had the highest rate (1.8 cases per 100,000 population).

The rate of notifications of TB in 2002 was highest in overseas-born people (20.2 cases per 100,000 population), which was a slight increase from the previous two years (19.3 cases per 100,000 population in 2001 and 18.0 cases per 100,000 population in 2000). However, the rate for 2002 remains lower than that of 1999 where 21.6 cases were reported per 100,000 population (Figure 3).

Tuberculosis notifications in people born overseas

Of the 1,028 cases of TB reported in 2002, 819 cases (79.7%) were in people born overseas. Table 4 shows the number of TB notifications and incidence rate of TB based on the estimated Australian resident population for each country. Of these cases, 36.6 per cent (300/819 cases) come from three countries: India (106 cases), Vietnam (102) and the Philippines (92).

The incidence of TB amongst people born overseas in the Australian population was highest in people from Somalia (565.6 cases per 100,000 population), India (111 cases per 100,000 population) and Papua New Guinea (101.6 cases per 100,000 population). Some caution is required in interpreting these results, as the rates may include temporary as well as permanent residents.

Table 4. Notifications of tuberculosis and estimated rate per 100,000 population for selected countries of birth, Australia, 2002

Country of birth	New cases	Relapsed cases	Total cases	Estimated Australian resident population by country of birth, 2001	Rate per 100,000 population in Australia by country of birth, 2002*	WHO incidence rate (per 100,000 population for country, 2001†
India	106	0	106	95,455	111.0	43
Vietnam	98	4	102	154,833	65.9	115
Philippines	91	1	92	103,942	88.5	139
China‡	54	1	55	142,778	38.5	38
Indonesia	48	0	48	47,156	101.8	134
Papua New Guinea	21	3	24	23,618	101.6	323
Somalia	22	1	23	3,713	619.4	75
Thailand	22	0	22	23,599	93.2	78
Hong Kong	19	1	20	67,121	29.8	109
Korea§	16	1	17	38,958	43.6	79‖
Malaysia	15	0	15	78,858	19.0	66
United Kingdom	14	0	14	1,083,318	1.3	10
Cambodia	13	0	13	22,979	56.6	143
Sri Lanka	12	0	12	53,460	22.4	39
Others	247	9	259	21,48,140		
Overseas	798	21	819	4,087,928	19.9	
Australia	197	7	204	15,619,272	1.3	
Not stated	2	2	4			
Total	997	30	1,027	19,707,200	5.2	

* Country of birth for denominator is from the 2001 census.

† Rates from the World Health Organization 2003 global tuberculosis report.

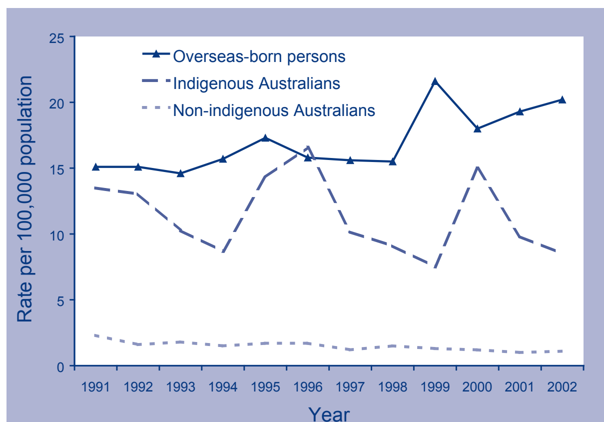
‡ China excludes Hong Kong SAR and Taiwan.

§ The notifications for Korea include both the Republic of Korea and the Democratic Peoples Republic of Korea.

|| The WHO figure quoted is for the Republic of Korea, as virtually all of Korean-born people in Australia are from the Republic of Korea.

There was one case where relapse status was unknown.

Figure 3. Trends of tuberculosis incidence rates, Australia, 1991 to 2002, by Indigenous status and country of birth



Tuberculosis notifications by age and sex

There were a total of 40 cases of TB in children under 15 years of age and the overall notification rate for this age range was one case per 100,000 population. The notification rate was highest in overseas-born children (6.9 cases per 100,000 population, 14 cases), but was only slightly higher than the notification rate in Indigenous Australian-born children (4.3 cases per 100,000 population, 7 cases) (Table 5). The rate for Indigenous Australian-born children increased from the 2001 figures, where there were 2.4 cases per 100,000 population (4 cases) for the same age group. The rate in non-Indigenous Australian-born children remained low at 0.5 cases per 100,000 population (19 cases).

The notification rate in the Indigenous Australians was highest in the 55–64 year age range (43.4 cases per 100,000 population). The rate for Indigenous Australians in the 65+ year age group (26.2 cases per 100,000 population) was lower than that in 2001 (52.2 cases per 100,000 population). Amongst the non-Indigenous Australian-born population, the notification rate was highest in people aged over 65 years (4.1 cases per 100,000 population). For people born overseas, the notification rate was highest in the 25–34 year age range (31.6 cases per 100,000 population).

The age- and sex-stratified incidence rates for TB in overseas-born and Australian-born (Indigenous and non-Indigenous combined) populations are shown in Figure 4. The pattern of distribution of TB cases was different in the overseas-born population and Australian-born population. In the Australian-born population, there was approximately one case per 100,000 population for people up to the 45–54 year age range for both males and females, after which the incidence rate increased gradually for both sexes. The highest rates of TB for the Australian-born population were in the 65+ age group, where the rate for males was 5.6 cases per 100,000 population and 3.3 cases per 100,000 population for females. The overall male:female ratio in Australian-born TB cases was 1.3:1, which was similar to that in 2001.

The highest rate of TB in overseas-born females was in the 25–34 year age range (34.3 cases per 100,000 population). The rate decreased for the 55–64 year age range (9.7 cases per 100,000 population) and then increased again to 15.5 cases

Table 5. Notifications and estimated incidence rate of tuberculosis per 100,000 population, Australia, 2002, by age group, Indigenous status and country of birth

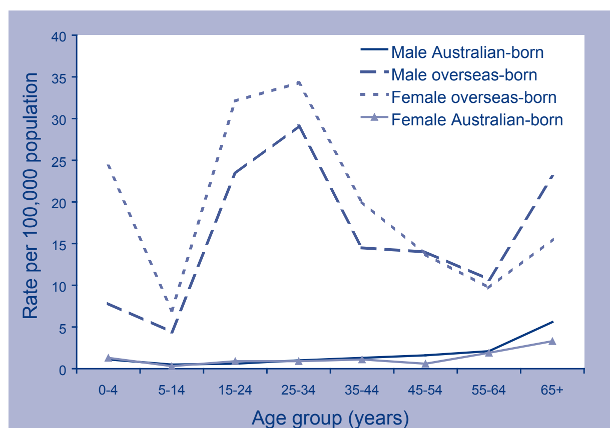
Age group	Indigenous Australian-born		Non-Indigenous Australian-born		Overseas-born	
	n	%	n	%	n	%
0-4	3	5.7	12	1.0	4	15.9
5-14	4	3.7	7	0.3	10	5.6
Sub total for <15 years	7	4.3	19	0.5	14	6.9
15-24	2	2.7	15	0.7	109	27.7
25-34	6	9.4	15	0.7	191	31.6
35-44	9	17.8	17	0.8	148	17.1
45-54	3	9.3	16	0.9	120	13.7
55-64	7	43.4	18	1.5	69	9.9
65+	3	26.2	67	4.1	159	18.8
Australia	37	9.0	167	1.1	810	18.1

The denominator used for total non-Indigenous Australian-born population is from the 2001 census, whilst age group breakdowns use denominators from estimated resident population in 2000 based on the 1996 census results.

There were five cases where country of birth was unknown and nine cases where Indigenous status was unknown.

per 100,000 population for the 65+ year age group (Figure 4). The pattern of TB rates in overseas-born males was similar to that of overseas-born females, but there was a greater increase in incidence of TB for males aged 65+ (23 cases per 100,000 population) than females in the same age group. The overall male:female ratio of TB cases in the overseas-born population was 1.4:1, which was the same as in 2001.

Figure 4. Incidence rate of tuberculosis in Australian-born and overseas-born people, 2002, by age group and sex



There were five cases where country of birth was unknown.

Tuberculosis and selected risk factors

Information on selected risk factors, excluding HIV were reported for 220 of the 1,028 (21.4%) cases. Caution must be taken in interpreting these results as it is unclear whether there were no risk factors identified in the other TB notifications or if the information was not collected. Of the notifications where risk factors were identified, 139 cases were household members or close contacts of TB cases, nine cases either resided or had recently resided in a correctional service and 17 cases either resided or recently resided in an aged care facility. For individuals working in high risk settings, three cases were employed or recently employed in institutions such as correctional facilities or aged care facilities and 10 cases were employed or recently employed in the health industries. There were 140 cases identified as having previously resided for three or more months in high risk countries as defined by the Department of Immigration, Multicultural and Indigenous Affairs.

Tuberculosis and HIV status

Information on HIV status was reported for 281 of the 1,028 (27.3%) TB cases notified in Australia in 2002. Of the TB cases where HIV status was known, there were 11 persons who were HIV positive. Two of these cases were born in Australia and nine were born overseas. The National Strategic Plan recommends that HIV status of all TB cases be collected. Australia is working towards reporting HIV status on all TB patients. In 2002, reporting of HIV status was higher than in 2001 when only 4.2 per cent of cases had HIV status reported.

Anatomical site of disease

In 2002, 602 (58.7%) of the TB cases had pulmonary disease. This was the only identified site of disease for 510 (84.7%) of the pulmonary TB cases (Table 6). Of the TB cases amongst both Indigenous and non-Indigenous Australian-born populations, 76 per cent had pulmonary TB as a site of disease. In contrast, 54.3 per cent of the overseas-born cases had pulmonary as a site of disease. The second most common site of disease in TB cases in 2002 was the lymphatic system (165 cases; 16.1%), followed by pleurae (97 cases; 9.5%).

Table 6. New and relapsed cases in Australia, 2002, by site of disease

Site	New cases	Relapse cases	Total cases	Per cent of cases
Pulmonary	585	17	602	58.7
Lymphatic	160	5	165	16.1
Pleural	93	4	97	9.5
Bone/joint	51	1	52	5.1
Meningeal	21	0	21	2.0
Genitourinary	19	1	20	2.0
Miliary	11	0	11	1.1
Peritoneal	7	0	7	0.7
Other	23	0	23	2.2

The total number of cases does not add up to 1,028 and the per cent of cases is greater than 100 per cent as some cases had multiple sites of infection.

Antimicrobial therapy

The antimicrobial drug regimen given to cases was reported for 930 (90.5%) cases of TB. In 2002, there were 23 cases on a two drug regimen, 101 on a three drug regimen, 804 on a four drug regimen and two cases on a regimen of five or more antimicrobial TB drugs. Of the cases provided with a four drug regimen, 801 (99.6%) were prescribed the four drug regimen of isoniazid, rifampicin, pyrazinamide and ethambutol, which is commonly used as the standard short course treatment for active TB in those aged eight years and above. Ethambutol is not recommended for use in young children where visual testing cannot be assured and of the 17 cases under eight years of age for which antimicrobial therapy was reported, 13 had the three drug regimen of isoniazid, rifampicin and pyrazinamide.

Treatment outcomes

Treatment outcomes were reported for 802 (78%) cases in September 2003. The remaining 226 (22%) cases were either still under treatment or their treatment status was unknown. Satisfactory outcomes were reported for 652 (80%) cases, comprising 74 patients with bacteriologically confirmed cure and 578 who had completed treatment (Table 7). Of the rest, 3 (0.4%) cases had interrupted treatment for TB and 6 (0.7%) cases were reported as defaulters. One case was reported to have failed treatment (0.1%).

The proportion of cases who completed treatment by September 2003 was highest amongst Indigenous Australians (84.8%). The proportion of cases who completed treatment was 76.6 per cent for the non-Indigenous Australian-born population and 82.7 per cent for the overseas-born population. There were 18 deaths reported in 2002 due to TB and the case fatality rate was 2.2 per cent.

National Performance Indicators

The performance criteria for the National Performance Indicators were set by the National Tuberculosis Advisory Committee in 2002 and reviewed in 2003. Some of the indicators remain under review.

As in last year's TB annual report, the performance criteria for people born overseas applies to people who have been living in Australia for more than five years. Based on this, of the 819 cases born overseas, 471 had been living in Australia for more than five years. The incidence rate for people born overseas who have been living in Australia for more than five years was 11.5 cases per 100,000 population.

The incidence of TB in children less than 15 years of age in the Indigenous population increased from the previous year (2.4 cases per 100,000 population in 2001 and 4.3 cases per 100,000 population in 2002).

Table 7. Outcome of treatment for tuberculosis amongst cases evaluated, Australia, 2002, by Australian-born (Indigenous and non-Indigenous) and overseas-born individuals

Treatment outcomes	Indigenous Australian-born	Non-Indigenous Australian-born	Overseas-born	Unknown	Total	Per cent of cases
Cured (bacteriologically confirmed)	19	11	43	1	74	9.2
Completed treatment	9	87	482	0	578	72.1
Interrupted treatment	0	1	2	0	3	0.4
Died of TB	2	5	10	1	18	2.2
Died of other cause	2	20	40	2	64	8.0
Defaulted*	0	2	4	0	6	0.7
Failed†	0	0	1	0	1	0.1
Transferred out of Australia	1	2	53	2	58	7.2
Total	33	128	635	6	802	

* Defaulted means failed to complete treatment.

† Failed means treatment completed but failed to be cured.

Table 8. National tuberculosis performance indicators, performance criteria and the current status of tuberculosis in Australia, 2002

National TB performance indicator	Performance criteria	Annual incidence of TB (per 100,000 population)	
		2001 %	2002 %
Crude incidence			
Indigenous Australians	<1	9.8	8.5
Non-Indigenous Australian-born	<1	1.0	1.1
Overseas-born persons*	†	10.2	11.5
Relapse cases initially treated in Australia	<2% of total treated cases	NA	2.3
Incidence in children <15 years, by risk group			
Indigenous Australian children	<0.1	2.4	4.3
Non-Indigenous Australian-born children	<0.1	0.5	0.5
Overseas-born children*	†	1.0	0.1
Collection of HIV status in TB cases (% of cases with data collected)	100% over next 3 years	4.2	27.3
Treatment outcome measures (%)			
Cases evaluated for outcomes‡	100	76.9	78.0§
Cases that have treatment completed and are cured	>90	83.6	80.0
Cases recorded as treatment failures‡	<2	0.9	0.1

* The performance criteria for overseas born are applied to people who have been living in Australia for more than 5 years. The denominator for this rate is the total overseas born population living in Australia in 2002.

† Performance criteria currently under review.

‡ The denominator used for both 2001 and 2002 was the number of cases evaluated for treatment outcome.

§ Evaluation was at September 2003.

NA Not available (data incomplete).

Data for 2001 have been recalculated using the denominators specified above.

Discussion

Australia has one of the lowest incidence rates of TB in the Western Pacific Region of the World Health Organization and in the world. In 2002, the incidence rate was 5.2 cases per 100,000 population, which is the third lowest rate ever recorded in Australia. The incidence of TB in Australia has remained between five and six cases per 100,000 population since the mid-1980s.

There was an overall decrease in the incidence rates of TB in Indigenous Australians, where the rate dropped from 9.8 cases per 100,000 population in 2001 to 8.5 cases per 100,000 population in 2002. However, TB incidence was still eight times higher in the Indigenous Australian population than in the non-Indigenous Australian-born population. Two-thirds of TB cases in Indigenous Australians occurred in the Northern Territory, where the Indigenous population comprises 28 per cent of the overall population compared to two per cent nationwide. The highest rates of TB in Indigenous Australians were in people over the age of 55, which was similar to the non-Indigenous Australian- born population. The treatment outcome data indicate that among Indigenous Australians, the proportion of people who complete treatment and are cured was higher than that of non-Indigenous Australian-born people.

The incidence of TB in Indigenous Australians for 2002 was the second lowest rate reported for this group since 1991. Yet, despite the overall decrease in the incidence of TB in Indigenous Australians, there was an increase in the incidence rate of TB for Indigenous Australians under 15 years of age. These rates should be interpreted with caution however, as the identification of cases may vary depending on screening campaigns and any small changes in the numerator when dealing with such a small population, can affect rates considerably. For the seven cases of TB in Indigenous Australians under 15 years of age, six of the seven were identified in the Northern Territory, where five were identified as a result of contact tracing and extended community screening.[3] All these cases were diagnosed early in their disease and had good treatment outcomes. Early detection and management of cases will help Australia achieve the goal set by the National

Strategic Plan to bring the incidence of TB among Indigenous Australians down to that in non-Indigenous Australian-born people.

The other sub-population in Australia in which the TB burden is high is for those born overseas. In the 2002 TB notifications, the highest rate of TB in the overseas-born population occurred in persons aged 15–34 years, which is characteristic of areas where TB is endemic. Nevertheless, the proportion of cases that completed treatment and were cured was similar to that of the Australian-born population.

In 2002, 34.5 per cent of all TB cases notified in Australia were in people born in India, Vietnam, the Philippines and China, all of which have been identified by WHO as high TB incidence countries.[1] Recent research on the association between TB rates among migrants and the estimated incidence of TB in the country of birth has highlighted that for people born overseas, the prevalence of TB in the country of birth is likely to be the most important risk factor upon coming to Australia.[4] Furthermore, for people from high-prevalence countries, research has indicated that the prevalence of resistance to current treatment drugs is high and that this should guide treatment protocols for this group.[5]

The TB notifications for 2002 were the second set of surveillance data matched to the National TB Performance Indicators set by the National Strategic Plan. The report highlighted that Australia is close to meeting the annual incidence of TB performance criteria for the non-Indigenous Australian-born population but not for other groups. In particular, the gap between TB rates for Indigenous Australian-born and non-Indigenous Australian-born is not narrowing.

The reporting of HIV status for TB cases has increased from 4.2 per cent in 2001 to 27.3 per cent in 2002, which is slowly addressing the performance criteria set by the plan. Knowledge about HIV status for people infected with TB is critical in the public health management of cases as it has been estimated that an individual who is infected with both HIV and TB has a 7–10 per cent risk per year of developing active TB, as opposed to the 10 per cent lifetime chance of someone who is infected with TB alone.[6] An evaluation of TB cases in Queensland between 1989 and 1998 also highlighted that HIV infection in TB patients increased the risk of dying but was nevertheless, uncommon.[7]

The performance of treatment outcome measures against the performance criteria need to be interpreted with caution as at the time of data collection, not all cases had completed treatment. In 2002, Australia did not achieve the 90 per cent treatment completion and cure targets set by the Plan but achieved the target for the number of cases recorded as treatment failures (less than 2% of cases). This pattern was similar to that in 2001.

In October 2002, the Australian public health system demonstrated its capacity to respond quickly to the exposure of approximately 1,500 people to an untreated active case of TB in a worker in a Queensland resort.[8] Screening was offered to people both in Australia and overseas, and Queensland Health was tasked with the collation of the national information. To date, there has been no evidence of local transmission of TB from the case to people exposed at the resort. Through the National Strategic Plan, the surveillance data and the global trends, public health practitioners can best tailor campaigns for the screening, case finding and management of TB cases. Such action will help ensure that Australia can progress towards the goals set in the National Strategic Plan in the future.

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Tuberculosis in Australia: bacteriologically confirmed cases and drug resistance, 2002

A report of the Australian Mycobacterium Reference Laboratory Network

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Abstract

The Australian Mycobacterium Reference Laboratory Network collected and analysed laboratory data on new cases of disease caused by *Mycobacterium tuberculosis* complex in the year 2002. A total of 712 cases were identified by bacteriology, representing an annual reporting rate of 3.6 cases of laboratory-confirmed tuberculosis per 100,000 population. The most commonly encountered culture-positive specimens were sputum (n=325), lymph node (n=142) and bronchoscopy (n=100). Smears containing acid fast bacilli were present in sputum (53.2%), bronchoscopy (37.9%) and lymph node (21.2%). Eight children (male n=3, female n=5) under 10 years of age had bacteriologically-confirmed tuberculosis. A total of 55 isolates (7.7%) of *M. tuberculosis* were resistant to at least one of the standard anti-tuberculosis agents. Resistance to at least isoniazid and/or rifampicin was noted for 53 isolates (7.4%), with multidrug-resistance (MDRTB) observed in 12 (1.9%) isolates. Of the 12 MDRTB isolates, eight were from the respiratory tract and five were from smear positive specimens. Of the patients with drug resistant *M. tuberculosis* isolates, 51/55 (92.7%) were classified as having initial resistance, none had acquired resistance during treatment in Australia. The country of birth was known for 54 of 55 such patients; four were Australian-born, and 50 (90.9%) had migrated from a total of 17 countries. Nucleic acid amplification testing (NAAT) was performed on 139 (19.5%) of the 712 culture-positive specimens. Of smear positive respiratory specimens, 74/80 (92.5%) were NAAT positive. For smear negative respiratory specimens, 12/17 (70.6%) reported a NAAT positive result. Importantly, false-negative NAAT results were obtained from 1/16 and 5/64 of smear positive bronchoscopy and sputum specimens respectively. *Commun Dis Intell* 2003;27:459–465.

Keywords: Mycobacterium tuberculosis, laboratory diagnosis, tuberculosis, drug resistance, nucleic acid amplification test

Introduction

Australia continues to record one of the lowest notification rates (5–6 cases per 100,000 population) of tuberculosis (TB) in the world.[1] As part of the World Health Organization (WHO) Western Pacific Region, Australia's near neighbors have some of the highest burdens of TB in the region. These countries include China, Philippines, Papua New Guinea, Cambodia and Vietnam.[2] Australia also shares a close geographic relationship with the WHO South East Asia Region, in particular, Indonesia which has the third highest burden of TB in the world.[1] These countries also have to deal with drug resistance and co-infection with HIV in a setting of chronically under-funded and under-resourced national TB programs.

There are two sources of TB-related data for Australia. Since 1991, the National Notifiable Diseases Surveillance System (NNDSS) has provided statistics on cases of tuberculosis reported to public health authorities in Australia's states and territories.[3] The second source, the Australian Tuberculosis Reporting Scheme has been conducted by the Australian Mycobacterium Reference Laboratory Network (AMRLN) since 1986.[4] Statistics compiled by the AMRLN relate to cases of bacteriologically-confirmed tuberculosis whereas NNDSS data will have a proportion of cases that are identified on the basis of clinical and epidemiological information, or on non-bacteriological laboratory investigations. This report describes the bacteriologically-confirmed TB diagnoses for the year 2002.

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Methods

The data are based on clinical specimens that were culture-positive for *Mycobacterium tuberculosis* complex (MTBC). Although the bacille Calmette-Guérin strain of *M. bovis* is a member of the MTBC, no information on this organism is included in the present report. Almost all isolates of MTBC were referred to one of the five laboratories comprising the AMRLN, for specific identification and drug susceptibility testing. Comparable methodologies are used in the reference laboratories. Relapse cases, as defined by the *National Strategic Plan for TB Control in Australia Beyond 2000*, prepared by the National TB Advisory Committee, were included in the laboratory data as laboratories are generally unable to differentiate relapse cases from new cases.[5] Temporary visitors to Australia were included as were illegal immigrants within correctional services facilities and asylum seekers located in detention centres or on temporary visas within Australia.

For each new bacteriologically-confirmed case, the following information was collected (where available):

- demography: patient identifier, age, sex, HIV status and state of residence;
- specimen: type, site of collection, date of collection and microscopy result;
- isolate: species of mycobacterium and results of drug susceptibility testing;
- nucleic acid amplification testing: results of testing; and
- if the isolate was drug resistant: patient country of origin, and history of previous TB treatment to determine whether resistance was initial or acquired.

Data from contributing laboratories were submitted in standard format to the scheme coordinator for collation and analysis. Duplicate entries (indicated by identical patient identifier and date of birth) were deleted prior to analysis. Rates were calculated using mid-year estimates of the population for the year 2002, supplied by the Australian Bureau of Statistics.[6]

For each case, the nature of the first clinical specimen that yielded an isolate of MTBC was used to record the nominal site of disease. Culture-positive specimens collected at bronchoscopy or by gastric lavage were considered to indicate pulmonary disease. Cases with multi-site isolations, provided a sputum or bronchoscopy specimen was culture-positive, were listed as having pulmonary disease, the most important category for public health purposes. Cases for which there were multiple-site isolations were not categorised as having miliary or disseminated disease as differentiation is based on clinical findings that are generally not available to the reporting laboratories. Initial drug resistance was defined as the presence of drug resistant strains of *M. tuberculosis* in cases of tuberculosis in which there was no known history of anti-tuberculosis treatment. Patients who had begun anti-TB treatment and had developed resistance to one or more of the drugs used during treatment were recorded as having acquired drug resistance.[7]

Results

There were 712 bacteriologically confirmed cases of tuberculosis in 2002 (Figure 1), representing an annual rate of 3.6 cases per 100,000 population. State-specific reporting rates varied from 1.7 (South Australia and Tasmania) to 13.0 cases per 100,000 population (Northern Territory) (Table 1). There were 10 patients from Papua New Guinea who were diagnosed in Australia (included in the Queensland data), and most jurisdictions had at least one person visiting from overseas being diagnosed with TB.

Figure 1. Comparison between tuberculosis notifications and laboratory data, Australia, 1990 to 2002

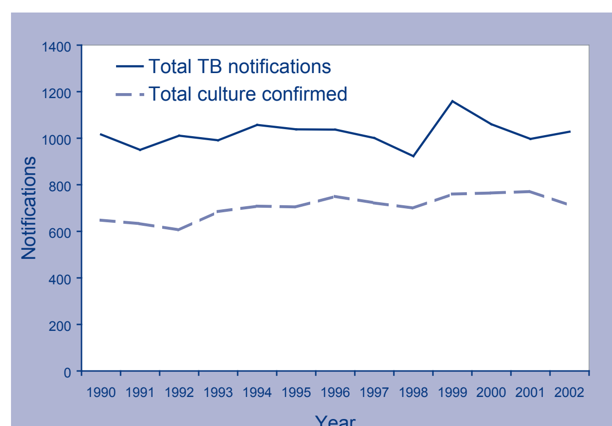


Table 1. Bacteriologically confirmed cases of tuberculosis in Australia, 1992 and 1998 to 2002, cases and rate per 100,000 population, by state or territory*

State or territory	2002		2001[8]		2000[9]		1999[10]		1992[11]	
	n	%	n	%	n	%	n	%	n	%
New South Wales†	301	4.3	327	4.8	307	4.5	291	4.3	252	4.0
Victoria	208	4.3	222	4.6	231	4.8	261	5.5	164	3.7
Queensland	97	2.6	81	2.2	76	2.1	75	2.1	90	3.0
Western Australia	46	2.4	68	3.6	63	3.3	64	3.4	31	1.9
South Australia	26	1.7	38	2.5	41	2.7	46	3.1	41	2.8
Tasmania	8	1.7	12	2.8	2	0.4	2	0.4	7	1.5
Northern Territory	26	13.0	23	11.6	45	23.0	21	10.9	21	12.4
Total	712	3.6	771	4.0	765	4.0	760	4.0	606	3.5

* Data from previous reports from the Australian Mycobacterium Reference Laboratory Network.

† Data from the Australian Capital Territory are included with those from New South Wales.

Causative organism

Almost all isolates were identified as *M. tuberculosis* (710), the remaining two isolates being *M. africanum*. For 2002, there were no bacteriologically confirmed cases of TB caused by *M. bovis*.

Distribution by gender, age and site of disease

Complete information for gender and age were submitted for 697 (97.9%) of the 712 cases. Eight children (male n=3 female n=5) under 10 years of age had bacteriologically confirmed tuberculosis (sputum n=4, gastric aspirate n=2, lymph node n=1, tissue n=1). The relationship of tuberculosis to age and gender is shown in (Figure 2). The overall male: female ratio was 1.05:1. The age and gender rates varied depending upon the site of infection. The predominant culture-positive specimen type was sputum (n=325, 45.6%); a further 100 (14.0%) were bronchoscopy, 15 were biopsy/tissue, and two were aspirate specimens. Thirty-four pleural specimens (31 fluid, 3 biopsy/tissue) accounted for only 4.8 per cent of all culture-positive specimens.

The most commonly encountered extrapulmonary culture-positive specimen was lymph tissue (n=142, 19.9%) followed by those from the genitourinary tract (n=32, 4.5%) and bone/joint (n=23, 3.2%). The female:male ratio of 1.95:1 demonstrated the skewed isolation of extrapulmonary MTBC from females, particularly in the 15–49 year age group (Figure 3). There were 10 isolates from other sites including tissue (spleen, liver, caecum, pericardium, breast, tongue; n=1 for each), pericardial fluid (n=1), and abscess (n=3).

Figure 2. Laboratory diagnosis of *Mycobacterium tuberculosis* complex disease, Australia 2002, by age and sex

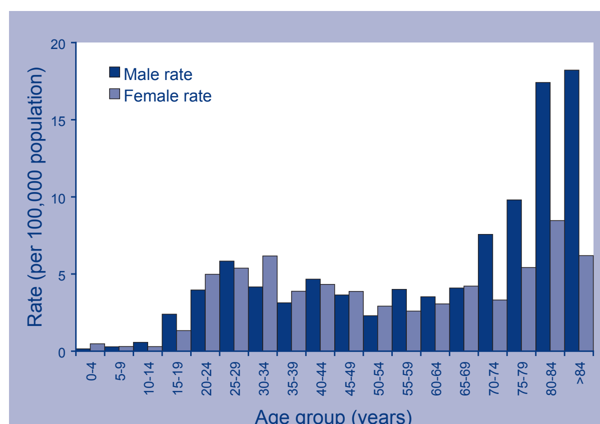
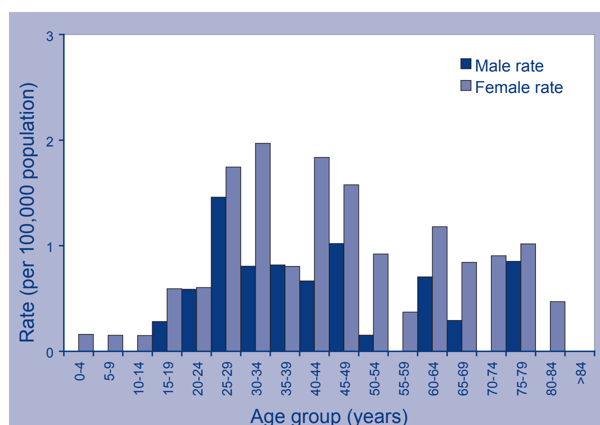


Figure 3. Isolation of MTBC from lymph node, Australia, 2002, by age and sex



Association with HIV

The AMRLN database recorded the HIV status for only 51 (7.2%) patients. A single patient was identified as HIV seropositive; and had a multidrug-resistant strain of *M. tuberculosis* isolated from smear negative sputum.

Microscopy

Results of microscopy were available for 669/712 (94.0%) of specimens; microscopy was not performed on eight specimens and results for a further 35 were unknown. For specimens reporting a microscopy result, smears were positive for 165/310 (53.2%) of sputum and 36/97 (37.9%) of bronchoscopy specimens respectively (Table 2). A total of 34 pleural specimens were culture positive for *M. tuberculosis* with only 3 (9.1%) smear-positive for acid fast bacilli (AFB). Of the 142 specimens of lymph node, microscopy results were available for 133; and 28 (21.2%) were smear-positive for AFB.

Drug susceptibility testing

Results of *in vitro* drug susceptibility testing were available for all 712 isolates for isoniazid (H), rifampicin (R), ethambutol (E), and pyrazinamide (Z). A total of 55 isolates (7.7%) of *M. tuberculosis* were resistant to at least one of the above anti-tuberculosis agents. Results of testing for streptomycin (S) were available for 194/712 (27.2%) of isolates with four demonstrating mono-resistance, and a further five isolates resistant to both S+H. Resistance to at least H and/or R was noted for 53 isolates (7.4%), with resistance to both H and R (i.e. defined as multidrug-resistance) observed in 12 (1.9%) isolates. All of the MDR isolates were *M. tuberculosis* (Table 3). Of the 12 MDRTB isolates, eight were from the respiratory tract (sputum n=7, lung tissue n=1); the remaining four isolates were from lymph node. Five of the MDRTB-positive sputum specimens were smear positive as was the lung tissue. All four lymph tissues were smear negative.

Table 2. Site of specimens smear- and culture-positive for *Mycobacterium tuberculosis* complex disease, Australia, 2002

	Number*	Smear positive (%)†
Sputum	325	53.2
Bronchoscopy	100	37.9
Lymph node	142	21.2
Pleural	34	9.1
Genito-urinary	32	30.8
Bone/joint	23	17.4
Peritoneal	3	ND
Skin	3	ND
CSF	3	ND

* Specimens not tabulated: 15 pulmonary tissue samples, 2 aspirates from upper respiratory tract, from 15 specimens from miscellaneous sites, and 15 of unknown site.

† Based on specimens that reported a microscopy result and excludes (i) microscopy not performed or (ii) result unknown.

ND Percentage of specimens smear positive not calculated due to small numbers.

There was no mono-resistance to either rifampicin or ethambutol, but pyrazinamide mono-resistance was demonstrated in two strains. There were 53 strains that demonstrated resistance to H at a concentration of 0.1 mg/L in the radiometric BACTEC system. Of these, 43 (81.1%) demonstrated resistance at the higher level of 0.4 mg/L. Eighteen of 55 (32.7%) specimens culture-positive for drug resistant *M. tuberculosis* were also smear-positive for AFB.

Table 3. Drug resistance patterns in multidrug-resistant strains, Australia, 1996 to 2002

Resistance pattern (standard drugs)*	2002	2001[8]	2000[9]	1999[10]	1998[10]	1997[12]	1996[13]
H+R only	8	8	3	2	2	6	10
H+R+E	1	1	1	1	1	1	1
H+R+Z	1	3	3	1	2	5	4
H+R+E+Z	2	0	1	0	1	0	0
Total (%)	12 (1.7)	12 (1.6)	8 (1.0)	4 (0.5)	6 (0.9)	14 (1.9)	15 (2.0)

* The streptomycin result was not considered for this table.

H = Isoniazid, R = rifampicin, E = ethambutol, Z = pyrazinamide

Initial or acquired resistance and country of origin

There were 55 *M. tuberculosis* isolates resistant to at least one of the standard drubs (H, R, E or Z). Of these, 51/55 (92.7%) were classified as having initial resistance, none had acquired resistance during treatment in Australia, and no data were available on the presence or absence of previous treatment for four patients. The country of birth was known for 54/55 patients; four were Australian-born, and 50 (90.9%) had migrated from a total of 17 countries. Of the 50 migrants with drug resistant disease, 31 (62.0%) had migrated from one of four countries: Vietnam (n=9), Philippines (n=9), India (n=9), and China (n=4). The four patients with mono-resistance to streptomycin all migrated from Vietnam.

Use of nucleic acid amplification tests

Nucleic acid amplification testing (NAAT) was performed on 139/712 (19.5%) specimens, all of which subsequently grew *M. tuberculosis* on culture. Sputum (n=76), bronchoscopy (n=24), lymph node (n=16) and tissue samples (n=12) were the most frequently tested. Of the 139 specimens, 118 were NAAT positive and 21 were negative. For smear positive respiratory specimens, 74/80 (92.5%) were NAAT positive. For smear negative respiratory specimens, 12/17 (70.6%) reported a NAAT positive result (Table 4). Importantly, 1/16 and 5/64 of smear positive bronchoscopy and sputum specimens respectively that were culture positive for *M. tuberculosis* were NAAT negative. For extrapulmonary specimens such as lymph node, other tissues and urine, smear positives were more likely than smear negatives to yield a positive NAAT, although a small proportion of smear and culture positive specimens produced a negative NAAT result.

Table 4. Results for nucleic acid amplification tests performed on respiratory specimens, Australia, 2002

NAAT result*	Culture positive respiratory specimens	
	Smear positive	Smear negative
Positive	74	5
Negative	6	12
Total (97)†	80	17

* Various NAAT methods were used, depending upon laboratory.

† Three respiratory samples did not record a smear result.

A further five specimens were NAAT positive but culture negative for MTBC. These specimens were not included in the 2002 laboratory data. Specimen types included lymph node (n=2), and one each of sputum, pleural fluid and a bone biopsy. One non-viable culture received into an AMRLN laboratory was NAAT positive.

Discussion

The isolation of 710 *M. tuberculosis* and two *M. africanum* from clinical specimens for 2002 yielded a rate of 3.6 cases per 100,000 population, an outcome consistent with the laboratory data reported for the previous 17 years.[4,8–13] The NNDSS reported 1,028 tuberculosis notifications in 2002,[14] marginally up on the 997 cases reported in 2001.[15] The NNDSS has consistently reported higher notifications than the AMRLN laboratory data (range 24–40%) and for 2002, there was a 44 per cent difference between the two datasets. In 2002, the NNDSS dataset recorded 602 cases from the respiratory tract, 97 pleural, 165 lymphatic, and 52 bone/joint.[14] If the two databases are compared, 70.5 per cent, 35 per cent, 86 per cent and 44 per cent of respiratory, pleural, lymphatic and bone/joint cases respectively were bacteriologically confirmed. In contrast to the 2001 laboratory report where there was almost 90 per cent bacteriological confirmation of respiratory tuberculosis, only 70.5 per cent of respiratory tuberculosis was bacteriologically confirmed in 2002. The consistent finding of a much lower proportion of extrapulmonary disease being confirmed by culture suggests an ongoing reliance being placed upon clinical, histological or radiological diagnoses of these forms of tuberculosis.

In Australia, lymph node tuberculosis is the most common extrapulmonary site and accounts for around 20 per cent of all bacteriologically confirmed disease. The majority of cases occur in overseas-born women in the 15–49 year age range; and these findings have been noted elsewhere.[16,17,18] Specimens submitted for culture included pus from draining sinuses, fine needle aspirates, or lymph tissue. In 2000, 2001 and 2002, smears from lymphatic tissue were positive for AFB on 28.7 per cent, 19.2 per cent and 21.2 per cent of cases respectively.[8,9] Other studies have demonstrated AFB in 25–50 per cent of lymph node biopsy smears.[19] Cultures are positive for MTBC on only 60–70 per cent, in part due to the small population of AFB within the lymph tissue. Interestingly, affected lymph nodes may increase in size, or new nodes may appear whilst on appropriate anti-TB treatment.

Such a paradoxical response is not indicative of inadequate treatment or relapse, and the nodes are sterile on culture.[16] The AMRLN laboratories continue to receive requests for molecular evaluation of formalin-fixed material, notably lymph tissue. These techniques are demanding, time consuming, expensive, and frequently yield a negative result due to the presence of inhibitors within the sample.

A total of 55 isolates (7.7%) of *M. tuberculosis* were resistant to at least one of the standard anti-tuberculosis agents. For 2002, there were 12 (1.9%) isolates of MDRTB. There was no mono-resistance to either rifampicin or ethambutol, but pyrazinamide mono-resistance was demonstrated in two strains. In Australia, the level of drug resistance and MDRTB continues to remain stable. The level of acquired drug resistance in Australia remains low, most drug resistance occurred in patients born overseas and reflects the performance of the TB program of their country of origin.

Data were collected on results of nucleic acid amplification tests. As expected, there was a high level of agreement between NAAT and culture results but a lower level of agreement with smear negative specimens. The level of NAAT lies somewhere between that for culture and smear microscopy. In studies where quantitative culture was performed, the majority of false-negative NAA test results were due to low concentrations of MTBC.[20,21,22] The most important consideration is that culture remains the 'gold standard' for laboratory investigation of mycobacterial disease and a sufficient amount of an appropriate specimen must be used for culture.[23] NAAT should be used to complement 'traditional' laboratory investigations and be limited to situations where the result is likely to influence clinical and/or public health decisions.[24] In 1996, a workshop among clinical, laboratory and public health practitioners considered the clinical suspicion of TB and sputum microscopy in conjunction with the outcomes of NAAT. NAAT was felt to be most useful in situations where there was a smear-negative patient at high risk of TB or a smear-positive patient considered a low risk patient. NAAT results can influence decisions on whether to begin anti-TB treatment, to consider further diagnostic investigations, or to institute public health actions.[25]

Bacteriological confirmation of tuberculosis is important as an isolate is required for identification to species level, drug susceptibility testing and genotyping. The recent agreement among the AMRLN laboratories to a common approach to genotyping based on mycobacterial interspersed repeat units means that the potential to compare all *M. tuberculosis* isolates at a national level is now available.[26] It is proposed to include genotyping data in future AMRLN reports.

Acknowledgements

The Australian Mycobacterium Reference Laboratory Network comprises the Mycobacterium Reference Laboratories at the following facilities:

- Institute of Medical and Veterinary Science, Adelaide, South Australia
- Queensland Health Pathology Services, Prince Charles Hospital, Chermside, Queensland
- Victorian Infectious Diseases Reference Laboratory, North Melbourne, Victoria
- Western Australian Centre for Pathology and Medical Research, The Queen Elizabeth II Medical Centre, Nedlands, Western Australia
- Institute of Clinical Pathology and Medical Research, Westmead Hospital, Westmead, New South Wales

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Invasive pneumococcal disease in Australia, 2002

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Abstract

There were 2,271 cases of invasive pneumococcal disease (IPD) notified to the National Notifiable Diseases Surveillance System in Australia in 2002; a rate of 11.5 cases per 100,000 population. The notification rate varied between states and territories and by geographical region with the highest rates in the north of the country. Invasive pneumococcal disease was reported most frequently in children aged less than five years (57.3 per 100,000 population). Enhanced surveillance for IPD in 2002 was carried out in all states and territories, providing additional data on 1,929 (85%) of all notified cases. Rates of IPD in Indigenous Australians were 2.7 times the rate in non-Indigenous Australians. The clinical presentation of IPD was most commonly pneumonia (44%) and bacteraemia (35%). There were 175 deaths attributed to IPD resulting in an overall case fatality rate of 9.2 per cent. Forty-two per cent of all cases had a recognised risk factor for IPD. Seventy-five per cent of all pneumococcal isolates serotyped were serotypes in the seven-valent conjugate vaccine and 93 per cent were serotypes in the 23-valent polysaccharide pneumococcal vaccine. The clinical presentation and rates of risk factors varied between Indigenous and non-Indigenous cases and non-vaccine serotypes occurred more frequently among Indigenous children and adults. *Commun Dis Intell* 2003;27:466–477.

Keywords: disease surveillance, pneumococcal disease, polysaccharide pneumococcal vaccine, Streptococcus pneumoniae

Introduction

Infection with *Streptococcus pneumoniae* is responsible for significant morbidity and mortality worldwide, especially in the very young, the elderly and those with predisposing risk factors. It is a leading cause of otitis media, pneumonia, bacteraemia, meningitis and a less frequent cause of other conditions including septic arthritis and mastoiditis. Invasive pneumococcal disease (IPD) is defined as a clinical condition in which *S. pneumoniae* infects a normally sterile site such as blood, cerebrospinal fluid (CSF) or pleural fluid. IPD presents most commonly as pneumonia in adults and bacteraemia in children. The risk of disease is highest among people who are immunocompromised or have a chronic illness.

In developed countries, the incidence rate of IPD is bimodal, with a peak in children under two years and another peak in adults over 65 years. The incidence rates can be many times higher in developing countries and in some populations of developed countries, including Australian and American Indigenous people. Rates in Indigenous children from Central Australia between 1994 and 1998 were 1,534 cases per 100,000 population[1] while between 1983 and 1990, rates of IPD among White Mountain Apache children were as high as 1,820 cases per 100,000 population.[2] Case fatality rates for IPD vary depending on the age and the focus of the disease.[3]

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Since the 1970s, large outbreaks of severe pneumococcal disease caused by penicillin resistant organisms occurred in South Africa and Papua New Guinea and subsequently the rates of penicillin resistance in pneumococci increased worldwide. In Australia, the rate of penicillin resistant pneumococci increased from one per cent in 1984 to 25 per cent in 1997. Reduced susceptibilities to other antimicrobials has also emerged in recent years with the rate of reduced susceptibility to third generation cephalosporins in Australia reaching 13 per cent in 1997.[4] The emergence of multi-drug resistant pneumococci has been an important factor for the development and use of new pneumococcal vaccines.

Ninety serotypes of *S. pneumoniae* identified by the polysaccharide composition of their capsule have been described. Immunity to pneumococcal infection is thought to be serotype specific. Vaccines containing pneumococcal polysaccharides from a varying number of serotypes have been available for many years, with a 23-valent polysaccharide vaccine produced in 1983 being licensed in Australia in 1986 (Table 1). Polysaccharide vaccines are poorly immunogenic in young children.[5] A vaccine in which polysaccharides from seven serotypes coupled to a protein carrier (mutated diphtheria toxoid) was developed to provide an effective vaccine for children and in a trial in the United States of America (USA) in infants aged 2 to 15 months demonstrated an efficacy of 93.9 per cent.[6] This conjugate vaccine was licensed for use in Australia in January 2001 and vaccination of children at high risk commenced in July 2001 (Table 1).

IPD was a notifiable disease in all Australian states and territories in 2002 and data were reported to the National Notifiable Diseases Surveillance System (NNDSS). In addition, the Commonwealth Government funded an enhanced surveillance program for IPD in five jurisdictions and three reference laboratories in 2002.

Methods and materials

Case definition

A case of invasive pneumococcal disease was defined as the isolation from or the detection in blood, cerebrospinal fluid or other sterile site of *Streptococcus pneumoniae*.

Data collection

Invasive pneumococcal disease has been a notifiable disease in some Australian states and territories for several years. In 2001, the Communicable Diseases Network Australia agreed to make IPD notifiable in all states and territories and data were forwarded to the National Notifiable Diseases Surveillance System. Since this required changes to legislation, the data in 2001 was incomplete in some states and territories, but was complete for all jurisdictions for the first time in 2002.

NNDSS data in 2002 comprised core data, which is a set of data collected on all cases of all notifiable diseases, as well as enhanced data specific for IPD. A list of the data fields collected in core and enhanced datasets are shown in Table 2.

Table 1. Recommendations for pneumococcal vaccination, Australia, 2002

Vaccine	23-valent polysaccharide vaccine	7-valent conjugate vaccine
Pneumococcal serotypes	1, 2, 3, 4, 5, 6B, 7F, 8, 9N, 9V, 10A, 11A, 12F, 14, 15B, 17F, 18C, 19A, 19F, 20, 22F, 23F, 33F	4, 6B, 9V, 14, 18C, 19F, 23F
Date implemented	1998	July 2001
Target populations	All individuals aged 65 years and over Individuals with asplenia Immunocompromised patients Aboriginal and Torres Strait Islander people aged 50 years and over Immunocompetent individuals with chronic illness including chronic cardiac, renal or pulmonary disease, diabetes and alcohol-related problems	Tier 1: Indigenous children less than 5 years living in Central Australia Tier 2: Indigenous children aged less than 2 years particularly in rural and remote settings Tier 3: Indigenous children under 2 years living in other settings Non-Indigenous children less than 2 years living in Central Australia Non-Indigenous children with conditions predisposing to pneumococcal infection
Data source	NHMRC Immunisation Handbook 7th edition, 2000	ATAGI recommendations, 2001

There were differences between jurisdictions in the collection of enhanced IPD data. While enhanced data were collected on all cases in the Northern Territory, South Australia, Tasmania, Victoria and Western Australia, other jurisdictions collected enhanced data on limited age groups or in limited areas. In Queensland, enhanced data was collected on cases aged less than five years or more than 50 years in all areas except north Queensland, where enhanced data were collected on all cases. In New South Wales enhanced data were collected on all cases aged less than five years or more than 50 years. In the Australian Capital Territory, enhanced data collection was limited to data on the site of infection and pneumococcal serotype.

NNDSS data for 2002 were analysed by date of disease onset while data in the enhanced datasets were analysed by date of notification.

Clinical presentations were coded as pneumonia, meningitis, bacteraemia, other or unknown. Pneumonia was defined as blood culture positive for *S. pneumoniae* with clinical and/or radiological signs of pneumonia. Meningitis was defined as CSF and/or blood culture positive with supportive CSF findings. Bacteraemia was defined as blood culture positive with no localising signs. 'Other' included detection of *S. pneumoniae* in pleural, peritoneal or joint fluid. More than one clinical presentation could be recorded for each case.

Data analysis

The rates presented in this report were calculated using population data produced by the Australian Bureau of Statistics (ABS). The Estimated Resident Population (ABS 3201.0) in each state and territory and in Australia as a whole, as at 30 June 2002, was used as the denominator in rate calculations. Estimates of the Indigenous Australia population were based on projections from the 2001 census (ABS 3231.0). The ABS calculated projections based on assumptions about future births, deaths and migrations in the Indigenous population and a 'low' and 'high' estimate were reported. The 'low' estimate has been used in this report, consistent with the reporting of other national communicable diseases.

Table 2. Enhanced invasive pneumococcal disease surveillance data, supplied by states and territories, used in this report

Data type	Data fields
Demographic	Date of birth Age Indigenous status: (Aboriginal, Torres Strait Islander, Aboriginal and Torres Strait Islander, other, unknown) Location (optional) Postcode
Risk factors	Premature birth (gestation less than 37 weeks) Congenital abnormality Anatomical or congenital asplenia Immunocompromised (e.g. HIV, lymphoma, transplant, multiple myeloma, nephrotic syndrome, etc.) Chronic illness (e.g. cardiac disease, pulmonary disease, CSF leak, diabetes)
Clinical data	Clinical presentation (pneumonia, meningitis, bacteraemia, other, unknown) Date of onset Death due to invasive pneumococcal disease
Microbiology data	Specimen collection date Date laboratory results issued (report date) Date notification received Specimen type (blood, CSF, pleural fluid, joint fluid, other sterile site) Specimen culture positive or *S. pneumoniae* detected by nucleic acid tests Antibiotic susceptibility (penicillin, cefotaxime/ceftriaxone) Pneumococcal serotype
Vaccination history	Source of vaccination history (validated, not validated, information not collected) Pneumococcal vaccination dates, number of doses and type of vaccine Vaccination status (fully vaccinated for age, partially vaccinated for age, not vaccinated, not applicable, unknown)

The significance of differences in proportions were calculated using the Chi-square test with Yates correction using Epi Info 6.

Results

Notifications to the National Notifiable Diseases Surveillance System

There were 2,271 notifications of IPD to the NNDSS in 2002. The numbers of notification and the notification rate per 100,000 population are shown in Table 3. There was an overall increase in the number of notifications compared with 2001. As noted above, this was principally a product of under-reporting in 2001 due to delays in making IPD a notifiable disease in some jurisdictions.

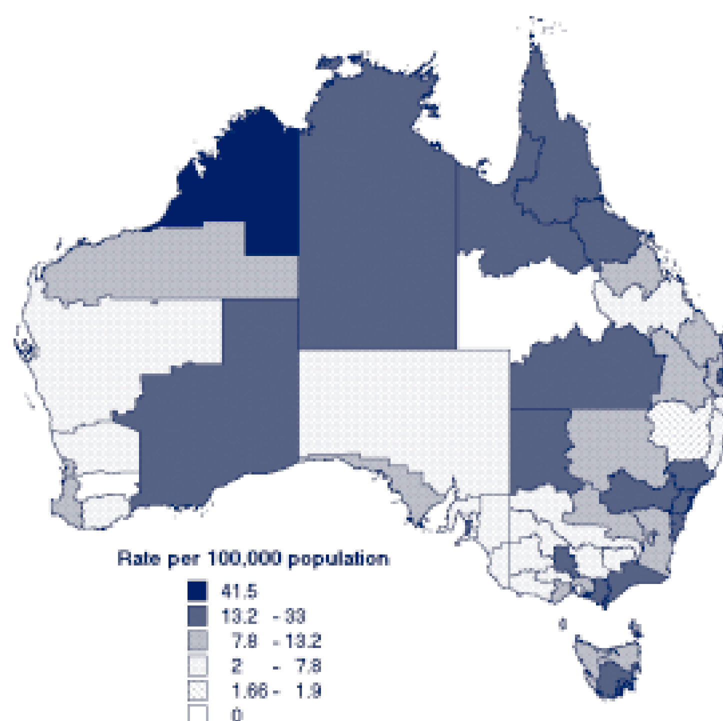
The differences in numbers of notifications between 2002 and 2001 varied by jurisdiction. In states and territories where IPD had been a notifiable disease before 2001, there were small increases noted in some (Queensland, 2%; Western Australia, 1% Tasmania, 3%), while in the Northern Territory there was a marked decrease (33%). This marked decrease is explicable by the large number of IPD reported in the Northern Territory in 2001 (n=97) which was aberrantly high compared to notifications in previous years. Notifications in the Northern Territory in 1998, 1999 and 2000 were 77, 77 and 70, respectively (Heather Cook, personal communication). In other jurisdictions, where IPD had not been a notifiable disease before 2001, there were marked increases in the numbers of notifications in 2002 compared with 2001 (Table 3).

The rates of IPD disease ranged between 9.3 and 13.3 cases per 100,000 population except in the Northern Territory where the rate was 32.8 cases per 100,000 population. When the notification rates of IPD were examined by geographical distribution, variation within states was evident (Map).

Table 3. Notifications and notification rate per 100,000 population, invasive pneumococcal disease, Australia, 2002

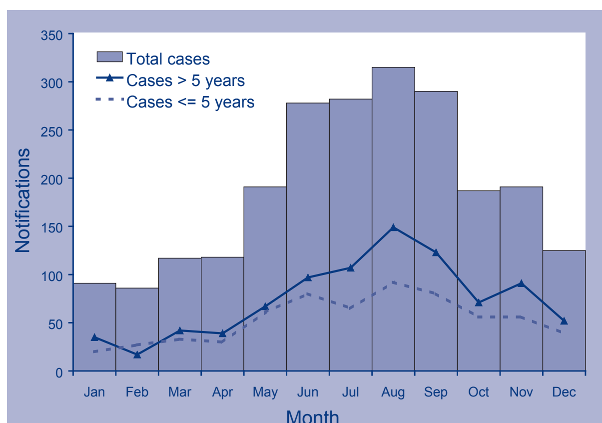
	ACT	NSW	NT	Qld	SA	Tas	Vic	WA	Australia
Notifications	30	841	65	437	174	63	454	207	2,271
Rate per 100,000 population	9.3	12.7	32.8	11.8	11.4	13.3	9.3	10.7	11.5
Notifications in 2001	18	434	97	425	114	61	327	205	1,681
(% change in 2002)	(+66%)	(+94%)	(–33%)	(+3%)	(+52%)	(+3%)	(+39%)	(+1%)	(+35%)

Map. Notification rates of invasive pneumococcal disease, Australia, 2002, by Statistical Division of residence



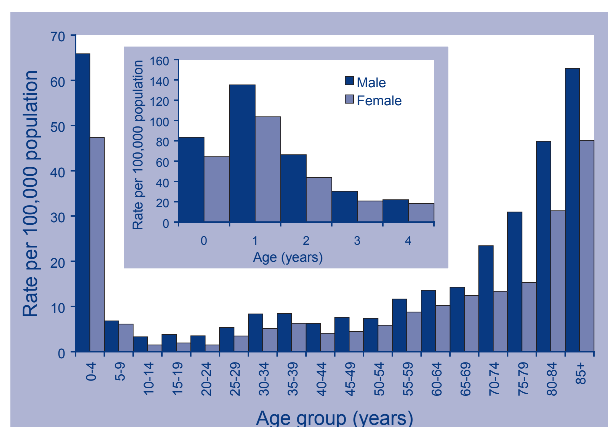
The frequency of cases varied by season with 875 (38%) cases reported in winter months (June to August). The effect of season was more evident on notifications of IPD among children and adults aged more than five years than in younger children aged five years or less (Figure 1).

Figure 1. Notifications of invasive pneumococcal disease, Australia, 2002, by month of report and age group



As previously noted, IPD in Australia is largely a disease of the very young and very old. The highest rates of disease were in children aged less than five years (57.3 cases per 100,000 population) and adults aged more than 85 years (51.7 cases per 100,000 population, Figure 2). Among children aged less than five years, the highest rates were recorded in children aged one year (male, 135 and female, 103 per 100,000 population). In all age groups there were more male than female cases (overall male to female ratio 1.3:1).

Figure 2. Notification rates of invasive pneumococcal disease, Australia, 2002, by age and sex



Enhanced invasive pneumococcal disease surveillance data

Enhanced data were available for 1,929 cases or 85 per cent of notified cases—a similar proportion of cases to that reported on in the last annual report. The percentage of notified cases with enhanced data was lower in New South Wales and Queensland since additional data were only collected for children aged less than five years or adults aged more than 50 years. Since NNDSS data for 2002 was analysed by date of disease onset while data in the enhanced datasets was by date of notification, the enhanced dataset included 35 additional cases in South Australia and three additional cases in Western Australia compared with NNDSS notifications. In all other jurisdictions all notified cases in 2002 were followed up for the collection of enhanced data.

Demographics

The demographic characteristics of cases on which enhanced data were collected are shown in Table 4. In all jurisdictions, except Tasmania, the male to female ratio was between 1.1 to 1.6:1, similar to the 1.3:1 ratio recorded in NNDSS data. In Tasmania, the ratio was 3.2:1. The reasons for this strong preponderance of males among cases from Tasmania were unclear.

The age distribution of the enhanced data is biased by the limited enhanced surveillance in Queensland and New South Wales, where children aged less than five years and adults aged over 50 years only, were followed up.

The enhanced data identified 135 cases of IPD among Indigenous people, which represented seven per cent of all cases, a similar proportion to that in 2001. This represented a national rate of 31 cases per 100,000 population in Indigenous people compared with the national rate of 11.5 cases per 100,000 population. The rates of IPD in Indigenous people were highest in the Northern Territory (85.6 per 100,000 population) and Western Australia (51 per 100,000 population). These rates are estimates only, as under-reporting of Indigenous status continues to be a problem.

Clinical presentation

The clinical presentation was reported in 92 per cent (1,774/1,929) of cases (Table 5).

Pneumonia was the most common clinical presentation (878 cases, 4.5 per 100,000 population) followed by bacteraemia (697 cases, 3.5 per 100,000 population) and meningitis (0.47 per 100,000 population). Presentations of IPD in other

sites accounted for 106 cases (0.54 per 100,000 population). These rates were similar to those reported in 2001.

Clinical presentation varied by age with pneumonia being the most common presentation among the over 65 years (348, 63%) while bacteraemia was the most common presentation among children under five years (378, 49%).

The proportion of IPD presenting as pneumonia was significantly higher in Indigenous children (60%) compared with non-Indigenous children (21%), while presentations of bacteraemia were conversely more common in non-Indigenous children (60%) than Indigenous children (38%, Table 6).

IPD resulted in 175 deaths in Australia in 2002, a case fatality rate of 9.2 per cent (Table 7). The case fatality rate was significantly higher in cases aged more than 65 years (19.4%) compared with children aged less than five years (1.3%, $p<0.0001$). The case fatality rate was higher but not significantly different in non-Indigenous cases (9.5%) compared with Indigenous cases (5%).

Table 5. Clinical presentations of invasive pneumococcal disease, Australia, 2002, by jurisdiction

Clinical presentation*	State or territory							Total
	NSW	NT	Qld	SA	Tas	Vic	WA	
Pneumonia (n)	336	42	97	113	44	127	119	878
%	50	65	33	51	59	28	52	44
Meningitis (n)	31	2	7	12	6	18	17	93
%	5	3	2	6	8	4	4	5
Bacteraemia (n)	264	17	136	81	25	111	63	697
%	40	26	46	37	32	24	28	35
Other (n)	24	4	48	12	1	12	5	106
%	3	6	16	6	1	3	2	5
Unknown (n)	12	0	5	0	0	186	23	226
%	2		2			41	10	11

* Totals may exceed case total and percentages exceed 100 per cent since cases may have had more than one type of clinical presentation.

Table 4. Demographic profile of invasive pneumococcal disease cases reported by enhanced surveillance systems, Australia, 2002, by jurisdiction

		ACT	NSW*	NT	Qld*	SA	Tas	Vic	WA	Total
Number		30	668	65	228	211	63	454	210	1,929
Sex	Male:female ratio	1.1:1	1.3:1	1.3:1	1.6:1	1.1:1	3.2:1	1.3:1	1.4:1	1.5:1
Age	<5 years (n)	19 63%	265 39.7%	21 32%	159 69.7%	91 43%	16 25%	125 28%	66 31%	761 39%
	5 to 64 years	7 23.3%	130 19.3%	39 60%	48 20.6%	71 33.6%	29 46%	192 42%	100 47.6%	616 31%
	≥65 years	4 13.3%	273 40.9%	5 7.7%	21 9.2%	49 23.2%	18 29%	137 30%	44 20.9%	551 30%
Indigenous status	Indigenous	1 3.3%	15 2.2%	49 75.4%	29 12.7%	6 2.8%	0	3 0.6%	32 15.2%	135 7%
	Non-Indigenous	17 56.7%	614 92%	16 24.6%	171 75%	191 90.5%	41 65%	385 85%	162 77%	1,597 83%
	Unknown	12 40%	38 5.8%	0	28 11.8%	14 6.6%	22 35%	66 14.4%	16 7.6%	197 10%

* In New South Wales and Queensland enhanced data were only collected on cases aged less than five years or more than 50 years, except north Queensland where enhanced data were collected on all cases.

Risk factors for pneumococcal disease

Overall, 42 per cent (813/1,929) of cases had a recognised risk factor for pneumococcal disease. The most common of these was chronic illness, which included chronic respiratory, cardiac and renal disease. Immunocompromising conditions such as long-term immunosuppressant use were common among IPD cases. Risk factor categories were defined by the national surveillance working group. Other risk factors were recorded but varied between jurisdictions. More than one risk factor could be recorded for each case. The proportion of cases with an identified risk factor was significantly higher in cases aged five years and above (63%) compared with cases aged less than five years (19%, Chi=147, $p<0.0001$).

The frequency of risk factors for IPD in Indigenous and non-Indigenous people are shown in Table 8. The rates of premature birth and chronic illness were significantly higher in Indigenous children aged less than five years compared with non-Indigenous children. Among cases aged five years or more, the number of immunocompromised patients were significantly higher among non-Indigenous cases than Indigenous cases.

Table 6. Clinical presentations of invasive pneumococcal disease in Indigenous and non-Indigenous children aged less than five years, Australia, 2002

	Indigenous (n=45)		Non-Indigenous (n=647)		Significance of difference*
	n	%	n	%	
Pneumonia	27	60	137	21	p<0.0001
Meningitis	1	2	45	7	ns
Bacteraemia	17	38	391	60	p<0.005
Other	2	4	26	4	ns

* Chi-square test with Yates correction.

Table 7. Case fatality rates for invasive pneumococcal disease, Australia, 2002, by jurisdiction

	NSW	NT	Qld	SA	Tas	Vic	WA	Total
Cases	668	65	228	211	63	454	210	1,899
Deaths	99	4	4	12	11	26	19	175
Total case fatality rate (%)	14.8	6.1	1.8	5.7	17.0	5.7	9.0	9.2
Deaths in cases aged <5 years	4/265	0/21	0/159	1/91	1/16	2/125	2/66	10/743
Total cases aged <5 years(%)	1.5	0.0	0.0	1.0	6.0	1.6	3.0	1.3
Deaths in cases aged >65 years	71/273	1/5	2/21	6/49	4/18	16/137	6/44	106/547
Total cases aged >65 years (%)	26.0	20.0	9.5	12.2	22.0	11.6	13.6	19.4
Deaths in Indigenous people	0/15	3/49	2/29	0/6	0	0/3	2/32	7/134
Total Indigenous cases (%)	0.0	6.0	6.9	0.0			6.2	5.0
Death in non-Indigenous	99/653	1/16	2/199	12/205	11/62	26/451	17/178	168/1,764
Total non-Indigenous + 'unknown' cases (%)	15.2	6.2	1.0	5.9	17.0	5.7	9.5	9.5

Table 8. The frequency of risk factors for invasive pneumococcal disease, Australia, 2002, by age group and Indigenous status

	Cases aged less than five years			Cases aged five years or more		
	Indigenous (n=45)	Non Indigenous (n=647)	Significance of difference	Indigenous (n=88)	Non-Indigenous (n=1,022)	Significance of difference
Premature birth	8 (17%)	37 (6%)	p<0.0005	–	–	–
Congenital abnormality	2 (4%)	15 (2.3%)	ns	1 (1.1%)	2 (0.2%)	ns
Asplenia	0	1 (0.2%)	ns	1 (1.1%)	5 (0.5%)	ns
Immunocompromised	0	18 (2.8%)	ns	6 (6.8%)	171 (16.7%)	p<0.05
Chronic illness	11 (24%)	23 (3.5%)	p<0.0001	38 (43%)	393 (38.4%)	ns

Pneumococcal serotypes causing disease in Australia

Pneumococcal serotypes were identified in 84 per cent (1,624/1,929) of the cases under enhanced surveillance in 2002. Of these, 75 per cent (1,221/1,624) of serotypes were those in the 7-valent conjugate pneumococcal vaccine and 93 per cent (1,517/1,624) were serotypes in the 23-valent polysaccharide pneumococcal vaccine (Table 9).

The frequency of vaccine serotypes in the conjugate and polysaccharide was further analysed in the target age groups for these vaccines and by Indigenous status (Table 10). The proportion of 7-valent conjugate vaccine serotypes was significantly lower in Indigenous children aged less than 2 years (45.8%) than in non-Indigenous children (87.6%, $p<0.0001$). Similarly, the proportion of 23-valent polysaccharide vaccine serotypes in Indigenous cases aged two years and above was significantly lower (88.2%) than in non-Indigenous cases (94.8%, $p<0.05$).

Table 9. Proportion of pneumococcal serotypes in cases of invasive pneumococcal disease, covered by the 7-valent and 23-valent pneumococcal vaccines,* Australia, 2002, by jurisdiction

Vaccine	ACT	NSW	NT	Qld	SA	Tas	Vic	WA	Total
7-valent (n)	16/23	415/534	25/59	164/219	133/160	41/58	293/376	134/195	1,221/1,624
%	69.6	77.7	42	75	83	70	78	68.7	75
23-valent (n)	20/23	501/534	54/59	201/219	153/160	48/58	363/376	177/195	1,517/1,624
%	87	94	92	92	95	83	96	91	93

* As a proportion of serotyped isolates.

Table 10. The proportion of pneumococcal serotypes isolated from cases of invasive pneumococcal disease, which were serotypes in the 7-valent and 23-valent pneumococcal vaccine, Australia, 2002, by age and Indigenous status

	No. (%) serotypes in pneumococcal vaccines					
	Cases aged less than two years with serotypes in 7-valent conjugate vaccine			Cases aged two years or more with serotypes in 23-valent vaccine		
	Indigenous	Non-Indigenous	Significance of difference*	Indigenous	Non-Indigenous	Significance of difference*
ACT	–	4/4 100%	–	1/1 100%	15/17 88.2%	ns
NSW	2/2 100%	114/129 88%	ns	3/3 100%	443/454 98%	ns
NT	4/7 57%	4/5 80%	ns	33/37 89%	9/10 90%	ns
Qld	4/10 40%	68/80 85%	p <0.005	12/16 75%	81/85 95%	ns
SA	–	38/43 88%	–	3/3 100%	95/101 94%	ns
Tas	–	11/11 100%	–	–	34/43 79%	–
Vic	–	52/60 87%	–	3/3 100%	300/313 96%	ns
WA	1/5 20%	21/24 87.5%	p<0.01	20/22 91%	118/132 89%	ns
Australia	11/24 45.8%	312/356 87.6%	p<0.0001	75/85 88.2%	1,095/1,155 94.8%	p <0.05

* Differences tested by Chi square test with Yates correction; ns: not significant.

Vaccination status of invasive pneumococcal disease cases

Data on pneumococcal vaccination were available for half of the cases in 2002. Of the 973 cases with a vaccination history, the majority (687, 69%) were reported as unvaccinated (Tables 11a and 11b). IPD was reported in 9 children aged two years or less who had received vaccination with the 7-valent conjugate vaccine and 151 in older children and adults who had received the 23-valent polysaccharide pneumococcal vaccine.

An analysis of cases recorded as 'fully vaccinated for age' is shown in Tables 12a and 12b. Of the nine infants recorded as fully vaccinated for age, only one was judged to be a 'vaccine failure' that is, having IPD caused by a *S. pneumoniae* serotype in the 7-valent vaccine. This case occurred in the Northern Territory in an Indigenous child with significant risk factors for IPD.

Of the 151 cases of IPD occurring in older children and adults with a history of vaccination within the 23-valent polysaccharide vaccine, 103 were judged to be vaccine failures (Table 12b). The majority of these cases occurred in elderly people with significant risk factors for IPD. History of pneumococcal vaccination in these cases was poorly documented and infection may have occurred many years after vaccination.

Discussion

In 2002, the completeness of IPD reporting improved Australia-wide with notifications received from all jurisdictions for the entire year. The overall increase in notifications relative to 2001 can be accounted for by this improvement in surveillance completeness. In the Northern Territory, however, the number of notifications decreased. It is too early to ascribe this decline to the impact of the childhood pneumococcal vaccination.

Table 11a. Vaccination status of invasive pneumococcal disease cases aged less than two years, Australia, 2002, by age group and jurisdiction

Vaccination status	NSW	NT	Qld	SA	Tas	Vic	WA	Total
Fully vaccinated for age	2	5	2	0	0	0	0	9
Partially vaccinated for age	4	2	2	26	1	1	4	40
Not vaccinated	81	7	6	–	7	42	13	156
Unknown	73	0	104	37	2	35	16	267
Vaccine given								
7-valent	3	7	4	–	1	1	4	20
23-valent	–	*	–	–	–	–	–	–
Unknown	3	0	0	26	0	0	0	29

* One child had two doses of 7vPCV and one dose of PPV.

Table 11b. Vaccination status of invasive pneumococcal disease cases aged two years or more, Australia, 2002, by age group and jurisdiction

Vaccination status	NSW	NT	Qld	SA	Tas	Vic	WA	Total
Fully vaccinated for age	59	10	12	12	8	40	10	151
Partially vaccinated for age	1	5*	1	67	–	–	–	74
Not vaccinated	267	35	8	1	25	123	72	531
Unknown	181	1	93	68	20	213	95	670
Vaccine given								
7-valent	–	2	–	1	–	–	–	3
23-valent	60	15	11	9	8	40	10	154
Unknown	0	1	2	69	0	0	0	72

Data not available from the Australian Capital Territory.

* Partially vaccinated defined in the Northern Territory for cases >2 years as PPV vaccination more than five years before disease onset.

Recent publications have demonstrated the effectiveness of the pneumococcal vaccines in reducing IPD at a population level. An analysis of the burden of IPD nearly two years after the introduction of the conjugate vaccine in the USA has demonstrated a 69 per cent reduction in IPD in children less than two years of age. Interestingly there was also a decline in IPD in older age groups, which was taken as evidence for decreased transmission from children. This decline, together with prevention of disease caused by drug resistant strains and by disease caused by vaccine-related serotypes, provides reasons for optimism for the control of IPD in communities where children receive pneumococcal vaccination.[7] In Native American children the same conjugate vaccine has shown a primary efficacy of 76.8 per cent in communities with very high burden of IPD, comparable to those experienced in Central Australian Indigenous communities.[2] In addition, evidence has been presented recently that there is a small but significant impact of vaccination on otitis media with reductions of 7.8 per cent in visits to medical practitioners, reductions of 5.7 per cent in antibiotic prescriptions for otitis media, and reductions in repeated episodes of otitis media and tube replacements.[8] Continued application of the conjugate vaccine in Australian Indigenous communities may also reduce IPD burden in the unvaccinated groups and the burden of non-invasive pneumococcal disease, for example otitis media.

Nevertheless there are continuing questions about the impact of vaccines, particularly whether vaccine pressure will result in disease caused by non-vaccine serotypes and a gradual diminution of vaccine efficacy.[9] Whitney and colleagues reported that while disease caused by vaccine and vaccine related serotypes decreased (by 78% and 50% respectively), disease caused by non-vaccine serotypes increased by 27 per cent, although this increase did not reach statistical significance.[7] In the high-prevalence Native American setting which had a relatively high proportion of non-vaccine serotypes circulating, there was an increase in IPD caused by non-vaccine serotypes following the introduction of the 7-valent vaccine, but again this difference was not significant.[2] It is essential to continue the surveillance of serotypes causing disease in Indigenous communities to monitor whether IPD from non-vaccine serotypes are increasing, compromising the impact of the 7-valent vaccine.

Table 12a. Details of the cases of invasive pneumococcal disease that occurred in those fully vaccinated for age with the 7-valent conjugate pneumococcal vaccine, Australia, 2002, by jurisdiction

	NSW	NT	Qld	Total
Number	2	5	2	9
Age range (years)	0–1	0–1	0–1	0–1
Indigenous	2 (100%)	5 (100%)	2 (100%)	9 (100%)
Risk factors present	0	4 (80%)	2 (100%)	6 (67%)
7-valent vaccination confirmed	2	5	2	9
Serotypes in 7-valent vaccine/ number with known serotype	0/2	2/4	0/2	2/8
Number of vaccine failures*	0	1	0	1

* Where vaccination was confirmed and disease was caused by a serotype in the appropriate vaccine.

Table 12b. Details of the cases of invasive pneumococcal disease that occurred in those fully vaccinated for age with the 23-valent polysaccharide vaccine, Australia, 2002, by jurisdiction

	NSW	NT	Qld	SA	Tas	Vic	WA	Total
Number	59	10	12	12	8	40	10	151
Age range (years)	53–93y	2–82y	30–73y	24–87y	58–80y	14–89y	50–86y	2–93y
Indigenous	2	10	8	1	0	0	5	26
Risk factors present	51	7	11	11	8	31	4	123
23-valent vaccination confirmed	57	10	11	9	8	38	6	139
Serotypes in 23 valent vaccine/ no. with known serotype	45/52	8/9	9/12	7/8	3/7	34/35	4/8	110/131
Number of vaccine failures*	43	8	7	5	3	34	3	103

* Where vaccination was confirmed and disease was caused by a serotype in the appropriate vaccine.

Pneumococcal disease in Australia is also a disease of the elderly with 26 per cent of cases aged 65 years and over and rates of 51.7 cases per 100,000 population in those aged 85 years or over. Current recommendations in Australia are for all non-Indigenous Australians over 65 years and all Indigenous Australians over 50 years to receive 23-valent polysaccharide pneumococcal vaccination. Re-vaccination is recommended every five years.[10] In Victoria, vaccine has been offered free to all residents aged 65 years or more since 1998[11] and coverage in a recent survey was estimated to be between 47 and 51 per cent.[12] The Northern Territory has additionally recommended the 23-valent vaccine for all Aboriginal people 15 years and older since mid-2000 with coverage rates calculated, via a vaccine register capturing 50 per cent of distributed vaccine, to be 21 per cent in the 15–49 year age group and 50 per cent in those 50 years or older.[13] Coverage in the rest of Australia in 2002 was estimated to be between 25 and 28 per cent.

Two recent systematic reviews of pneumococcal polysaccharide vaccine effectiveness in adults[14,15] found protective effects of the vaccine against disease and mortality in non-industrialised countries, but no protective effect except possibly against pneumococcal bacteraemia in industrialised countries. Similar results were evident in a retrospective cohort study in elderly Americans.[16] Likewise, recent studies of the 23-valent polysaccharide vaccine among Native American adults also shows a low effectiveness in these communities with high pneumococcal disease burden and high rates of chronic diseases.[17] However, evidence from other case control and cohort studies indicates a vaccine effectiveness of 50–80 per cent in preventing pneumococcal bacteraemia or invasive disease in the elderly.[18] Difficulties in the diagnosis of pneumococcal pneumonia, variation in IPD epidemiology, and circulating serotypes, decreased vaccine efficacy in the presence of certain and multiple risk factors, and uncertainties over the duration of vaccine-induced immunity complicate debate over effectiveness. While vaccine induced antibody responses appear to decline after three years and can be boosted by re-vaccination, the clinical efficacy of re-vaccination has not been demonstrated.[19] A cost effectiveness analysis in Australia[20] indicated that extending pneumococcal vaccination for all persons aged 50 to 64 years and to all Indigenous people aged between 15 and 49 years would be cost effective. Enhanced surveillance of IPD in the adult Australian population will assist with evaluating present recommended vaccine schedules and funded programs.

The use of pneumococcal vaccines has been promoted as a means to reduce the need for the use of antibiotics and so reduce the spread of antibiotic resistant pneumococci.[21] After two years of use of the conjugate vaccine in American children, declines in rates of disease caused by penicillin resistant and penicillin susceptible strains were not significantly different, although there was a small decline in the overall proportion of resistant strains.[7] The impacts of vaccination on antibiotic resistance in the pneumococci will not be evident until the vaccine is more widely available. Reports on antibiotic resistance from the enhanced IPD surveillance in Australia, in 2002, will be reported separately.

Surveillance of IPD in children aged less than five years will continue in Australia, supported by laboratory surveillance of serotypes and antibiotic resistance. A reduction in the disease burden in Indigenous children as the vaccine coverage in the target population increases seems likely with a possible reduction in Indigenous adults as well. On-going surveillance will be essential to monitor serotype replacement and the impact of vaccination on antibiotic susceptibility.

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Laboratory surveillance of invasive pneumococcal disease in Australia in 2001 to 2002—implications for vaccine serotype coverage

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Abstract

This paper reports the results of comprehensive laboratory surveillance of invasive pneumococcal disease (IPD) in Australia during 2001 and 2002. The 7-valent conjugate pneumococcal vaccine was introduced for high risk paediatric groups, including Indigenous children, in late 2001. Of 1,355 isolates from non-Indigenous children, 86 per cent belonged to serotypes and 93 per cent to serogroups represented in the 7-valent pneumococcal conjugate vaccine. Thirteen per cent and 24 per cent of isolates had reduced susceptibility to penicillin and erythromycin, respectively and of these, more than 99 per cent belonged to serogroups represented in the 7-valent vaccine. Of the 1,504 isolates from non-Indigenous adults, 96 per cent belonged to serotypes included in the 23-valent polysaccharide vaccine; 14 per cent and 15 per cent had reduced susceptibility to penicillin and erythromycin, respectively and more than 95 per cent of these belonged to serotypes included in the 7-valent conjugate vaccine. In Western Australia and the Northern Territory (the only states for which Indigenous status was consistently available), there were 29 cases of IPD in Indigenous children, of which 21 were due to 7-valent vaccine serotypes in 2001, compared with 24 cases, including 10 due to vaccine serotypes, in 2002. This represents a statistically significant increase in the proportion of total isolates due to non-vaccine serotypes ($\chi^2 = 3.93$, $p = 0.048$) following the introduction of the 7-valent conjugate vaccine, principally due to serotypes 7F and 12F. The number of episodes due to penicillin resistant isolates decreased from nine in 2001 to two in 2002. Ninety per cent of isolates from Indigenous adults were included in the 23-valent polysaccharide vaccine and six per cent and five per cent had reduced susceptibility to penicillin and erythromycin, respectively. Conjugate pneumococcal vaccines can be expected to reduce the incidence of IPD due to vaccine serotypes in vaccinated children and potentially, their adult contacts. It may also impact favourably on the incidence of IPD due to penicillin and erythromycin resistant strains. Continued surveillance of both serotype distribution and antibiotic susceptibility are required to identify serotype replacement by non-vaccine serotypes and to monitor the overall impact of current and future vaccine programs on invasive pneumococcal disease in Australia. *Commun Dis Intell* 2003;27:478–487.

Keywords: disease surveillance, pneumococcal disease, Streptococcus pneumoniae

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Introduction

Streptococcus pneumoniae is a major cause of morbidity and mortality worldwide.[1–4] Knowledge of the serotypes responsible for invasive pneumococcal disease (IPD) is essential for planning and monitoring the introduction of vaccines against pneumococcus.[5,6] Although laboratory surveillance of IPD was already taking place independently in Australian states and territories,[7–10] the commencement of funding of laboratory surveillance by the Commonwealth Government in January 2002 has facilitated a national approach to laboratory surveillance of IPD. Data on serotypes responsible for IPD is now comprehensive, although data on antimicrobial resistance remains incomplete.

In late 2001, a 7-valent conjugate pneumococcal vaccine program was introduced for high risk paediatric groups including Indigenous children. We report on the early impact this program has had on IPD in Indigenous children in Western Australia and the Northern Territory and the potential impact in the wider community.

Antimicrobial resistance in invasive pneumococci is an emerging problem in Australia.[11] Laboratory data on resistance to penicillin and erythromycin is presented although these data are not available for Victoria and are largely generated from the routine clinical laboratories in other states such as New South Wales. Analysis has necessarily been limited to categorisation into fully susceptible or resistant (intermediate and high level resistance) to penicillin and erythromycin. The serotype coverage for the future 11-valent conjugate pneumococcal vaccine and the existing 23-valent polysaccharide vaccine have been examined.

Methods and materials

Case definition

A case of IPD was defined as isolation of *Streptococcus pneumoniae* from a normally sterile body site (e.g. blood culture, cerebrospinal fluid, joint fluid, etc). A new episode was deemed to occur if the isolate occurred more than 14 days from the previous positive culture.

Data sources and collection

A network of pneumococcal laboratories in Australia (see list of participating laboratories) obtained pneumococcal isolates referred from all major private and public microbiology laboratories in Australia. Isolates were referred for storage and later serotyping at one of the three designated pneumococcal typing laboratories. Indigenous status data was obtained from enhanced surveillance data and was able to be efficiently linked to laboratory data for Western Australia and the Northern Territory. IPD data for Indigenous patients from Western Australia and the Northern Territory were combined for the purpose of data analysis. In other states, the Indigenous status data was either incomplete or could not be linked efficiently with laboratory data. Where Indigenous status was unknown, patients were deemed to be non-Indigenous for the purposes of data analysis. This may have resulted in some Indigenous patient data from New South Wales, South Australia, Victoria and Queensland being included in the non-Indigenous Australian paediatric and adult data that has been presented. Data from the Australian Capital Territory and Tasmania were only available for 2002 and so were not included in the analysis.

Serotyping

Pneumococcal serotyping was performed at the Pneumococcal Reference Laboratory of Queensland Health Scientific Services (Western Australia, the Northern Territory, Queensland), the Children's Hospital at Westmead's New South Wales Pneumococcal Reference Laboratory (New South Wales) and the Microbiological Diagnostic Unit (Victoria, South Australia). Serotyping was performed by the Quellung reaction using antisera from the Statens Seruminstitut, Copenhagen, Denmark.

Susceptibility testing

Susceptibility testing was performed by a range of different methods. In New South Wales and South Australia the available results were from routine diagnostic laboratories. These laboratories used NCCLS disc diffusion, CDS disc diffusion or agar dilution susceptibility testing methods. Most laboratories also confirmed penicillin resistance using the E test method. Results from Queensland and Western Australia were performed using NCCLS disc diffusion and E test methods in a reference laboratory.

Isolates were categorised as fully sensitive to penicillin or resistant (includes intermediate and high level resistance using NCCLS breakpoints). Isolates to erythromycin were categorised as either sensitive or resistant (those with intermediate resistance were categorised as resistant).

Statistical analysis

Yates corrected Chi square test was used for univariate analysis using Epi Info statistical software V6.02 (Centers for Disease Control and Prevention, USA). Patients were classified as children if their age was under 15 years and adults if they were 15 years of age or over.

Results

Serotypes responsible for invasive pneumococcal disease and antimicrobial resistance in non-Indigenous Australian children

Data from Queensland, Victoria, New South Wales, South Australia, Western Australia and the Northern Territory for children were initially analysed separately but then combined, as the serotype distributions were comparable. Of the 1,383 episodes recorded by the laboratory surveillance system, isolates from 1,355 (98%) were serotyped. The serotype distribution and vaccine serotype coverage for children for the 7-valent and future 11-valent conjugate pneumococcal vaccines is illustrated in Figure 1. Those serotypes not covered in the conjugate vaccines are illustrated in Table 1. Eighty-six per cent of isolates were a serotype match for the 7-valent vaccine and 93 per cent of isolates were a serogroup match. The future 11-valent vaccine (addition of serotype 1, 7F, 5 and 3) provided an additional three per cent serotype coverage. The serotype distribution for neonates (<28 days of age) was found to be different when compared to older children (Table 2).

Of the 1,383 isolates from children, 1,140 were serotyped and had susceptibility results recorded for penicillin. One hundred and forty-six (12.8%) had reduced susceptibility to penicillin. Over 99 per cent of the penicillin resistant isolates belonged to serogroups in the 7-valent conjugate vaccine (Table 3).

Table 1. Non-conjugate vaccine serotypes from non-Indigenous children less than 15 years, Australia, 2001 to 2002

Serotype	23-valent polysaccharide vaccine serotype								23-valent polysaccharide vaccine serogroup		Non-vaccine serotypes					
	15B	8	22F	17F	11A	33F	12F	10A	15C	22A	35F	13	35B	38	16F	NT
Number of isolates	8	1	5	1	3	3	3	2	6	1	2	1	2	7	4	3
Cumulative (%)	15.4	17.3	26.9	28.8	34.6	40.4	46.2	50.0	61.5	63.5	67.3	69.2	73.1	86.5	94.2	100.0

NT Non-typable

Table 2. Non-Indigenous neonatal (<28 days) serotypes, Australia, 2001 to 2002

Serotype	7-valent conjugate vaccine serotype							7-valent conjugate vaccine serogroup		11-valent conjugate vaccine serotype		23-valent polysaccharide vaccine serotype		Non-vaccine serotype	
	14	6B	19F	18C	4	23F	9V	19A	6A	1	7F	22F	10A	16F	38
Total number	1	2	3	1	1	0	1	0	1	0	1	1	1	1	1
Cumulative %	6.7	20.0	40.0	46.7	53.3	53.3	60.0	60.0	66.7	66.7	73.3	80.0	86.7	93.3	100.0

Table 3. Penicillin resistant serotypes in non-Indigenous Australian children less than 15 years, 2001 to 2002

Serotype	7-valent conjugate vaccine serotype					7-valent conjugate vaccine serogroup		23-valent polysaccharide vaccine serotype
	19F	9V	6B	14	23F	19A	6A	15C
Number of isolates	40	33	30	22	9	9	2	1
Cumulative (%)	27.4	50.0	70.5	85.6	91.8	97.9	99.3	100.0

The percentage of each serotype that was resistant to penicillin varied by state. The percentage resistance also varied for each serotype with a very high proportion of serotype 9V isolates being resistant to penicillin whilst others such as serotype 14 had low percentage resistance (Figure 2).

Serotyping results and erythromycin susceptibility was available for 1,092 of 1,383 isolates from children. Two hundred and sixty (23.8%) of these were resistant to erythromycin. The predominant serotype responsible for erythromycin resistance was serotype 14 (Table 4). All erythromycin resistant strains belong to serogroups contained in the 7-valent conjugate vaccine.

Serotypes responsible for invasive pneumococcal disease and antimicrobial resistance in non-Indigenous Australian adults

Serotype data from Queensland, Victoria, New South Wales, South Australia, Western Australia and Northern Territory adults were combined. The serotype distribution and serotype coverage for adults for the 7-and 11-valent conjugate pneumococcal vaccines is illustrated in Figure 3. The serogroup coverage for the future 11-valent conjugate vaccine is 85.5 per cent. The 23-valent polysaccharide vaccine provides 96 per cent serotype coverage (Figure 4). Serotype 16F is the predominant serotype not covered by the 23-valent vaccine (Table 5).

Figure 2. Percentage of penicillin resistant isolates of each serotype in non-Indigenous children less than 15 years, Australia, 2001-2002, by state

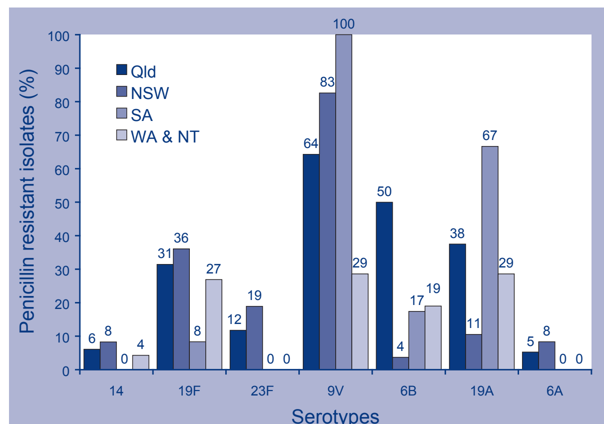


Figure 1. Serotype distribution of *Streptococcus pneumoniae* from non-Indigenous Australian children less than 15 years, 2001 to 2002

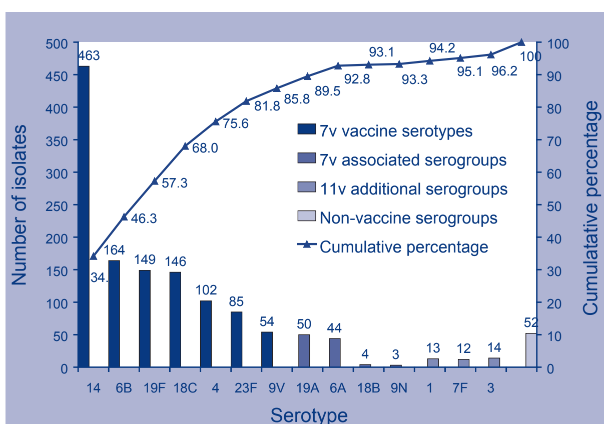


Figure 3. Conjugate vaccine related serogroups of *Streptococcus pneumoniae* responsible for invasive pneumococcal disease in non-Indigenous Australian adults, 2001-2002

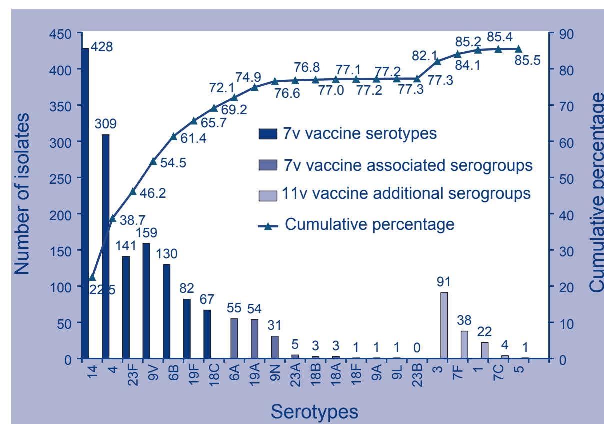


Table 4. Erythromycin resistant serotypes in non-Indigenous Australian children less than 15 years, Australia, 2001 to 2002

Serotype	7-valent conjugate vaccine serotype							7-valent conjugate vaccine serogroup	
	14	19F	6B	23F	9V	18C	4	19A	6A
Number of isolates	170	38	30	11	1	1	0	6	3
Cumulative (%)	65.4	80.0	91.6	95.8	96.2	96.6	96.6	98.9	100.0

Of the 1,948 isolates from adults, 1,504 were serotyped and had susceptibility results recorded for penicillin. Two hundred and four (13.6%) had reduced susceptibility to penicillin. Over 95 per cent of the penicillin resistant isolates belonged to serogroups in the 7-valent conjugate vaccine (Table 6). Serotyping results and erythromycin susceptibility was available for 1,439 of 1,948 isolates in adults. Two hundred and fourteen (14.9%) of these were resistant to erythromycin. The predominant serotype responsible for erythromycin resistance was serotype 14 (Table 7). Over 95 per cent of erythromycin resistant strains belong to serogroups contained in the 7-valent conjugate vaccine and 99 per cent of the 23-valent polysaccharide vaccine.

Figure 4. Additional 23-valent vaccine-related serotypes responsible for IPD in non-Indigenous Australian adults, 2001-2002

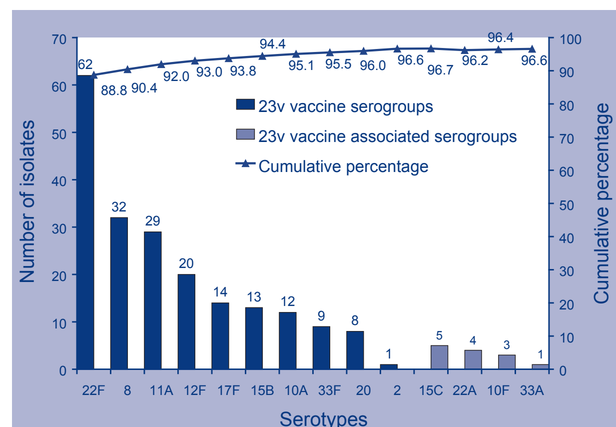


Table 5. Non-vaccine serotypes from non-Indigenous adults less than 15 years, Australia, 2001 to 2002

Serotypes	13	34	16F	38	25	35*	35B	35F	48	NT
Number of Isolates	3	3	26	6	1	5	3	6	1	9
Cumulative (%)	96.8	97.0	98.4	98.7	98.7	99.0	99.2	99.5	99.5	100.0

NT Non-typable.

* Serogrouping only, performed.

Table 6. Penicillin resistant serotypes in non-Indigenous Australian adults greater than 15 years, 2001 to 2002

Serotype	7-valent conjugate vaccine serotype					7-valent conjugate vaccine serogroup		11-valent conjugate vaccine serotype	23-valent polysaccharide vaccine serotype			23-valent poly-saccharide vaccine serogroup
	9V	14	6B	19F	23F	19A	6A	3	12F	15B	22F	15C
Number of isolates	88	39	26	22	15	5	3	2	1	1	1	1
Cumulative %	43.1	62.3	75.0	85.8	93.1	95.6	97.1	98.0	98.5	99.0	99.5	100.0

Table 7. Erythromycin resistant serotypes in non-Indigenous Australian adults greater than 15 years, 2001 to 2002

Serotype	7-valent conjugate vaccine serotype						7-valent conjugate vaccine sero-group			11-valent conjugate vaccine serotype	23-valent polysacch-aride vaccine serotype		23-valent polysacch-aride vaccine sero-group	Non-vaccine sero-type
	14	6B	19F	23F	9V	4	19A	6A	9N	3	22F	33F	10F	NT
Number of isolates	129	32	23	14	2	1	2	3	1	2	1	1	1	2
Cumulative %	60.3	75.2	86.0	92.5	93.5	93.9	94.9	96.3	96.7	97.7	98.1	98.6	99.1	100.0

Serotypes responsible for invasive pneumococcal disease in Indigenous children from Western Australia and the Northern Territory and the potential impact of the 7-valent conjugate vaccine program

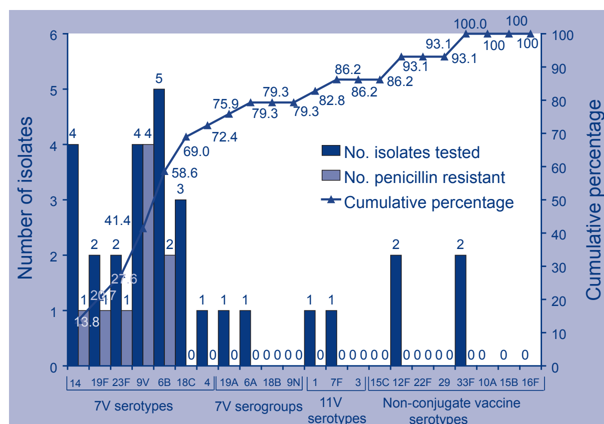
Of the 57 cases of IPD in Indigenous children from 2001 and 2002, 53 isolates were serotyped and sensitivity tested. Of the four cases without these data two cases were from 2001 and two cases were from 2002. In 2001, 29 cases of IPD occurred of which 21 were serotypes in the 7-valent vaccine. In 2002 there were 24 cases of IPD, 10 of which were serotypes in the 7-valent vaccine. The rate of disease for 7-valent vaccine and non-vaccine serotypes was not statistically significantly different between 2001 and 2002. The proportion of 7-valent vaccine serotypes was, however, significantly lower in 2002 compared to 2001 (χ^2 = 3.93, p = 0.048). The serotype and penicillin resistance data for 2001 and 2002 are represented in Figures 5 and 6. There were nine penicillin resistant isolates in 2001 and only two in 2002. This difference was not statistically significant. No difference was seen in erythromycin resistance with two cases occurring in each year.

Serotypes responsible for invasive pneumococcal disease and antimicrobial resistance in Indigenous adults from Western Australia and the Northern Territory

Laboratory data from Western Australia and the Northern Territory for Indigenous adults were combined. The serotype distribution and serotype coverage for Indigenous adults for the 7- and the future 11-valent conjugate pneumococcal vaccines is illustrated in Figure 7. The serogroup coverage for the 11-valent conjugate vaccine was 61.3 per cent. The 23-valent polysaccharide vaccine provided 90.1 per cent serogroup coverage (Figure 8). The serotypes not covered by the 23-valent vaccine are shown in Table 8.

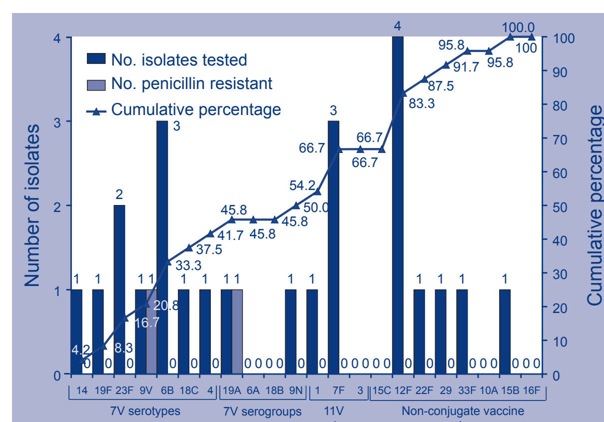
Of the 115 isolates from Indigenous adults in 2001 and 2002, 111 were serotyped and had susceptibility results recorded for penicillin. Seven (6.3%) had reduced susceptibility to penicillin. All the penicillin resistant isolates belonged to serogroups in the 7-valent conjugate vaccine. Serotyping results and erythromycin susceptibility were available for 104 of 115 isolates in adults. Five (4.8%) of these were resistant to erythromycin. Only one of the five erythromycin resistant strains belonged to serotypes contained in the 7-valent conjugate vaccine and two of the five were serotypes in the 23-valent vaccine.

Figure 5. Serotypes and penicillin resistance in Indigenous children* less than 15 years, 2001, (pre 7v vaccine introduction)



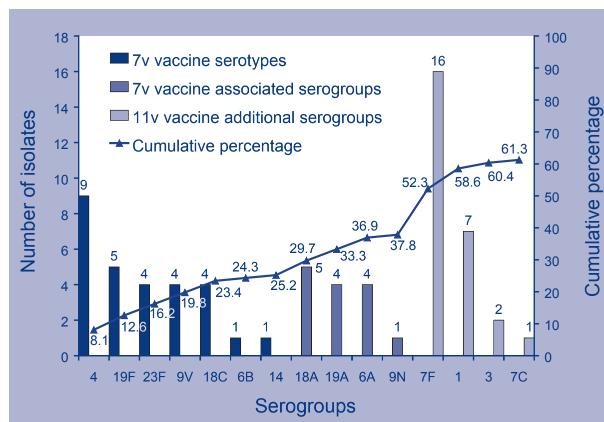
* Data from Western Australia and the Northern Territory combined.

Figure 6. Serotypes and penicillin resistance in Indigenous children less than 15 years,* 2002 (post 7v vaccine introduction)



* Data from Western Australia and the Northern Territory combined.

Figure 7. Conjugate vaccine-related serogroups of *Streptococcus pneumoniae* in Indigenous adults,* 2001-2002



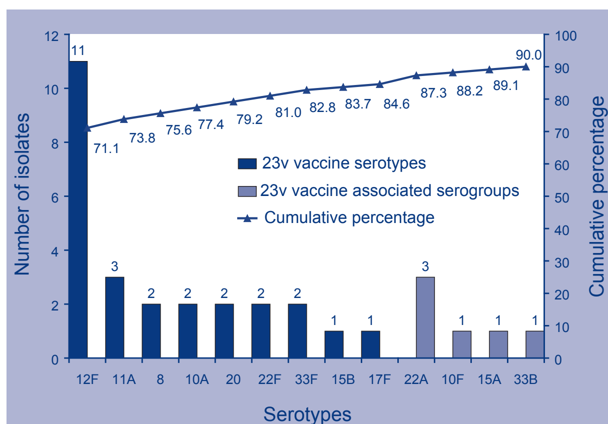
* Data from Western Australia and the Northern Territory combined.

Table 8. Non-vaccine serotypes in Indigenous adults in Western Australia and the Northern Territory, 2001 to 2002

Serotype	13	29	31	34	16F	38	25	35*	35B	35F	48	NT
Number of isolates	0	1	3	0	3	2	0	0	1	0	0	1
Cumulative %	90.1	91.0	93.7	93.7	96.4	98.2	98.2	98.2	99.1	99.1	99.1	100.0

NT Non-typable.

Figure 8. Additional 23-valent vaccine-related serotypes of *Stretococcus pneumoniae* in Indigenous adults,* 2001-2002



* Data from Western Australia and the Northern Territory combined.

Discussion

The impact of the 7-valent conjugate pneumococcal vaccine on invasive pneumococcal disease has now been clearly demonstrated in the United States of America (USA).[12] Recently, the National Health and Medical Research Council (NHMRC) has recommended 7-valent pneumococcal conjugate vaccine for all children in Australia as part of their primary immunisation series.[13] It is therefore vital to have baseline data on serotype prevalence in Australian children.

One limitation of the current laboratory surveillance system has been the incompleteness of Indigenous status data and/or the inability to link Indigenous status data with laboratory surveillance data in states other than Western Australia and the Northern Territory. We have made an assumption in combining data from other states that if the Indigenous status was not known, that the patient was not Indigenous. We believe that the overall vaccine serotype coverage results are valid for non-Indigenous children and adults given the relatively small numerical contribution of Indigenous cases to the combined data from these states. Furthermore, we believe that the accidental inclusion of some Indigenous patients in the serotype analysis is likely to result in our vaccine serotype coverage rates for non-Indigenous adults and children being more conservative (i.e. underestimating serotype coverage).

The data for non-Indigenous Australian children for the years 2001 to 2002 clearly demonstrates a high percentage serotype coverage (86%) and even higher for serogroup coverage (93%). There is now evidence of at least some cross-protection for serogroups contained in the vaccine.[12] It is therefore reasonable to predict that Australia will see a significant decline in IPD in children if vaccine programs based on the new NHMRC guidelines are funded and successfully implemented. There were relatively few cases of IPD in neonates, however it was interesting to note the lower 7-valent vaccine serotype coverage in this age group. This may be a result of the fact that the infection may be acquired from the mother during or soon after birth.

The serotype distribution for penicillin resistant strains in non-Indigenous children is also favourable with over 99 per cent of serotypes covered in the vaccine. There is evidence that a reduction in the rate of IPD due to penicillin resistant strains can be expected with the introduction of the 7-valent conjugate vaccine.[12] It is important to note that the efficacy of the vaccine for the prevention of IPD varies with the serotype. Vaccine efficacy in protecting against disease caused by serotypes 19F and 6B (which are two of the most common strains associated with penicillin resistance in our study) is lower than other serotypes in the 7-valent vaccine, both being around 85 per cent.[14] This could result in a higher proportion of penicillin resistant strains after vaccine implementation, although there would still be a marked reduction in the absolute numbers of cases of these serotypes. The trends in percentage penicillin resistance of each serotype is a very interesting observation. The potential for some penicillin resistant serotypes such as 9V to completely replace penicillin sensitive strains of the same serotype is very concerning. Overall, the percentage of isolates with resistance to penicillin in non-Indigenous children in Australia is 12.8 per cent but erythromycin resistance is almost double this at

23.8 per cent. This is due to the fact that in addition to the predominant penicillin resistant strains in children (19F and 6B), which are often also resistant to macrolides, there is a high proportion of serotype 14 isolates that have isolated macrolide resistance. Macrolide resistance is likely to be reduced by the introduction of the 7-valent conjugate vaccine.

Although it is unlikely that the 7-valent conjugate vaccine will be recommended for use in adults, it has now become clear that a reduction in the prevalence of these vaccines serotypes in adults in the community may occur as a result of vaccination of children through the development of herd immunity.[12] Our serotype data suggests that this could have a significant impact in the Australian adult population as almost 70 per cent of cases of adult IPD are due to serotypes within the 7-valent vaccine. This figure is even higher (77%) when serogroups are considered. One problem with the current 23-valent polysaccharide vaccine is the need to give repeat doses. The future 11-valent conjugate vaccine does appear to have acceptable serotype coverage in non-Indigenous adults with the vaccine serogroups covering over 85 per cent of strains. This raises the interesting possibility of the use of an 11-valent conjugate vaccine in non-Indigenous adults if long term immunity can be demonstrated with this vaccine in this target population.

The predominant serotype responsible for penicillin resistance in non-Indigenous adults appears to be different from those in non-Indigenous children. Serotype 9V and 14 are the two most common serotypes associated with penicillin resistance in adults. Both serotypes were shown to have significantly reduced in frequency (in adults over 65 years of age) following introduction of the 7-valent conjugate vaccine in children the USA.[12] This holds the exciting possibility of reducing penicillin resistance in elderly Australians by use of the 7-valent vaccine in children. The percentage of macrolide resistance seen in non-Indigenous adults is almost half that seen in children. This is partly due to the fact that the principal penicillin resistant serotypes in adults (9V and 14) are generally not also resistant to macrolides.

One has to be cautious in attributing the change in numbers of cases of IPD in Indigenous children in Western Australia and the Northern Territory to the impact of the 7-valent conjugate vaccine program. However, it is encouraging that there has been a significant shift in serotypes away from those in the 7-valent vaccine. This however, could result both from a reduction in numbers of 7-valent vaccine serotype isolates, but also an increase in the number of non-7-valent vaccine serotypes. The predominant increase in non-vaccine serotypes was due to serotype 7F and 12F. At this stage it is difficult to determine the relevance of this finding as natural fluctuations in the numbers of these isolates could occur even in the absence of a 7-valent vaccine program. It does however reinforce the need for careful monitoring to ensure that serotype replacement does not become a significant problem in the future. Another encouraging finding is that the number of cases due to penicillin resistant isolates also fell, although this change did not achieve statistical significance.

The 23-valent polysaccharide vaccine continues to provide good serotype coverage for adults including Indigenous adults in Western Australia and the Northern Territory. Of interest is the relatively low rate of penicillin and erythromycin resistance of pneumococcal isolates from Indigenous adults compared to non-Indigenous adults in Australia.

The continued laboratory surveillance on IPD is a vital component of the pneumococcal vaccine strategy for Australia. The funding of this surveillance has facilitated a national approach to surveillance and reporting of this important reference laboratory work. Our surveillance suggests that implementation of the NHMRC recommendation to introduce 7-valent conjugate vaccine to all children in Australia is likely to lead to major benefits for both children and adults in this country.

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List of contributors to pneumococcal laboratory surveillance

New South Wales

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Surveillance of antibiotic resistance in *Neisseria gonorrhoea*e in the WHO Western Pacific Region, 2002

WHO Western Pacific Gonococcal Antimicrobial Surveillance Programme

Abstract

The World Health Organization's Western Pacific Region (WHO WPR) long standing programme for surveillance of antimicrobial resistance in *Neisseria gonorrhoeae,* GASP, continued in 2002. Seventeen countries contributed data on about 11,500 gonococci by determining susceptibility patterns using standardised methodologies. Resistance to quinolone and penicillin antibiotics remained widely dispersed and at historically high levels. Gonococci with decreased susceptibility to third generation cephalosporins were again observed in several centres. Spectinomycin resistance was infrequently encountered. Control of gonorrhoea in the WHO WPR is compromised by the further reduction in options for cheap and effective treatment of gonorrhoea. *Commun Dis Intell* 2003;27:488–491.

Keywords: surveillance; Neisseria gonorrhoeae*; antimicrobial resistance; gonorrhoea; antibiotics; quinolones; penicillins; spectinomycin; cephalosporins*

Introduction

Antimicrobial resistance in *Neisseria gonorrhoeae* has deleterious consequences for the successful treatment of the individual patient and on control measures for gonococcal disease. The potential for emergence and spread of antibiotic resistant gonococci is well documented in the World Health Oragnization Western Pacific Region (WHO WPR). Resistance to the quinolone group of antibiotics is the most recent example of antimicrobial resistance compromising the efficacy of a gonococcal treatment, and quinolone resistant gonococci (QRNG) have now spread widely within and beyond the WPR. Ominously, there have been instances of altered susceptibiity to third generation cephalosporins in the region[1,2] and recently these strains have also appeared in centres outside the WHO WPR.[3] Currently in the WHO WPR it is difficult to define cheap and effective standard treatments for gonorrhoea. It thus becomes increasingly important to have available accurate data on antimicrobial resistance in the gonococcus in order to guide selection of an appropriate standard treatment schedule.

The WHO WPR Gonococcal Antimicrobial Surveillance Programme (GASP) is a continuing program of susceptibility surveillance in the Region and has published surveillance data annually since 1992.[4] This communication provides an analysis of surveillance of antimicrobial resistance in *N. gonorrhoeae* in the WHO WPR in 2002.

Methods

The methods used by the WHO WPR GASP have been published[5] and provide full details of the source of isolates, sample populations, laboratory test methods and quality assurance programs used to generate data. These methods were unaltered in 2002. Most isolates were collected from symptomatic sexually transmitted disease clinic patients. As a guide to the interpretation of the following data, a WHO expert committee has recommended that treatment regimens be altered once resistance to a particular antibiotic reaches five per cent.[6]

Results

About 11,500 gonococcal isolates were examined for susceptibility to one or more antibiotics in 17 participating countries (listed in the acknowledgements) in 2002.

Quinolones

Quinolone resistant gonococci (QRNG) have been widely distributed in the WHO WPR for many years and use of this group of agents is no longer recommended for treatment of gonorrhoea in many countries. Travellers who acquire infection in the WPR, but present in other regions, would require treatment with agents other than quinolones.

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QRNG were detected in 13 of the 14 countries that examined a total of about 10,700 isolates for quinolone resistance in 2002. The exception was Papua New Guinea where use of these quinolones is limited. Data from these 14 WPR countries are shown in Table 1 and QRNG are divided into 'less susceptible' and 'resistant' categories on the basis of susceptibility determinations. Rates of QRNG, where detected, ranged from five per cent (New Caledonia) to 95 per cent (Korea). Very high proportions of QRNG were detected in Brunei, China, Hong Kong, Japan, Korea, Laos, Malaysia, the Philippines, Singapore and Vietnam. Most of the QRNG in the majority of countries displayed high level resistance.

Cephalosporins

A small number of isolates with altered susceptibility to third generation cephalosporins was noted in both the 2000 and 2001 reports. In 2002 these were again detected in small numbers in Australia, Brunei, China, Korea and Malaysia. Non-GASP data from Japan[7] indicate the continuing presence of these resistant strains in that country.

Spectinomycin

A small number of spectinomycin resistant strains were found in China and Vietnam. Only very occasional strains resistant to this injectable antibiotic have been found in recent WPR surveys.

Penicillins

Resistance to the penicillins has been at high levels for many years and use of this group of antibiotics has been largely discontinued except for a few areas such as rural Australia, where monitoring demonstrates a continued susceptibility to the agent. For most centres in the region, resistance by both chromosomal (CMRNG) and plasmid-mediated mechanisms (penicillinase producing *N. gonorrhoeae* – PPNG) remained widespread. Table 2 provides details of CMRNG, PPNG and/or total penicillin resistance in 17 WPR countries.

Very high rates of penicillin resistance (CMRNG +/– PPNG) were recorded in Laos (100%), the Philippines (92%), Korea (81%), China (85%), Papua New Guinea (82%) and Hong Kong (70%). Malaysia and Brunei (60%), Singapore (55%), Tonga (25%) and Vietnam (30%) also had high rates of penicillin resistance. In past years, low rates of penicillin resistance were observed in Pacific Island states, and continued to be low in New Caledonia (3%) and Fiji (1.8%). However, PPNG were prominent in Vanuatu (20%). Other participants submitting data in 2002 (Australia, Japan and New Zealand) had rates of penicillin resistance between 9 and 30 per cent.

Table 1. Quinolone resistance in *Neisseria gonorrhoeae* isolated in 14 countries in the WHO Western Pacific Region in 2002

	Tested	Less susceptible		Resistant		All QRNG	
Country	n	n	%	n	%	n	%
Australia	3,861	77	2.0	312	8.1	389	10.1
Brunei	33	1	3.0	20	61.0	21	63.6
China	1,249	–	–	–	–	1,115	92.5
Hong Kong SAR	3,488	165	4.7	3,205	89.1	3,272	93.8
Japan	211	24	11.4	155	73.4	179	84.8
Korea	210	68	32.4	133	63.3	201	95.7
Laos	58	1	1.7	43	74.1	44	75.9
Malaysia	10	1	10.0	4	40.0	5	50.0
New Caledonia	62	3	4.8	0	–	3	4.8
New Zealand	718	48	6.7	61	8.5	109	15.2
Papua New Guinea	279	0	–	0	–	0	0.0
Philippines	99	2	2.0	57	57.5	59	59.6
Singapore	200	9	4.5	93	46.5	102	51.0
Vietnam	213	49	23.2	97	46.0	146	79.2

QRNG Quinolone-resistant *Neisseria gonorrhoeae*

Table 2. Penicillin sensitivity in *Neisseria gonorrhoeae* isolated in 17 countries in the WHO Western Pacific Region in 2002

Country	Tested	PPNG		CMRNG		All penicillin resistant	
	n	n	%	n	%	n	%
Australia	3,861	274	7.1	421	10.9	695	18.0
Brunei	42	25	59.5	0	0.0	42	59.5
China	1,250	434	34.7	637	50.9	1,071	85.6
Fiji	672	9	1.3	3	0.5	12	1.8
Hong Kong SAR	3,488	768	22.0	1,651	47.3	2,419	69.4
Japan	211	2	1.0	61	29.0	63	30.0
Korea	210	48	22.8	123	58.6	171	81.4
Laos	20	18	90.0	2	10.0	20	100.0
Malaysia	10	3	30.0	3	30.0	6	60.0
New Caledonia	62	2	3.2	0	–	2	3.2
New Zealand	718	23	3.2	43	6.0	66	9.2
Papua New Guinea	279	111	40.0	114	41.0	245	82.0
Philippines	99	88	88.8	3	3.0	91	91.9
Singapore	200	103	51.5	6	3.0	109	54.5
Tonga	41	–	–	–	–	10	25.0
Vanuatu	55	11	20.0	NT	NT	11	20.0
Vietnam	213	61	28.6	3	1.4	64	30.0

PPNG Penicillinase producing *Neisseria gonorrhoeae.*

CMRNG Chromosomally mediated resistance in *Neisseria gonorrhoeae.*

NT Not tested.

Tetracyclines

Although tetracyclines are not a recommended treatment for gonorrhoea, these agents are widely used and readily available in the WPR. One particular type of plasmid-mediated resistance gives rise to high level tetracycline resistance (TRNG). Ten thousand five hundred and seventeen gonococci were examined for high level tetracycline resistance in 15 WPR countries in 2002 (Table 3). High rates of TRNG continue to be reported from Brunei, Laos, Malaysia, Singapore, China, Hong Kong, Vietnam and the Philippines, all with rates between 26 and 97 per cent. In other countries rates of TRNG ranged between 1 and 11 per cent of strains examined.

Table 3. High level tetracycline resistance in *Neisseria gonorrhoeae* isolated in 15 countries in the WHO Western Pacific Region in 2002

Country	Tested	TRNG	TRNG
	n	n	%
Australia	3,861	442	11.4
Brunei	30	28	93.0
China	1,250	388	31.0
Hong Kong SAR	3,488	996	28.6
Japan	211	2	1.0
Korea	210	9	4.3
Lao PDR	58	56	96.6
Malaysia	10	7	70.0
New Caledonia	62	1	1.6
New Zealand	718	45	6.3
Papua New Guinea	279	6	2.1
Philippines	99	28	28.0
Singapore	200	128	64.0
Tonga	41	1	2.4
Vietnam	213	56	26.3

TRNG Tetracycline resistant *Neisseria gonorrhoeae.*

Discussion

The 2002 WHO WPR GASP surveillance data confirmed trends in resistance patterns in gonococci seen in recent years. This surveillance is difficult to undertake, and sample sizes in some settings are restricted. Despite these limitations, there are sufficient data to determine that resistance to cheap oral antibiotic agents such as the penicillins and quinolones in most countries is so high as to preclude their use. Any contemplated use of these agents for patients infected in these countries would be ill advised unless laboratory confirmation of susceptibility is available.

The choice of alternative treatment regimens is limited by the cost of suitable alternative antibiotics. The continued occurrence of gonococci with altered susceptibility to third generation cephalosporins associated with documented treatment failure remains a matter of considerable concern. However this problem with oral third generation cephalosporins has not as yet extended to injectable agents such as ceftriaxone. Spectinomycin resistance remains sporadic and has been observed in parts of the region in the past. The current situation is one for great concern, as the resistance occurred in a pathogen causing a high incidence of disease and with a documented propensity to widely disseminate resistant subtypes.

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Report of the Australian Rotavirus Surveillance Program 2002–03

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National Rotavirus Reference Centre

Abstract

The National Rotavirus Reference Centre, together with collaborating laboratories Australia-wide, has conducted rotavirus surveillance since June 1999. This report describes the serotypes of rotavirus strains responsible for the hospitalisation of children with acute gastroenteritis during the period 1 July 2002 to 30 June 2003. We examined 573 faecal samples using monoclonal antibody immunoassays, reverse transcription-polymerase chain reaction, and polyacrylamide gel analysis. For the second consecutive year, serotype G9 strains were the most prevalent type nationally (74.7%) and were found in all seven contributing centres. Serotype G1 strains were the second most prevalent type (11.3%), identified in four of the centres. These findings have implications for vaccine development strategies which have targeted protection of disease due to serotypes G1–G4. *Commun Dis Intell* 2003;27:492–495.

Keywords: communicable disease, rotavirus, surveillance

Introduction

Rotaviruses are the single most important cause of severe gastroenteritis in young children worldwide. While there are few deaths in developed countries, there is considerable morbidity, with 10,000 Australian children hospitalised each year.[1] There is wide acceptance of the need for a vaccine to prevent rotavirus disease in children under five years of age throughout the world.

The previous rotavirus surveillance report from the National Rotavirus Surveillance Program, covering the period July 2001 to June 2002, highlighted the emergence of serotype G9 as the dominant serotype nationally.[2] For the first time since national surveillance began in 1993, serotype G1 was not the dominant type in Australia.

The National Rotavirus Reference Centre in Melbourne, together with collaborating laboratories in Western Australia and the Northern Territory, has undertaken the surveillance and characterisation of rotavirus strains causing annual epidemics of severe diarrhoea in young children. In this report we describe the results of the Australian Rotavirus Surveillance Program for the period 1 July 2002 to 30 June 2003.

Methods

Collaborating laboratories undertook rotavirus detection by enzyme immunoassay (EIA) or latex agglutination. Rotavirus positive specimens were collected, stored frozen and forwarded to Melbourne, together with relevant age and sex details. Specimens were then tested using an in-house monoclonal antibody (MAb) based serotyping EIA. The EIA employed a panel of MAbs specific for the major glycoprotein VP7 of the outer capsid of the five major group A human rotavirus serotypes (G1, G2, G3, G4 and G9). Strains unable to be assigned a serotype were genotyped by reverse transcription/polymerase chain reaction (RT/PCR) using serotype specific oligonucleotide primers.[3] Polyacrylamide gel electrophoresis was used to classify rotavirus strains genetically into electropherotypes, and to confirm the sharing of the same electropherotype between collaborating centres.

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Results

Number of isolates

A total of 573 specimens were received from Melbourne and the collaborating centres in Western Australia and the Northern Territory. Specimens containing insufficient specimen for testing or specimens that were not confirmed to be positive for rotavirus were omitted. A total of 487 rotavirus positive specimens over a 12 month period from 1 July 2002 to 30 June 2003 were analysed.

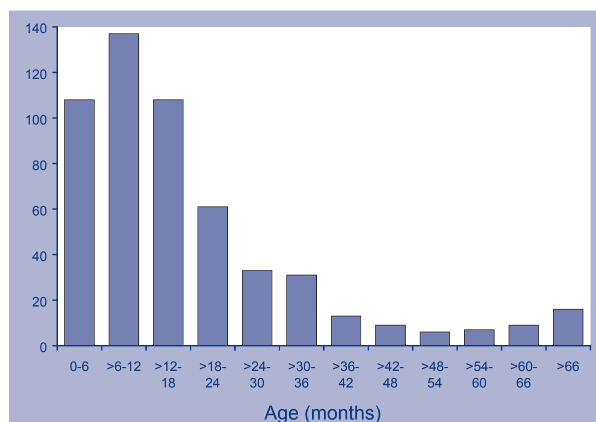
Age distribution

The age distribution of the children with acute gastro-enteritis was typical of rotavirus infection (Figure 1). In the reporting period, 33 per cent of cases were from infants 12 months of age or less, 39 per cent were from patients 13–24 months of age, and 15 per cent were from patients 25–36 months of age. Overall, 87 per cent of samples were from children three years or less, and 94 per cent were from children five years or less. The male to female ratio was 1.1:1.

Serotype distribution

Rotavirus serotypes identified in Australia from 1 July 2002 to 30 June 2003 are shown in the Table. Serotype G9 was the most common, representing 74.7 per cent of all specimens and 50 per cent or more of serotypes identified in all seven centres. G1 was the second most common serotype, and represented 11.3 per cent of specimens overall, but was identified in only four centres (Melbourne, Perth, Darwin-Western Pathology and Western Australia's PathCentre) (Table). Serotypes G2, G3 and G4 each represented less than two per cent of all specimens. Serotype G3 was identified in three centres, Melbourne, Perth and WA PathCentre, while G2 and G4 were identified only in Perth (Table).

Figure 1. Age distribution of children with rotaviral infection, Australia, 1 June 2002 to 30 June 2003



During the reporting period, 3.4 per cent of the rotavirus samples analysed contained multiple sero-types. The presence of mixed infections provides the opportunity for rotavirus to undergo reassortment, potentially resulting in new strains. In 7.2 per cent of the samples a serotype was unable to be identified. These could represent samples with low virus numbers which are below the detectable limits of our assays. Alternatively, these could represent unusual serotypes not identified using standard methods. Future studies will include further characterisation of the genes encoding the outer capsid proteins of these strains.

Table. Rotavirus G serotypes in Australia, 1 July 2002 to 30 June 2003

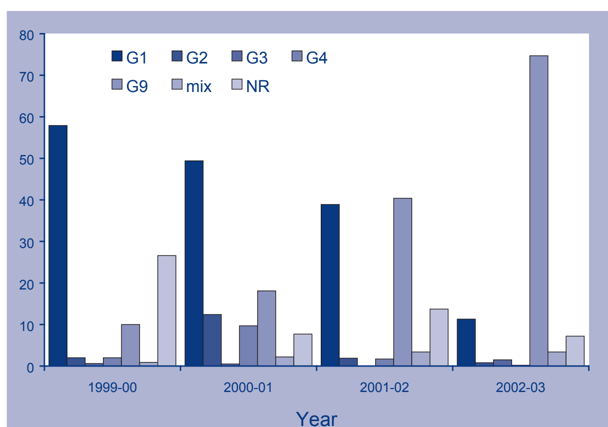
Centre	Total number	Serotype percentage						
		G1	G2	G3	G4	G9	Mixed serotypes	No result*
Melbourne	109	16.5	0.0	1.83	0.0	65.1	5.5	11.0
Perth	147	16.3	2.7	2.0	0.7	71.4	4.8	2.0
WA PathCentre	104	11.5	0.0	1.9	0.0	78.9	1.0	6.7
Darwin	39	0.0	0.0	0.0	0.0	87.2	10.3	2.5
Darwin-Western Pathology	6	16.7	0.0	0.0	0.0	50.0	0.0	33.3
Alice Springs	70	0.0	0.0	0.0	0.0	87.1	4.3	8.6
Gove	12	0.0	0.0	0.0	0.0	66.7	0.0	33.3
Total	487	11.3	0.8	1.5	0.2	74.7	3.4	7.2

* No result—unable to be serotyped with monoclonal antibodies or genotyped by RT/PCR.

Discussion

National rotavirus surveillance from 1 July 2002 to 30 June 2003 showed that serotype G9 continued to be the nationally dominant serotype comprising 74.7 per cent of all strains. Serotype G9 has steadily increased since it was first identified during Australia-wide surveillance in 1997.[5] G9 became the second most prevalent serotype nationally during the 1999–00 and 2000–01 surveys, representing 10 per cent and 18.1 per cent respectively, of specimens collected in those years (Figure 2).[6,7] G9 became the dominant strain nationally in 2001–02, comprising 40 per cent of the strains, but represented less than 14 per cent of strains in Melbourne and Perth.[2] The dominance of serotype G9 nationally in 2002–03 may be due to a large outbreak of acute gastroenteritis caused by rotavirus G9 in Central Australia during 2001.[4]

Figure 2. Rotavirus serotypes identified in Australia, June 1999 to June 2003



The four major rotavirus serotypes (G1, G2, G3 and G4) represented over 90 per cent of strains in epidemiological studies conducted throughout the world since the early 1980s.[8] Serotype G9 is an emerging serotype, and since 1996 has been recognised as a frequent cause of acute diarrhoea in children. It is now recognised as the fifth most common serotype worldwide. Its apparent re-emergence is illustrated by serotyping studies from Japan. Serotype G9 strains were first identified in Japan in 1985–1986, but were absent for nine years until identified in two specimens in 1994–1995.[9] The re-emergence of G9 in Japan has continued with an increased prevalence in 1996–2000.[10]The high prevalence of G9 in Australia is remarkable when compared with prevalence of this serotype in other countries reported to date. Serotype G9 has been identified on all continents and in more than 17 countries, with prevalence rates of 1–8 per cent.[11,12,13] Few countries have reported prevalence rates comparable to Australia. Epidemiological studies from Bangladesh and Nigeria found 38 per cent and 53 per cent of rotavirus strains were type G9 during 1998 and 1997, respectively.[14,15]

The decline in the prevalence of serotype G1 in Australia has been dramatic. G1 was the dominant serotype from 1993 to 1996[16] and in two surveys conducted Australia-wide during 1999–00 and 2000–01, represented 58 per cent and 49.5 per cent of specimens.[6,7] However, during the next two years (2001–02 and 2002–03), G1 declined to 38.9 per cent and 11.3 per cent respectively.[2] The decline in the prevalence of G1 strains around Australia can be attributed to the increase in the prevalence of G9 strains in Central Australia.

The increase in prevalence of serotype G9 has not been associated with changes in the age distribution of children infected with rotavirus. The majority of children (87%) infected with rotavirus were under three years of age.

These results together with those of previous years highlight the continuing change in the prevalence and emergence of new rotaviral serotypes. Multi-centre surveillance of rotavirus is important to continue to monitor strains and essential to inform the development of new rotavirus vaccines.

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Surveillance of viral pathogens in Australia

For many years, a sentinel laboratory system, the Laboratory Virology and Serology Reporting Scheme (LabVISE) has been collecting data on viral pathogens of public health importance in Australia. This report is one in a series of articles focussing on the epidemiology and public health aspects of viruses and viral groups under surveillance by LabVISE, which are of current public health interest.

Rotavirus

Charlie Blumer,[1] Paul Roche,[1] Carl Kirkwood,[2] Ruth Bishop,[2] Graeme Barnes[2]

Introduction

Since the discovery of rotaviruses in 1973, at the Royal Children's Hospital in Melbourne,[1] they have been recognised as a leading cause of severe and acute diarrhoeal illness in young children throughout the world. The global burden of rotaviral disease was recently estimated as 111 million cases requiring home care, 25 million cases requiring medical attention, one million requiring hospitalisation and 440,000 deaths in children aged less than five years, annually.[2] In the United States of America (USA), rotaviral infections are responsible for an estimated 500,000 physician visits, 50,000 hospitalisations and 20 to 40 deaths per year.[3] In Australia, it has been estimated that rotavirus infection is the cause of diarrhoea in 10,000 of the nearly 20,000 children admitted to hospital each year with severe diarrhoea.[4]

In this report, surveillance data for rotavirus infections in Australia are analysed from four sources: the Laboratory Virology and Serology Reporting Scheme (LabVISE), the National Hospital and Morbidity database, the Northern Territory notifiable diseases data and the Australian Rotavirus Surveillance Programme. The importance of surveillance data for future rotavirus vaccine development is discussed.

Molecular biology and nomenclature

Rotaviruses comprise a genus within the family Reoviridae that includes viruses that inhabit both the respiratory and enteric systems of birds and mammals. Initially, members of this family were considered 'orphan viruses', as they were not associated with any disease in humans, and it was this combination of properties that led to their being named reoviruses (**R**esp, **e**nteric, **o**rphan, – viridae).

The rotavirus is named for its 'spoked wheel' appearance in electron micrographs of negatively stained faecal extracts. The double-stranded RNA can be separated into 11 segments by gel electrophoresis. Six of the genes code for structural proteins (VP1–4, VP6, VP7) and five for non-structural proteins. Six groups of rotavirus (A, B, C, D, E, F) are recognised based on differences in serology and genetic patterns. Group A is common in humans, Group B is uncommon in infants, but has caused large epidemics in adults in China while Group C appears to be uncommon in humans (Chin, 2000).[5] All six groups of rotavirus occur in animals and birds.

Variations in the two proteins making up the outer layer, viral protein 4 (VP–4, or P (protease-sensitive) and viral protein 7 (VP–7, or G (glyco-protein) are used to further classify rotaviruses. In a similar manner to the classification of influenza virus strains, serotypes of Group A rotaviruses are defined by the characteristics of the two outer proteins (e.g. G1, G2, or P1, P2) and the two in combination are used to identify particular serotypes (e.g. G1,[P1A]).[6]

The mode of transmission of rotaviruses is predominantly by a faecal oral route, although airborne transmission has been postulated, particularly in nurseries, child day care centres, and hospital wards. Infected infants shed virus in large numbers (>10^{12} particles/ml faeces) for four to seven days after the onset of disease. Chronic shedding of virus by healthy adults or partially immune infants may provide a reservoir of infection. Public water supplies may also be a source of infection.[7] Within hospital nurseries rotavirus may be endemic with a single strain circulating for years[8,9] and nosocomial infections are common. A recent study of an outbreak of a novel rotaviral strain in a neonatal hospital unit was shown to be associated with persistence of the virus on hard surfaces and with lapses in infection control practices.[10]

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Pathology and clinical outcomes

Following ingestion, the virus infects mature absorptive epithelial cells lining the small intestine where they replicate, followed by lysis of infected cells. There is a depression of lactase enzyme in the damaged intestinal mucosa, which increases gut osmolarity and results in watery diarrhoea and lactose malabsorption that may persist for 10 to 14 days.[7] Fever, vomiting and watery diarrhoea are the major clinical signs of rotavirus infection. In a neonatal intensive care setting, rotaviral infection may manifest as more frequent production of watery or bloody stools.[11] Re-infections that occur in childhood and adult life are common but are usually associated with mild enteric symptoms or are asymptomatic.

The key treatment of rotaviral diarrhoea is replacement of fluids and electrolytes. The promotion in developing countries of oral fluid replacement therapy in children with diarrhoea has been critical in saving many lives. The oral rehydration mixtures consist of pre-weighed glucose, sodium, potassium, chloride and base in appropriate proportions to replace electrolyte losses, increase the uptake of water and provide calories.[7]

Epidemiology

Globally nearly all children experience a primary rotavirus infection by the age of five years. One in five will have visited a clinic, one in 65 will have been hospitalised and one in 293 will die of rotaviral infection. Eighty-two per cent of deaths due to rotaviral infection occur in developing countries.[2]

Infection in children less than three months is uncommon, possibly due to the protective effect of maternal antibodies,[12] although breast-feeding has not been clearly shown to reduce the incidence or severity of rotaviral disease. However, a recent study in Italy noted an attack rate of rotaviral infection of 10.6 per cent in hospitalised breast-fed infants aged 1 to 18 months compared with an attack rate of 32.4 per cent in non-breast-fed infants.[13] Susceptibility to rotaviral infection in children may increase as the gut epithelium matures.[7]

Prospective studies in Australia, Mexico, Africa and elsewhere have shown that primary infection with rotavirus gives little or transient protection against re-infection, but does protect against clinically severe diarrhoea after reinfection.[9,14,15] Reinfection with rotavirus occurs into adult life, but infections are usually asymptomatic or cause mild disease presumably because of the immunity gained in early childhood. Epidemics associated with rotavirus infection have occurred in nursing homes for the elderly.[16] Intestinal secretory IgA antibodies directed to the VP4 and VP7 proteins of the viral outer coat play a critical role in immunity to rotaviral infection by preventing viral attachment and entry into the intestinal epithelium.[17] A recent review has concluded that serum anti-rotaviral antibodies are a correlate of protective immunity when present at sufficient levels.[18]

There is a strong association of rotaviral infections with the winter seasons in temperate climates, while in tropical climates cases occur year round. The reasons for the seasonal epidemics in winter are uncertain, but may reflect increased opportunity for transmission within families and longer survival of the virus in the environment during colder months.[19]

Prevention of rotaviral disease

Passive immunisation and probiotics

Treatment of acute rotaviral diarrhoea by oral administration of antibodies to rotavirus has been investigated. Despite numerous studies on the use of oral administration of rotaviral immunoglobulin preparations, a recent Cochrane review was unable to identify randomised control trials to assess the efficacy of this approach in low birth weight babies.[20] The passive administration of bovine milk antibodies containing high titres of neutralising antibodies to human rotaviral strains was shown to have significantly reduced the duration of excretion of rotavirus from infected children.[21,22] In immunodeficient children passive administration of antibodies may also reduce chronic rotaviral diarrhoea.[7]

An alternative approach has been to administer the non-pathogenic *Lactobacillus* GG bacteria to modulate the diarrhoeal disease caused by rotaviral infection. A randomised trial of *Lactobacillus* GG therapy has shown reductions in the frequency and duration of rotavirus diarrhoea in children with acute respiratory infection being treated with antibiotics.[23] However, *Lactobacillus* GG therapy in hospitalised infants failed to prevent nosocomial rotaviral infection.[24]

Vaccines

In view of the high disease burden and high mortality rates in the developing world from rotaviral infection, a rotavirus vaccine is the only control measure likely to have a significant impact on the incidence of the disease. The World Health Organization has given a high priority to the development of a safe and effective rotaviral vaccine.[25]

The first licensed vaccine against rotavirus, a live rhesus-human reassortant rotavirus tetravalent vaccine (RRV-TV, known also as RotaShield), was released for use in the United States of America in 1998. The RRV-TV was an oral vaccine designed to provide protection against the four major human serotypes G1, G2, G3 and G4. Extensive trails had demonstrated a vaccine efficacy of up to 91 per cent in preventing hospitalisation for severe diarrhoeal disease and a three dose regimen (at 2, 4, and 6 months) was recommended for all healthy infants in the USA.

A retrospective review of RRV-TV vaccine effectiveness in the prevention of hospitalisations in infants under three years for rotaviral related illness, demonstrated a reduction in attack rates from 0.34 per 100 child years in unimmunised infants, to 0.2 per 100 child years in partially, or to zero per 100 child years in fully, RRV-TV immunised children. This implied a protective effectiveness of 70 per cent and that one episode of rotaviral diarrhoea requiring hospitalisation was prevented for every 64 infants fully vaccinated.[26]

Nine months after the release of the vaccine, when at least one million doses had been administered, the vaccine program was suspended. Fifteen reports of intussusception (a bowel obstruction resulting from the infolding of one segment of the intestine within another) had been reported, with 13 occurring within one week of receiving the first dose of RRV-TV.[27] Although the risk of intussusception due to RRV TV vaccination was low (about 1 in 11,000), trials of new vaccines will need to exclude this rare but serious complication. There will be a need for post-licensure surveillance of intussusception.

In addition, two further human-animal rotavirus based reassortant vaccines are undergoing clinical trials. Merck have completed extensive trials of pentavalent bovine (WC3)-based vaccine incorporating serotypes G1–G4 and P8 specificity.[28] The National Institutes of Health, USA, have developed a bovine (UK) based reassortant vaccine, incorporating G1–G4 antigens and capable of including G5, G8, G9 and G10 antigens as required.[29,30]

Several live attenuated rotavirus vaccine candidates are currently in human trials around the world. These include the attenuated human monovalent live vaccine 89–12 (Rotarix) which is currently undergoing phase III trials in several countries, including the USA and South America. The vaccine efficacy in a recent follow up study in the USA has been estimated at 76 per cent against rotaviral gastroenteritis, 83 per cent against severe rotaviral disease and 100 per cent against rotaviral disease requiring medical intervention.[31]

A naturally attenuated human neonatal rotaviral strain RV3 has been tested recently in a limited phase II trial in Australia. Three oral doses at 3, 5 and 7 months induced immune responses in 46 per cent of recipients and partial protection (54%) against rotaviral disease in a subsequent epidemic.[32]

An estimate of the cost effectiveness of a rotaviral vaccine in Australia was undertaken in 1999. The analysis was based on the RRV–TV vaccine in a universal program and concluded that such a program would be cost-neutral. The authors acknowledged that uncertainties about vaccine and delivery costs made any cost effectiveness estimates difficult.[33]

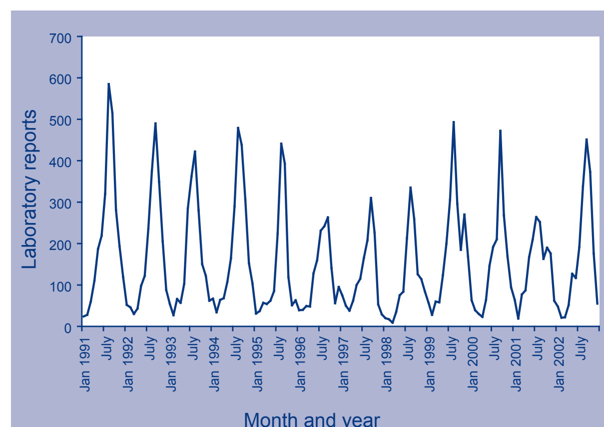
Surveillance of rotavirus in Australia

Laboratory Virology and Serology Reporting Scheme

Laboratories perform a central role in the definitive diagnosis of rotaviral disease. Since 1991, between 13 and 26 sentinel laboratories have voluntarily notified the Department of Health and Ageing each month of isolations of viruses of public health importance. From 1991 to 2002, between 1,372 and 2,642 reports of rotavirus were received annually, with the largest numbers of reports from New South Wales (32%).

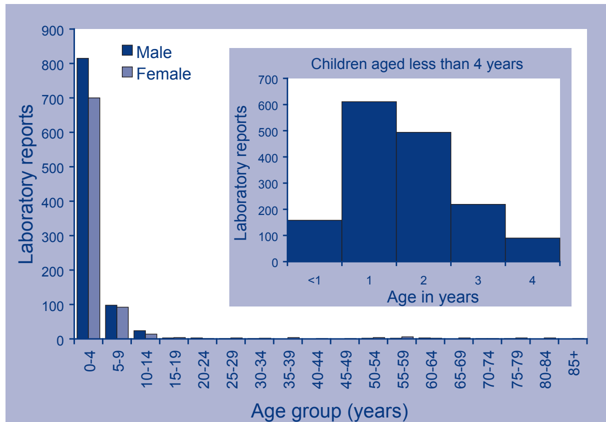
The number of reports received by the Laboratory Virology and Serology Reporting Scheme (LabVISE) are a product of the number and location of reporting laboratories; hence in the 12 year period, 1991 to 2002, only 77 reports were received from the Northern Territory (reported by interstate laboratory as there is no Northern Territory laboratory reporting to LabVISE). LabVISE data on rotavirus show that in Australia there is an annual winter epidemic, with a peak in reports every August or September (Figure 1).

Figure 1. Laboratory reports to LabVISE of rotavirus infection, Australia, 1991 to 2002, by month of specimen collection



The age and sex of rotavirus cases reported to LabVISE in 2002 are shown in Figure 2. The male to female ratio was 1.1:1. The largest number of reports was for children aged 0 to 4 years (77% of all reports), among whom the highest rates were in children aged one year (31% of all reports).

Figure 2. Laboratory reports to LabVISE of rotavirus, 2002, by age and sex



National Hospital Morbidity Database

The National Hospital Morbidity Database (NHMD) is compiled by the Australian Institute of Health and Welfare from data supplied by the state and territory health authorities. The NHMD is a collection of summary records for patients admitted to public and private hospitals in Australia in the years 1993–94 to 2000–01. The total number of records for 2000–01 was 6.14 million. Almost all hospitals in Australia are included: public acute, public psychiatric hospitals, private acute and psychiatric hospitals, and private freestanding day hospital facilities.

Data from the NHMD for rotaviral hospitalisations for rotaviral enteritis are shown in the Table as 'separations.' This term is used to refer to the episode of care, which can be a total hospital stay (from admission to discharge, transfer or death), or a portion of a hospital stay beginning or ending in a change of type of care (for example, from acute to rehabilitation). 'Separation' also means the process by which an admitted patient completes an episode of care by being discharged, dying, transferring to another hospital or changing type of care.

Data fromt he two most recent years are shown in the Table. Rotaviral enteritis was recognised as the primary diagnosis in between 3,000 and 4,300 separations in Australian hospitals. These represented between a quarter and a third of all separations for viral gastroenteritis. Of hospital separations for rotaviral enteritis, 94 per cent were in children aged less than five years. Patients admitted for rotaviral enteritis were hospitalised for two to three days on average and accounted for between eight and 11,000 hospital bed days.

Rotavirus surveillance in the Northern Territory

In the Northern Territory, infection with rotavirus has been a notifiable disease since 1994. The number of notifications and the rates of rotaviral disease in Indigenous and non-Indigenous populations are shown in Figure 3.

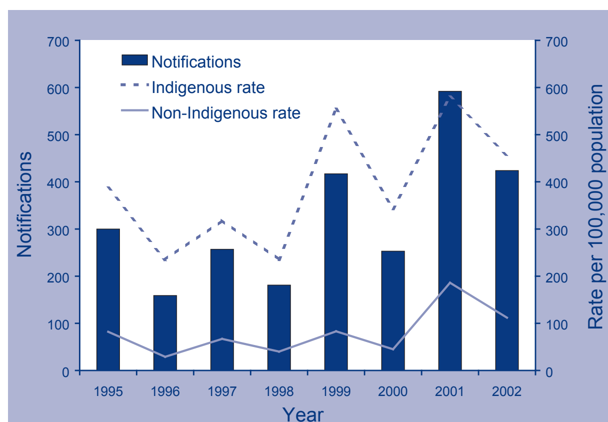
Table. Separation, patient day and average length of stay for rotaviral enteritis, Australia, 1998–99 and 1999–00*

Separations	1998–1999	1999–2000
AO8 Viral and other specified intestinal infections	13,026	14,110
AO8.0: Rotaviral enteritis (percentage of separations under AO8)	3,043 (23%)	4,306 (31%)
Separations in < 1 year olds	809	1,018
Separations in 1 to 4 year olds	2,055	3,032
Percentage of rotaviral separations in infants aged less than 5 years	94.5%	94%
Total patient days for rotaviral enteritis (all ages)	8,194	11,029
Average length of stay for rotaviral enteritis (all ages)	2.7	2.6

* Source Australian Institute of Health and Welfare National Hospital Morbidity Database.

The magnitude of the annual rotaviral epidemic varies year by year with biennial peaks.[34] The Northern Territory contains both tropical regions around Darwin and hot arid regions around Alice Springs. Neither area experiences consistent seasonal peaks in rotaviral activity.[35] The rate of rotaviral enteritis in Indigenous Australians in the Northern Territory was between three and eight times the rate in the non-Indigenous population (Figure 3).

Figure 3. Notifications of rotaviral enteritis and rates per 100,000 population in Indigenous and non-Indigenous populations, Northern Territory, 1995 to 2002



Source: Data supplied by Dr Peter Markey, Centre for Disease Control, Darwin.

These surveillance data were also used to analyse an outbreak of G9-associated rotavirus in Central Australia, in which over 500 cases of childhood diarrhoea were notified.[34] The impact of this outbreak, unprecedented in Australia, placed an extreme demand on the Central Australian health services.[36]

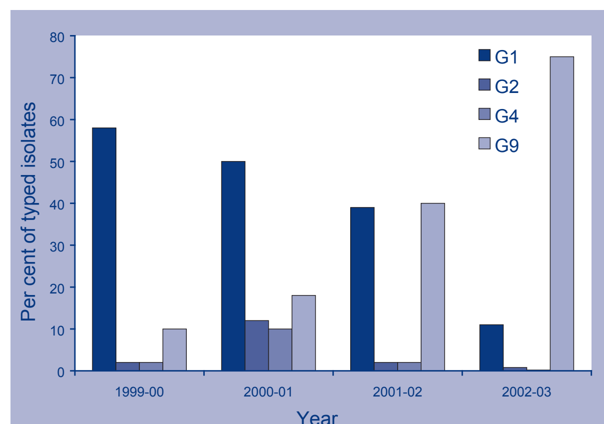
Australian Rotavirus Surveillance Program

A 13-year survey (1980–1993) of children hospitalised with acute gastroenteritis at the Royal Children's Hospital in Melbourne identified Group A rotavirus in 40 per cent of children, and 55 per cent of children aged between 12 and 23 months. The study also found that rotavirus was responsible for 19 per cent of acute gastroenteritis in children aged under six months. This implies that any vaccine must be able to be given early in life, or a significant number of events in infants will not be prevented.[37] Rotavirus appeared to be largely responsible for the seasonal increase in cases during the winter epidemics.

This work prompted a national survey in eight Australian cities of rotavirus isolates from children aged less than five years admitted to hospital for acute gastroenteritis between 1993 and 1996. Admissions for gastroenteritis averaged just under 20,000 per year nationwide, and 50 per cent could be attributed to rotavirus infection. This study showed a predominance of the G1 serotype in all centres. Subsequent work demonstrated outbreaks of rotaviral enteritis in the Northern Territory in 1993–04 due to unusual rotaviral strains, including a G8 strain with some 'bovine' characteristics.[38]

From June 1999, a national rotavirus surveillance program was initiated to undertake the characterisation of rotavirus strains causing severe diarrhoea in Australian children. The program was designed to monitor changes in the prevalence of rotaviral serotypes prior to the anticipated introduction of a rotaviral vaccine into Australia. The Australian Rotavirus Surveillance Program, funded by the Commonwealth Government has documented rotaviral serotypes circulating in Australia over the past four years (Figure 4).

Figure 4. Changes in prevalence of serotypes of subgroup A rotavirus, Australia, 1999 to 2002



The major finding is the appearance and rapid increase in the prevalence of the G9 serotype.[39] National rotavirus surveillance has shown serotype G9 to be the dominant type for the past two reporting periods (2001–02 to 2002–03). In Darwin in 2001–02 the G9 serotype made up 95 per cent of serotypes and was the serotype responsible for a major outbreak in 2001 in the Northern Territory (described above), while in Perth and Melbourne the G1 serotype was still the most prevalent serotype (72 and 48% of reports, respectively). Since then, in 2002–03, G9 has become the most common serotype throughout Australia, and was dominant in all seven contributing centres.

Internationally, the emergence and spread of the G9 serotype has been documented in several countries since 1995[39,40] and should now be considered the fifth globally important serotype along, with serotypes G1 to G4.[6] Continued surveillance of the circulating Group A serotypes is essential preparation for the introduction of new rotaviral vaccines.

The future of rotavirus surveillance in Australia

The current surveillance systems capturing data on rotaviral infection in Australia each contribute complementary data essential for public health action. LabVISE provides basic information on the annual epidemics of rotaviral disease, largely from hospitalised children in south-eastern Australia. LabVISE data on rotaviral infections in the Northern Territory is inadequate. The LabVISE system is not sensitive enough to detect year to year variations in the size and extent of the annual epidemics.

The inclusion of rotavirus infection in the Northern Territory notifiable diseases surveillance system however, is able to identify the extent of disease activity in the communities most affected by the disease. These data are useful in describing the variation in the size of annual epidemics, outbreaks of disease and the burden of rotaviral disease in Indigenous communities.

The National Hospital Morbidity Database provides some estimates on the hospitalisation costs associated with rotaviral enteritis on a national basis. Delays in these data limit their usefulness for surveillance purposes. Hospitalisation data is important for the analysis of the cost effectiveness of any future rotaviral vaccine program.

The Australian Rotavirus Surveillance Program has documented rapid and significant changes in the relative prevalence of different serotypes of Group A rotaviruses and the variation in the geographical distribution of serotypes across the country. It is important to maintain a central laboratory capable of detection and characterisation of new rotavirus strains as they emerge. Future rotavirus vaccines may require updating if they do not cross-protect against commonly found strains. These data are essential to policy makers in future decisions about the introduction of a rotaviral vaccine to Australia.

In combination, these four surveillance systems provide important information about rotaviral disease in Australia. Should a rotaviral vaccine be introduced, either as a universal childhood vaccine or targeted to Indigenous children, none of the present surveillance systems would be adequate to measure the vaccine effectiveness in disease control. The future of rotaviral surveillance depends largely on whether or not vaccines are introduced. Meanwhile the current systems are important in monitoring the epidemiology of the rotaviral disease, and for informing decisions about the introduction of any new vaccine in Australia.

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Erratum

Communicable Diseases Surveillance - Additional reports - Childhood immunisation coverage

The calculation for the 'Change in fully immunised since last quarter' for the 6 years of age cohort has been incorrect since the first publication of this table in November 2002. Table 11 for the birth cohort 1 January to 31 March 1997, published in the last issue of *Communicable Diseases Intelligence*, is reproduced below. For correct data on previous cohorts please contact the National Centre for Immunisation Research and Surveillance of Vaccine Preventable Diseases by telephone: +61 2 9845 1256, or email: brynleyh@chw.edu.au

Table 11. Proportion of children immunised at 6 years of age, preliminary results by disease and State for the birth cohort 1 January to 31 March 1997; assessment date 30 June 2003

Vaccine	State or territory								
	ACT	NSW	NT	Qld	SA	Tas	Vic	WA	Australia
Total number of children	1,071	22,050	816	12,638	4,681	1,501	15,697	6,588	65,042
Diphtheria, tetanus, pertussis (%)	82.3	84.3	82.0	83.6	83.2	83.7	86.5	82.5	84.4
Poliomyelitis (%)	82.5	84.1	84.9	83.9	83.7	84.1	87.0	82.9	84.6
Measles, mumps, rubella (%)	81.8	82.7	83.0	83.5	82.7	82.6	86.5	82.2	83.7
Fully immunised (%)	80.4	81.2	81.1	82.0	81.3	82.2	85.3	80.6	82.3
Change in fully immunised since last quarter (%)	-1.4	+0.7	-1.1	-0.3	+0.5	-1.6	-0.1	+0.6	+0.1

OzFoodNet: enhancing foodborne disease surveillance across Australia: Quarterly report, 1 July to 30 September 2003

The OzFoodNet Working Group

Introduction

The Australian Government Department of Health and Ageing established the OzFoodNet network in 2000 to collaborate nationally to investigate foodborne disease. OzFoodNet conducts studies on the burden of illness and coordinates national investigations into outbreaks of foodborne disease. This quarterly report is the first in a revised format, which reports on investigations of gastroenteritis outbreaks and clusters of disease potentially related to food occurring around Australia. For information on sporadic cases of foodborne illness, see Communicable Diseases Surveillance, Highlights for 3rd quarter 2003 (pp 552–553).

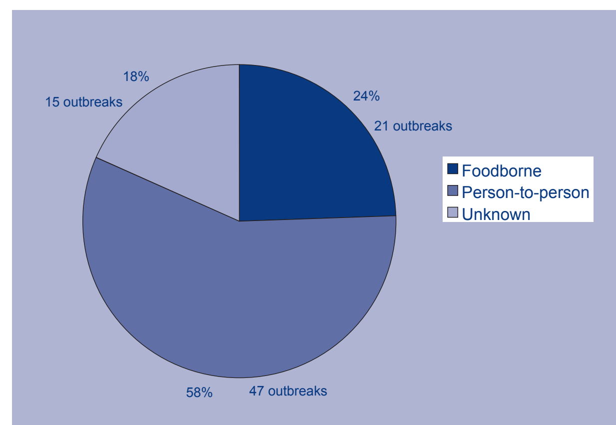
This report summarises the occurrence of foodborne disease outbreaks and cluster investigations between July and September 2003. Data were reported from all Australian jurisdictions and a sentinel site in the Hunter region. The data in this report are provisional and subject to change, as results of outbreak investigations can take months to finalise. We would like to thank state and territory investigators who contributed data to this report.

Foodborne disease outbreaks

During the third quarter of 2003, OzFoodNet sites reported 83 outbreaks of gastrointestinal infections (Figure). Sixty-two of these outbreaks were spread from person-to-person or of unknown transmission, affecting 1,523 persons, hospitalising 69 and causing two fatalities. Outbreaks of gastroenteritis not transmitted by food are often not reported to health agencies or the reports are delayed, meaning that these figures significantly under-represent of the true burden of these infections.

Twenty-one outbreaks were due to foodborne transmission compared to 11 in the previous quarter and eight outbreaks for the same quarter in 2002 (Table). The outbreaks affected 272 persons and hospitalised 13 persons. There was one fatality possibly related to contaminated food in one outbreak. There were six outbreaks of non-typhoidal *Salmonella* infection and three outbreaks each of *Clostridium perfringens* intoxication and norovirus infection. There was one outbreak each of *Staphylcoccus aureus* intoxication and ciguatera poisoning. The remaining seven outbreaks were of unknown aetiology. Four of the outbreaks occurred in association with meals at restaurants or aged care facilities, respectively. Eight of the outbreaks occurred in September.

Figure. Mode of transmission for gastrointestinal outbreaks reported by OzFoodNet sites, July to September 2003



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

Table. Outbreaks of foodborne disease reported by OzFoodNet sites,* July to September 2003

State	Month of outbreak	Setting	Agent responsible	Number exposed	Number affected	Evidence	Responsible vehicles
NSW	July	Home	Unknown	7	1	D	Soccerball ham
	August	Restaurant	Unknown	11	4	D	Unknown
	August	Restaurant	*S.* Typhimurium 170	–	20	A	Tofu, eggplant & prawn dish
	August	Takeaway	*S.* Typhimurium 126 var 4	–	10	M	Pigs ear salad, ducks gizzards
	September	Restaurant	Unknown	–	4	D	Unknown
	September	Residential college	*S.* Typhimurium 135	~100	20	D	
NT	August	Bus	*Staphylococcus aureus*	5	5	D	Rice, beef and black bean sauce
	August	Home	Unknown	21	18	D	Pizza
Qld	July	Restaurant	Norovirus	70	31	A	Trifle
	August	Home	Ciguatera	5	5	D	Barracuda (*Sphyraena* spp.)
	September	Function	Norovirus	100	13	D	Unknown
	September	Picnic	Norovirus	38	15	D	Unknown
SA	September	Community	*S.* Typhimurium 4	–	6	A	Cheesecake
Vic	July	Workplace	Unknown	13	7	D	Unknown
	July	Aged Care	Unknown	120	5		Unknown
	July	Community	*S.* Litchfield/Kinondoni	–	6	M	Suspect cucumbers
	August	Community	*S.* Montevideo	–	3	M	Lebanese tahini
	September	Restaurant	Unknown	–	14	D	Unknown
	September	Aged Care	*C. perfringens*	~600	28	D	Unknown
	September	Aged Care	*C. perfringens*	30	15	D	Unknown
WA	September	Aged Care	*C. perfringens*	190	42	AM	Suspect gravy mixed into vitamised meals

* No outbreaks reported from Australian Capital Territory or Tasmania

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.

Sites conducted five retrospective cohort studies and one case control study to investigate these foodborne outbreaks. The majority (65%) of investigations relied on descriptive epidemiology. One outbreak investigation obtained both epidemiological and microbiological evidence for an association with a food vehicle, in three outbreaks there was microbiological evidence only and in a further three epidemiological evidence only.

The three outbreaks of *C. perfringens* all occurred in aged care facilities. The food responsible was only determined in one of these outbreaks, which was gravy mixed into vitamised meals. Victoria reported another suspected *C. perfringens* outbreak in a nursing home, which was the third suspected foodborne outbreak in the same facility in the previous six months. *C. perfringens* outbreaks are caused by foods that have been poorly prepared and not held at the appropriate temperature. There is an urgent need to improve food safety in aged care facilities as the elderly experience more severe outcomes as a result of foodborne infections.[1]

Victoria investigated three cases of *Salmonella* Montevideo infection. Two cases were associated with the consumption of a commercially available tahini (sesame seed-based) product originating from Lebanon. The investigation triggered a recall of food products in Australia and New Zealand. New Zealand also identified people infected with *S.* Montevideo who had consumed this product. OzFoodNet and New Zealand health agencies prepared an international alert about these products on behalf of the Communicable Diseases Network Australia.[2] Positive tahini sampled before and after the investigation of these human infections triggered product recalls of Lebanese tahini in the United Kingdom and Canada.[3,4]

The Victorian Department of Human Services investigated four cases of *Salmonella* Litchfield and two cases of *Salmonella* Kinondoni. This followed a survey of Vietnamese pork roll ingredients that identified cucumbers contaminated with these serovars. Four out of five Victorian cases reported eating cucumbers. One case acquired their infection overseas and one case did not record eating cucumbers. The association with cucumber consumption between human illness was not confirmed microbiologically from cucumbers that the cases had eaten. No cases were identified in other Australian states or territories.

There was an outbreak of *Salmonella* Typhimurium 4 associated with cheesecake from a bakery in South Australia (reported in this issue).[5] Bakeries have been previously documented as a high risk setting for foodborne illness, particularly for products involving cream and custard.[6] Food safety agencies in Australia should conduct further work to determine where breaches in food safety practices in bakeries are occurring.

New South Wales reported three outbreaks of *Salmonella* Typhimurium during the quarter. New South Wales conducted a case control study into an outbreak of 20 cases of *S.* Typhimurium 170 who had eaten a Yum Cha meal. The case control study identified an association with a fried tofu eggplant and prawn dish. Further investigations revealed that uncooked prawn meat was positive for *S.* Dublin. This imported product was subsequently recalled, even though the exact cause of this outbreak remains unknown. An outbreak of 10 cases of *S.* Typhimurium 126 was associated with takeaway consumption of pigs ear salad and ducks gizzards. An outbreak of 20 cases of *S.* Typhimurium 135 occurred in a residential training college, but no food vehicle was identified.

There were five persons affected by ciguatera poisoning in Queensland following consumption of a whole barracuda. There was 120 kilograms of this barracuda sold to a supermarket and 80 kilograms were recalled. Forty kilograms were sold to the public with no other reported cases. Outbreaks of ciguatera involving fish purchased commercially are now relatively rare and usually result from amateur fishermen catching fish from locally affected reefs. This incident highlights the need for continuing vigilance in monitoring outbreaks of ciguatera.[7]

There was also a small outbreak of *Staphylococcus aureus* intoxication in the Northern Territory following a meal of rice that had not been stored at the correct temperature.

Cluster investigations

During the third quarter of 2003, Australian States and Territories conducted several investigations into clusters of various *Salmonella* serovar infections, including *S.* Havana and *S.* Anatum in Victoria; *S.* Chester, *S.* Oranienberg and *S.* Typhi in Western Australia and *S.* Typhimurium phage type 9 in the Northern Territory.

Queensland reported a cluster of 10 cases of *Salmonella* Typhimurium 170a in South East Queensland during early July. Seventy per cent of cases lived in the Sunshine Coast area. Four of the cases were children aged less than 10 years and six were adults aged 24 years or greater. This phage type is a variant of *S.* Typhimurium 170 and had previously only been detected overseas on rare occasions. The National Enteric Pathogens Surveillance System report that this phage type has not previously been reported in either human or non-human sources in Australia (Joan Powling, personal communication, July 2003). All isolates were resistant to sulphonamides and trimethoprim, which distinguished them from standard *S.* Typhimurium 170 strains that have been sensitive to these two antibiotics to date. Sulfas and trimethoprim are registered for use in the veterinary industry. All nine isolates had identical plasmid profiles providing further evidence of a common source. No overseas travel was reported among any of the cases. Hypothesis-generating interviews did not identify any single exposure or event common to all cases, although consumption of poultry products (eggs and/or chicken meat) were reported from all cases in the two days before onset of illness. Several potential food outlets were investigated following the collation of information from the case interviews, however, there was insufficient information to conduct any further traceback of potential food vehicles.

Queensland also reported a cluster of eight cases of *S.* Enteritidis 21b var in August. Further investigation revealed that all of the positive specimens came from the same pathology laboratory and all specimens except one were collected on the same day. Two of the eight cases had a history of overseas travel, which was not consistent with a single outbreak source. Following discussions with the pathology laboratory it was identified that all but one case resulted from a laboratory error.

OzFoodNet coordinated investigations of foodborne disease where clusters had possible multi-state spread. This included the continuing investigation into a multi-state outbreak of hepatitis A associated with an interstate gathering in the Northern Territory. OzFoodNet sites collected data from 213 participants from four states and identified 21 cases, giving an attack rate of 9.9 per cent. An examination of risk factors identified that coleslaw served at one function was the most likely food vehicle.

During the quarter, OzFoodNet sites continued to interview all cases of *Salmonella* Paratyphi B biovar Java infections to examine the role of contact with tropical fish in Australia. This investigation is in response to an increase across Australia this year, which is a concern as these infections are resistant to multiple antibiotics. Western Australia reported one case of this infection during the quarter, in a person who had a tropical fish aquarium. Testing of gravel from the aquarium was also positive for a *Salmonella* Paratyphi B biovar Java.

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Outbreak of *Salmonella* Potsdam associated with salad dressing at a restaurant

Leanne Unicomb,[1] Phillip Bird,[2] Craig Dalton[3]

Abstract

Between 27 January and 7 February 2002, 12 cases of *Salmonella* Potsdam infection were notified to NSW Health of which nine were residents of the Hunter Health Area. Interviews with two cases notified by two local doctors initiated the investigation and revealed exposure to foods from the same restaurant (restaurant A). All New South Wales *S*. Potsdam cases, those accompanying cases to restaurant A and people from restaurant A booking lists were interviewed. Of the 34 people interviewed, 17 met the case definition. The epidemiological investigation did not detect a food source of *S*. Potsdam infection, however, shell egg-based Caesar salad dressing and mayonnaise, and a swab of a cap from a mayonnaise bottle collected at restaurant A tested positive for *S*. Potsdam. Environmental and laying hen feed samples from the egg supplier to restaurant A and meat meal, (the major component of laying hen feed) tested positive for various *Salmonella* serotypes. The investigation identified problems of inadequate cleaning, time-temperature abuse, and ignorance of the hazardous nature of raw shell eggs at the restaurant level, poor sanitation and a lack of hygiene inspections at the egg production level, and problems with cleaning, storage and lack of bacterial monitoring of final product at the animal rendering plant. Investigation of 12 notified cases of *Salmonella* resulted in public health interventions, which likely prevented further cases of foodborne disease due to *Salmonella* and other pathogens in the Hunter Health Area and elsewhere in New South Wales. *Commun Dis Intell* 2003;27:508–512.

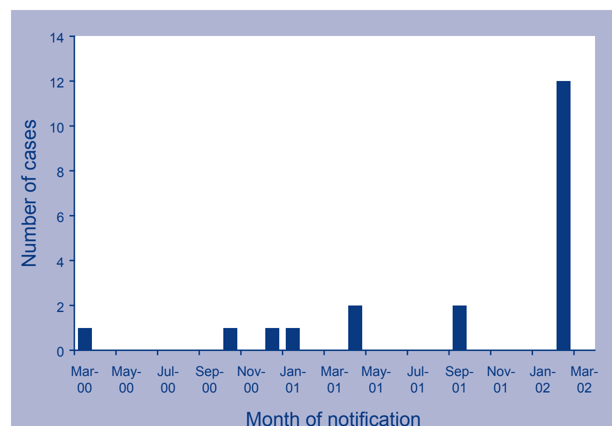
Keywords: Salmonella *Potsdam, salmonellosis, outbreak, eggs*

Introduction

Salmonella Potsdam is a relatively uncommon serotype in Australia with between 40 and 60 cases detected annually since 1991.[1] In New South Wales between 1 and 12 cases are detected annually[2] (Figure 1) and Queensland reports the greatest number of cases each year (20–40).[1] Non-clinical Australian sources of *S*. Potsdam include native animals and birds, nuts, vegetables, bottled oysters, eggs, domestic animals, farm animals, sewage effluent, spices, and meats, among samples tested between 1988 and 2002.[1]

We are not aware of published reports of outbreaks of *S*. Potsdam in Australia or elsewhere, however, an outbreak with no identified risk factors occurred in Queensland in 1988 with a total of 109 cases detected for the year.[1] During 1988, 62 of the 109 cases were detected from January to February but no investigation was reported. A further cluster of seven Queensland cases associated with foods consumed at an Asian stall at an Expo, was detected in December 1988.

Figure 1. *Salmonella* Potsdam notifications in New South Wales from March 2000 to March 2002



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On 8 February 2002 the NSW Health department received notification that *Salmonella* was detected in the stools of two patients (specimens collected on 4 and 8 February 2002) who had shared meals at restaurant A. Five of the seven people accompanying the two notified cases at restaurant A had been ill with vomiting and diarrhoea.

Methods

Epidemiological investigation

The investigating team attempted to ascertain as many cases as possible in addition to the notified *S.* Potsdam cases. This was undertaken by identification and interview of patrons from restaurant A's booking lists and interview of persons accompanying *S.*Potsdam cases to restaurant A. An ill staff member was also interviewed.

Interviews were conducted with patrons identified through the booking list who had eaten at restaurant A between 27 January and 7 February 2002, *S.* Potsdam positive cases and persons that had accompanied them to restaurant A. *S.* Potsdam positive cases were asked about their illness and asked about meals eaten outside the home in the week prior to illness and asked to describe or name the meal and beverages consumed during restaurant meals. Respondents identified through the booking list, those accompanying *S.* Potsdam cases to restaurant A and the ill staff member were asked about illness and the meal eaten at restaurant A. Those indicating that they had been ill were also asked about meals eaten outside the home in the week prior to illness, in addition to restaurant A and asked to describe or name the meal and beverages consumed. Restaurant A supplied the dinner and lunch menus. Among persons that ate at restaurant A, the menu was used as a prompt for recording meal information.

A case was defined as a person with a stool sample positive for *S*. Potsdam collected between 12 and 27 February 2002, or a person eating at restaurant A between 27 January and 7 February 2002 that developed symptoms of diarrhoea within 72 hours of consuming food from restaurant A.

Environmental health investigation

Restaurant investigations

An inspection of restaurant A was undertaken on 12 February 2002 to obtain a booking list (for the period 27 January to 7 February 2002), menus, information on staff illness, and a log of customer complaints. An environmental and regulatory investigation of the kitchen area was performed. The following day food and environmental samples were obtained. A further visit occurred on 21 February 2002 to assess compliance with previous directions and to obtain samples of all ingredients of all dressings. Information on recipes, preparation of dishes and a list of suppliers of ingredients and sources of shell eggs were also obtained. Three cases identified a second restaurant (Restaurant B) which was investigated on 7 March 2002.

Egg producer

A shell egg traceback was conducted. Between 27 January and 7 February 2002 there was a single supplier of shell eggs to restaurant A. Environmental samples were obtained from egg producer A on 18 February 2002 and submitted for microbiological examination. Each swab was taken from multiple sites to reflect the environment of the operation and not just isolated areas. Egg producer A was supplementing stocks between 27 January and 7 February 2002 with eggs from egg producer B, located in Sydney. This premises was also inspected.

Animal rendering plant

The dried feed given to laying hens by egg producer A was predominantly made up of meat meal produced by a single supplier. The plant was investigated on 5 March 2002 when samples of meat meal from the animal rendering plant were obtained and submitted for microbiological examination.

Laboratory investigations

Clinical samples were cultured for *Salmonella*, *Shigella* and *Campylobacter* species and examined for parasites by microscopy at local laboratories and *Salmonella* isolates were forwarded to the Institute of Clinical Pathology and Medical Research, Westmead, New South Wales for serotyping. Environmental samples were tested for *Salmonella* species at the Division of Analytical Laboratories, Lidcombe, New South Wales, and *Salmonella* isolates were forwarded to the Institute for Medical and Veterinary Sciences, South Australia, for serotyping.

Results

Epidemiological investigation

A total of 34 people were interviewed, 12 were identified through notifications, 14 identified using restaurant A's booking list, seven were identified by notified cases as accompanying them to restaurant A and one was a staff member from restaurant A. Of those interviewed 32 (94%) consumed food and/or beverages at restaurant A. Seventeen persons (50%) met the case definition, 12 (71%) had *S.* Potsdam detected in stool samples, the remaining

five cases were identified through interview but did not have a stool sample collected. Of the 17 cases, 10 had eaten lunch, two had eaten dinner, two had consumed coffee, two had not eaten at restaurant A and one staff member ate an item from the lunch menu (Table). Of the four *S.* Potsdam cases that did not eat a meal at restaurant A, two consumed coffee at restaurant A and ate dinner at restaurant B, one worked in the kitchen at restaurant B and one was a baby with no apparent connection to restaurant A or B. Thus 15/17 cases ate or consumed a beverage at restaurant A. The epidemic curve is shown in Figure 2.

Table. Summary of meals consumed at restaurant A among persons interviewed

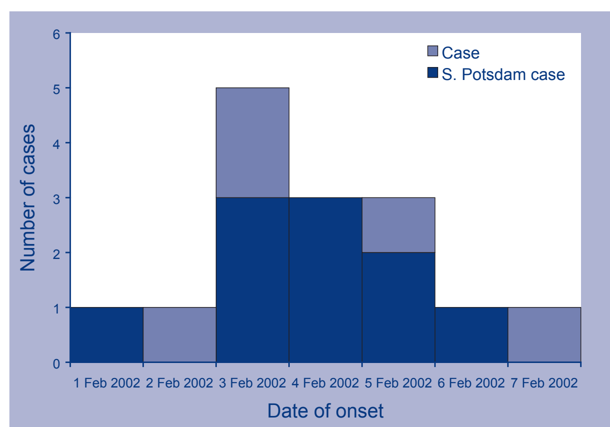
Restaurant meal	Interviewed	Number ill	*S.* Potsdam positive
Dinner	14	2	2
Lunch*	14	11	7
Coffee	4	2†	1
No food or beverage	2	2	2
Total	34	17	12‡

* Includes one staff member that ate an item from the lunch menu.

† 2/2 ate dinner at restaurant B.

‡ All ill.

Figure 2. Number of cases of gastrointestinal illness at restaurant A in New South Wales, 1 to 7 February 2002, by date of onset*



* (N=15, no onset date was recorded for 2 cases)

Of the 17 respondents that met the case definition, 18 per cent were males, 82 per cent were females, and the median age of cases was 28.8 years (range 1–77 years), not significantly different from the median age of those without illness (44.3 years, range 3–71 years). Symptoms included diarrhoea (94%), cramps (88%), nausea (65%), fever (59%), headache (53%), joint pain (35%), vomiting (29%) and lethargy (24%). Of cases with diarrhoea (N=16), none had blood in their faeces. One case reported faecal incontinence, one case reported metallic taste, one case reported loss of sensation in hands, and one case reported light-headedness. The median incubation period was 21 hours (range 3.5 to 95 hours) and the median duration of illness was five days (range 2 to 8 days). Eleven cases (65%) consulted a general practitioner, one person went to an emergency department and two were hospitalised.

An analysis of foods consumed at restaurant A revealed that cases ate a variety of foods with no particular menu item commonly eaten. Of the 15 cases that ate or consumed a beverage at restaurant A, 4 (27%) ate menu items that included egg-based dressing. Three cases implicated restaurant B but had eaten different meals at restaurant B. Furthermore, two of the three consumed coffee and biscuits at restaurant A.

Environmental investigation

Restaurant investigations

During the inspection of restaurant A undertaken on 13 February 2002 it was indicated that the majority of meals were served at lunchtime with approximately 300 lunches served on a typical day. Of food and environmental samples collected, Caesar dressing, dill mayonnaise, and the cap of the dill mayonnaise bottle tested positive for *S.* Potsdam. *Salmonella* species was not detected in samples of sweet chilli dressing, pesto dressing, olive dressing, whole shell eggs, and caps from other dressing bottles. No *Salmonella* species was detected in dressing ingredient samples collected on 21 February 2002. During an inspection it was noted that the procedure for preparing dressings involved making a single batch of mayonnaise (using raw, whole, shell eggs), which was divided to make the Caesar dressing and the four other types of mayonnaise. The laboratory recorded pHs of the mayonnaises ranging from pH 3.4 to 5.4.

Kitchen staff reported that dressings were stored in multiple dispenser bottles and that several partly used dressing stocks could be in use at a given time. The base-mix was not made fresh each day and topping-up of bottles occurred, often in anticipation of peak sales periods. This suggested that some stocks of raw egg containing mayonnaise were prepared several days prior to serving. On inspection the dispenser bottles used to store dressings were observed to be non-re-useable, stained and perished and contained food residues and odour. Thus restaurant A was not able to effectively clean the bottles. Several plastic bottles had become soft and tacky further hampering cleaning. Staff reported that the ready-to-use dressings in dispenser bottles were often kept out of the refrigerator for extended periods of time at warm room temperature.

The kitchen was observed to be very small (2 m x 7 m with approximately one-third of that space for food preparation) given the number of meals prepared on a typical day. The intensive use of food preparation areas provided many opportunities for cross-contamination between raw and prepared foods. Numerous breaches of food regulations were detected which resulted in the entire restaurant being disinfected under the supervision of food inspectors on 21 February 2002 to prevent a recurrence.

Egg producer investigation

Egg producer A produced approximately 300,000 shell eggs per week, mostly for the Hunter region in New South Wales and mostly for restaurants and cafes. Of the 16 environmental swabs obtained, 12 were positive for *Salmonella*. No *S*. Potsdam was detected, however, *S.* Agona was found in swabs obtained from egg collection trolley wheels, egg racks of the tier egg laying frame, and feed troughs of the tier egg laying frame. *S.* Infantis was detected in swabs from the egg cleaning cloth, egg racks of the tier egg laying frame, egg collection trolley wheels, and feed troughs of the A-frame laying cages. *S.* Broughton was detected in swabs from egg racks of the tier egg laying frame. Significant food safety deficiencies were identified through the entire production chain. Investigation of egg producer B failed to detect any likely source of *Salmonella* contamination. During the investigation egg producer A reported that there had been no inspection undertaken in over 10 years. It was determined that routine food hygiene inspections of egg producers in New South Wales had not been undertaken since the Egg Board disbanded in 1990.

Animal rendering plant investigation

Meat meal was the major component of laying hen feed at egg producer A and was found positive for *S*. Agona. The capacity of the producer was approximately 100 tonne per week, 97 per cent of which was supplied for broilers to a poultry producer other than egg producer A. The multiple-part, combined sample that was obtained was positive for *S*. Johannesburg. At the animal rendering plant, no documentation of the validation of the rendering process was found, no bacterial monitoring of the product was undertaken and storage bins were never cleaned. Furthermore, the Australian Standard for Hygienic Rendering of Animal Products permits the presence of *Salmonella* in three of the most recent 10 samples of the final product.[3] Transport of the meat meal by egg producer A was reported to be undertaken in a dirty truck indicating an ignorance of the need to keep the product under hygienic conditions.

Discussion

S. Potsdam is a relatively uncommon serotype with between 1 and 12 cases detected annually in New South Wales between 1991 and 2001. NSW Health detected a cluster of 12 notified cases between 12 and 27 February 2002 who predominantly (9 of 12) resided in the Hunter Health Area of New South Wales. During this investigation the investigation team found a link between illness due to *S*. Potsdam and eating at a restaurant with 10 of 12 *S*. Potsdam cases having consumed food or beverage at restaurant A in the 72 hours prior to onset.

S. Potsdam was detected in shell egg-based dressings collected at restaurant A up to 12 days after the first case had eaten there but in no ingredients of the dressings**.** There was a practice of not completely emptying the dressing in dispenser bottles and topping them up with fresh dressing. The dispenser bottles filled with dressing were known by staff to be kept on the bench during busy periods. The high ambient temperatures that occur during February likely created an environment for *Salmonella* to flourish. Attempts to determine the origin of the pathogen in food ingredients were made by sampling all ingredients of the various dressings (all negative for *Salmonella*) and inspecting and sampling at the egg producer and animal rendering plant. *S*. Potsdam was not found in any of these samples, however, there was gross environmental

contamination and a high occurrence of *Salmonella* contamination detected at the egg producer. There was evidence of contamination of the surface of shell eggs since the wet cloth used to wipe dirty eggs at the egg producer was positive for *S*. Infantis. These findings coupled with the fact that egg was an ingredient common to all *S*. Potsdam positive dressings suggest that shell eggs were the most plausible source of *S*. Potsdam. While information obtained at interview could only attribute 27 per cent of cases to dressing consumption, kitchen practices were conducive to cross-contamination which likely explained further cases.

Egg-associated outbreaks of salmonellosis and other foodborne illnesses have been reported in Australia and elsewhere.[4,5] During 2001 and 2002, 13 egg-associated outbreaks not including this outbreak, were reported in Australia, all of which were due to salmonellosis (OzFoodNet Outbreak Register, M. Kirk, personal communication, January 2003). *S*. Potsdam has been detected in egg samples from Victoria in 1982, egg samples from Western Australia in 1985 and 1990,[1] and *Salmonella* species has been detected on the surface of eggs,[7] and in this investigation, on the cloth used to wipe eggs. Furthermore, an outbreak of salmonellosis in South Australia has been linked to raw egg used in Caesar salad dressing (OzFoodNet Outbreak Register, M Kirk, personal communication, January 2003).

This outbreak highlighted a number of important issues. The storage at room temperature of raw shell egg-based dressing and dishes or condiments that are not further cooked should be discouraged. Dressings should be made fresh daily and the temperature of contents be maintained at ≤5° C. Plastic dispenser bottles, which were not intended for re-use, should not be used for storing food. SafeFood Production, New South Wales is in the process of developing a food safety scheme for egg producers, in consultation with the egg industry in response to issues raised during this investigation.

As this investigation has shown, a comprehensive through-food-chain approach of investigating small clusters of *Salmonella* can have an impact on the food industry. On-going monitoring and inspection within the context of accredited Hazard Analysis and Critical Control Point programs will be an important public health intervention.

Acknowledgements

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An outbreak of *Salmonella* Typhimurium phage type 4 linked to cold set cheesecake

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On 3 September 2003 the Australian Salmonella Reference Centre in Adelaide notified the Communicable Disease Control Branch, Department of Human Services, South Australia, of four cases of *Salmonella* Typhimurium phage type 4 infection. A fifth case was notified on 24 August and all cases had dates of onset between 16 and 19 August 2003.

Hypothesis generating interviews identified consumption of cheesecake in four of the five cases. Cheesecakes were consumed in three different commercial food outlets in metropolitan Adelaide. A trace back investigation revealed that they were supplied by a common bakery. Two different flavoured cheesecakes were consumed. All were prepared by the cold set method in which commercially supplied cream cheese, sugar, cream, milk and gelatine were mixed and poured into a hand-moulded biscuit crumb and butter base. No cooking was involved in any stages of preparation of the cheesecakes. Cream via piping bags and toppings were applied after the cakes had set. Cold set cheesecakes were distributed to 15 other outlets and sold between 12 and 18 August 2003.

A case control study commenced on 8 September 2003. A case was defined as a person with microbiologically confirmed *Salmonella* Typhimurium phage type 4 infection with date of onset of gastrointestinal symptoms between 11 and 31 August. Each case was matched to three controls by age, sex and postcode. One additional case was notified on 10 September bringing the total number of cases to 6 (2 male, 4 female). Cases were aged between 3 and 82 years (median 22 years) and distributed throughout metropolitan Adelaide. The most common symptom reported was abdominal pain (6, 100%), diarrhoea, nausea and vomiting (5, 83%). Bloody diarrhoea was reported by 3 (50%) cases and one case was hospitalised. The median incubation period where known (3 cases), was one day (range 1 to 2 days) and the median duration of illness was 13 days (range 7 to 17 days) with one case still ill after 58 days. Analysis showed an association between consumption of cheesecake and illness. Of the six cases, 4 (67%) reported consumption of cheesecake.

An environmental inspection of the bakery was conducted on 6 September. The inspection identified potential sources of *Salmonella* including several cracked and faecally contaminated eggs amongst the egg supply, frequent bare hand contact with the cold set cheesecake in its manufacture, staff unable to demonstrate how to prepare sanitiser at correct concentration and long standing food residues adhered to mixing equipment. There were no reports of gastrointestinal illness in bakery staff. Ten environmental samples were taken from piping bags, mixing equipment, wash basin tap handles, cracked and faecally contaminated eggs, cold set cheesecake and cream. The bakery discarded all cold set cheesecakes and suspended production of the product pending microbiological results. All samples were negative for *Salmonella* sp.

In Australia, food businesses must comply with the requirements of the Food Safety Standards. The requirements address broad issues which apply to all food businesses such as storage, temperature control, hygiene of food handlers and cleaning and sanitising of equipment, amongst many others. This is the third outbreak of *Salmonella* Typhimurium infection in South Australia since 2001 that has been linked to consumption of sweet bakery products.[1,2] A specific source was identified as piping bags in one outbreak but was unknown in this and a second outbreak. These outbreaks raise concerns about food-handling practices in bakeries. Bakery-specific food safety guidelines should be developed that address issues including cleanliness of the egg supply, storage, preparation and handling of dairy-based products and fillings (in particular the use of piping bags) and the potential for cross-

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contamination between products. These can be used as a supplement to the Food Safety Standards to prevent future outbreaks.

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Salmonella Typhimurium U290 outbreak linked to a bakery

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This report summarises the investigation of the first documented outbreak of *Salmonella* Typhimurium Phage type U290 exhibiting tetracycline resistance.

Typhimurium is the most commonly isolated *Salmonella* serovar in Australia and is responsible for the majority of identified outbreaks of bacterial gastroenteritis.[1,2] *S.* Typhimurium U290 has been recognised as a distinct phage type in Australia since October 2000, and re-examination of phage reaction records for unclassifiable isolates by the University of Melbourne's Microbiological Diagnostic Unit (MDU) suggests that the first case occurred in New South Wales in October 1999.

Since then and until this outbreak, only 96 human isolates and 14 non-human isolates (from broiler chickens, chicken litter, pet dogs and horses)[1,3] had been notified to the National Enteric Pathogen Surveillance Scheme (NEPSS) .

On 31 May 2002, the Communicable Disease Section of the Department of Human Services (DHS) Victoria identified a cluster of five salmonellosis cases in an area of Northern Victoria (Area A).

An outbreak investigation team was formed under the auspices of OzFoodNet and DHS to determine the source and prevent further infection.

Other state and territory health authorities were notified of the cluster via the OzFoodNet network and asked to report recent cases of this phage type in their jurisdictions.

The initial case definition was 'any laboratory confirmed *S.* Typhimurium U290 infection notified after 30 May 2002'. Cases were interviewed, focusing on food and environmental exposures in the fortnight preceding onset. Active case finding was initiated by alerting general practice clinics in Area A, and requesting the collection of faecal samples from suspected cases.

Upon identifying a suspected common time and place of exposure, we undertook a case control study, refining the case definition to 'any person with laboratory identified *S.* Typhimurium U290 infection, onset of illness from 30 April 2002 onward, and residing in or having visited Area A'. Controls within Area A were selected by progressive digit dial, excluding those under five years of age, or who had pre-existing illness. Cases and controls were interviewed regarding foods eaten from vendors in Area A on the weekend prior to onset of illness.

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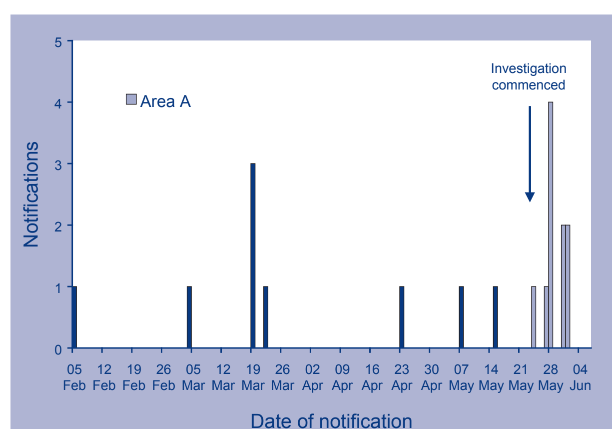
The Local Government Environmental Health Officer and the Regional Environmental Health Officer inspected Bakery X in Area A, and food and environmental samples were later obtained for testing. Food handlers from Bakery X were also interviewed regarding history of illness and potential for contamination of foodstuffs.

The MDU laboratory phage-typed all human isolates, and tested environmental and food samples obtained during the investigation.

A total of 10 cases of *S.* Typhimurium U290 were identified (Figure) and interviewed during this investigation. Consumption of food from one bakery (Bakery X) over the weekend 17–19 May 2002 was identified in nine cases. Area A is a popular tourist centre and Bakery X estimated that over 3,000 customers were served during this weekend.

The case control study results (Table) indicated that illness was strongly associated with eating at Bakery X and that cases were more likely than controls to have consumed cream and/or custard filled products from this bakery.

Figure. Victorian *Salmonella* Typhimurium U290 notifications and Area A cluster



Inspection of Bakery X revealed the potential for cross-contamination of food through the use of certain food handling practices and specialised equipment, specifically the use of an egg pulper, an industrial cream whipper and cloth piping bags. Effective control measures for cross-contamination were discussed with senior bakery staff and a clean up of the premises undertaken.

Three out of 37 food-handlers interviewed described having gastroenteritis coinciding with the onset period in cases, and all continued working whilst symptomatic. Proprietors were reminded that food-handlers should not work while suffering from gastroenteritis, and this was re-enforced during staff food hygiene and safety training sessions conducted in collaboration with the regional environmental health officer.

This is the first reported outbreak of *S.* Typhimurium U290 in Australia, and was epidemiologically linked to bakery products. The outbreak has public health importance for several reasons. Firstly, OzFoodNet reported four other bakery-related outbreaks of salmonellosis during 2002.[4] Secondly, *S.* Typhimurium U290 appears to be emerging across Australia with 122 notifications of apparently sporadic cases since this outbreak up to August 2003 (NEPSS, personal communication, 2003); Finally, this organism shows resistance to the antibiotic tetracycline.

Infection with *S.* Typhimurium U290 was strongly associated with the consumption of cream and/or custard-filled products from Bakery X. The literature suggests that contaminated eggs or cream are likely sources of infection in bakery-related outbreaks.[5–9] Whilst raw eggs were not used in custards and creams at Bakery X, the use of an on-site egg pulping machine created potential for cross-contamination.

Table. Results of *Salmonella* Typhimurium U290 outbreak case control study

Exposure	Cases (n=10)			Controls (n=24)			OR	(95% CI)
	Yes	No	% exposed	Yes	No	% exposed		
Ate bakery products (any bakeries)	10	0	100	10	13	43	1.30	0.33-5.13
Cream	6	4	60	4	19	17	7.13	1.06-55.2
Custard	5	5	50	5	18	21	3.60	0.58-24.1
Cream AND custard	9	1	90	10	13	43	11.7	1.1-289
Ate bakery X products	9	1	90	3	20	15	60	4.4-1890
Cream	6	4	60	0	23	0	Undefined	–
Custard	3	7	30	0	23	0	Undefined	–
Cream AND custard	9	1	90	0	23	0	Undefined	–

It seems likely that low level contamination of the cream occurred during preparation, and this theory is supported by the fact that only 10 cases were confirmed even though several hundred cream and/or custard products were sold over the weekend in question. Six cases reported time-temperature abuse of the bakery products after purchase and before consumption, allowing for bacterial growth, which further supports this scenario. It is possible that the magnitude of this outbreak was moderated by the cool temperatures of the Australian winter. Given the large customer base and post-purchase handling, this outbreak could have had far-reaching effects had it taken place in the summer, as was demonstrated recently by a large bakery-related outbreak in Spain.[9]

As a result of this outbreak investigation specific control measures were put in place to reduce the risk of cross contamination during food preparation. Particular emphasis was placed on the importance of hand washing when pulping eggs and whipping cream; the use of covers to reduce splashing; washing and sanitising of utensils and containers used with raw egg pulp; and, the use of dedicated work areas separate from other food preparation. Cloth piping bags have been replaced with single-use disposable piping bags. Specific training has been instigated to raise staff knowledge of the public health issues related to working while symptomatic of gastroenteritis.

The source and mechanism of contamination remain unidentified, however the investigation positively influenced the food handlers' knowledge and practice regarding illness and food handling.

Acknowledgments

We gratefully acknowledge the following people for their invaluable assistance in this investigation: Leanne Roberts (DHS Hume Region); Mark D'Agostino, Karen Carter, Catherine Ferreira, Joanna Gaston, Kerry-Ann O'Grady, Ross Andrews, (DHS Victoria Communicable Diseases Unit); Victor Di Paola, Violette Lazanas, (DHS Victoria Food Safety Unit); the proprietors and staff of Bakery X; Mary Beers, Gillian Hall, (NCEPH); Martyn Kirk (OzFoodNet Co-ordinating Epidemiologist); Stephen Lambert, (Royal Children's Hospital); special thanks to Joan Powling, Diane Lightfoot, Mary Valcanis (Microbiological Diagnostic Unit).

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Investigation of two clusters of shiga toxin-producing *Escherichia coli* cases in South Australia

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Shiga toxin-producing *Escherichia coli* (STEC) is an important cause of gastrointestinal illness in developed countries, and outbreaks have been reported in many countries including Australia.[1,2] STEC infection can cause bloody diarrhoea, with 3–7 per cent of sporadic cases developing haemolytic uraemic syndrome (HUS), a serious condition, defined by thrombocytopenia, anaemia and renal failure, which can result in death.[1,2,3] In Australia, the number of reported STEC cases, was 38, 48 and 52 in the years 2000, 2001 and 2002 respectively.[4] South Australia reported 38 (73%) of the 52 cases in 2002. Current surveillance STEC practices in South Australia involves screening of all bloody stools with a polymerase chain reaction (PCR) test for the toxin genes, which contributes to the number of cases reported from this state. Samples positive for toxin are tested for virulence and serotype genes.[5] This procedure complements standard epidemiological practices.

Relatively few STEC outbreaks have been reported in Australia.[2] This report describes the investigation of two clusters of STEC cases in South Australia, observed in February and March 2003.

The first cluster

Between 3 and 6 March 2003, four STEC cases from Adelaide suburbs were notified to the Communicable Disease Control Branch (CDCB). Of the four cases, three were PCR positive for serotype O157 and toxin gene STX2 and negative for toxin gene STX1. One O157 case was also positive for virulence genes *eae* (codes for intimin) and *hlya* (codes for enterohemolysin). The other two cases were negative for these genes possibly due to a low number of STEC in the stool sample. These typing results suggested that the cases may be epidemiological linked.[5]

These three cases were interviewed with a hypothesis generating questionnaire, which included a 10 day food history prior to illness, food purchasing habits and social activities undertaken during this period.

Of the three cases, one was male and two were females and all were aged 61 years or more. All three cases had bloody diarrhoea with dates of onset of 25, 27 and 28 February 2003. Two were hospitalised for a week and there were no reports of cases developing HUS.

These cases had no contact with each other and the food history revealed no common food vehicle or other exposure. However, the male case and a female case reported eating Hawaiian pizza. In addition, both female cases purchased meat and small goods including fruit and vegetables from the same supermarket.

The stool samples were cultured and *E. coli* isolates screened for STEC toxin genes. Unfortunately, isolates were only recovered from the male case and one of the female cases, who did not eat pizza. These isolates were subjected to PFGE using restriction enzyme *Xba*1 (New England Biolabs), and analysed using the software GelCompar 4.1 (Applied Maths, Sint-Martens-Latem, Belgium). The PFGE gel was interpreted according to Tenover criteria for strain identification.[6] Results showed that the isolates from community cases 1 and 2 had different patterns from each other (Figure) and from other STEC control isolates (community case 3). Overall, the molecular and descriptive epidemiology suggests that the two cases were unrelated. As an isolate was not cultured from one of the female cases it is unclear if the two female cases were associated with a common food source or other exposure. There were no further reports of STEC O157 with similar dates of onset.

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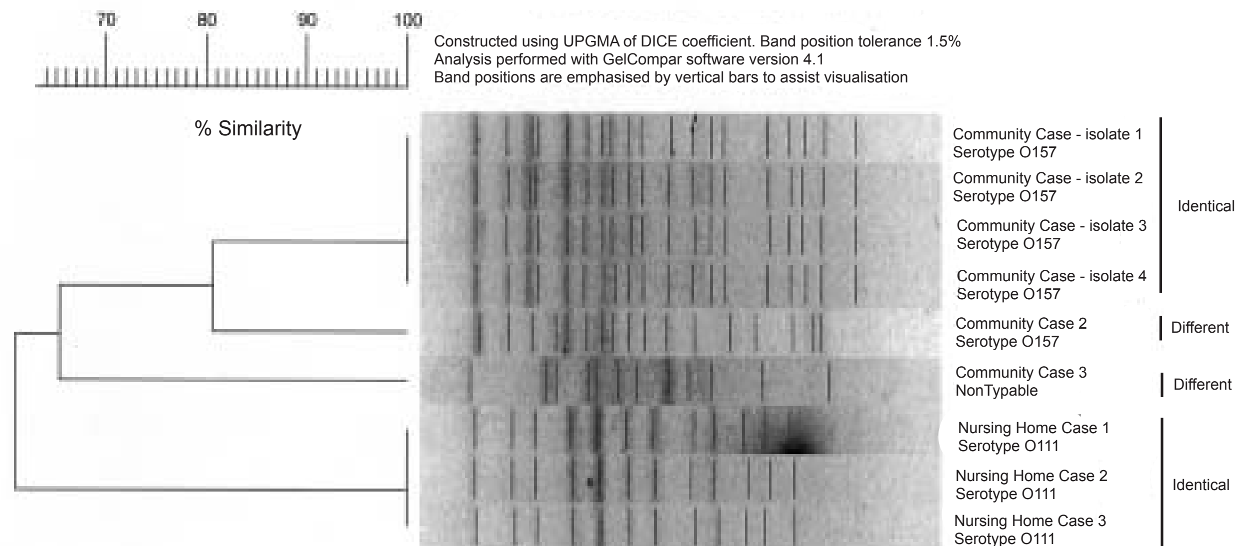
Figure. Dendogram of Pulsed Field Patterns of STEC isolates from a community cluster and a nursing home cluster



The second cluster/outbreak

On 20 March 2003, two STEC cases from a nursing home were notified to the CDCB and epidemiological, environmental and microbiological investigations were initiated. At the nursing home there were 115 residents and approximately 112 staff members. Investigations revealed 12 residents and one staff member had gastrointestinal illness. Stools from all people who were ill were screened for enteric pathogens. All were negative except for one more resident having a stool positive for STEC toxin genes.

The three laboratory confirmed cases, were aged 79 to 87 years, and all experienced diarrhoea. Two of these cases experienced bloody diarrhoea and were hospitalised. The dates of onset were 13, 14 and 16 March 2003.

The cases lived in different sections of the nursing home but staff reported occasionally working across all sections. There was a set menu for residents and two cases may have had contact with each other at lunch times in the dining room. The third case required feeding assistance and did not attend the dining room. Each section of the nursing home had a kitchen where kitchen staff prepared main meals, including snacks. The food safety methods and food hygiene practices in these kitchens were found to be satisfactory. Ingredients for meals were supplied by an external food manufacturing facility, which was reported to be well maintained and had good systems in place for food preparation. At the manufacturing facility there was no evidence of gastrointestinal illness among staff or among people at other locations that the facility supplied. In the 10 day period prior to illness there were no social activities when all three cases could have interacted.

STEC isolates were recovered from the three cases and were serotyped O111, which is a common STEC serotype within South Australia.[2] The isolates were PFGE typed and were found to have a common banding pattern (Figure), further confirming that the cases were epidemiologically linked. From the epidemiological and environmental investigations no common foodborne vehicle of infection was found. The only hypothesis that the descriptive epidemiology suggested was person-to-person transmission, perhaps via staff members.

Discussion

STEC continues to be an important cause of gastrointestinal illness in South Australia. Most infections are sporadic cases with occasional outbreaks identified. In 2002, there was an outbreak of STEC associated with children visiting a petting zoo.[7]

In the investigation of the O157 cluster described above, the PFGE typing later helped confirm that at least two of the cases were not linked despite cases having a similar date of disease onset, having the same serotype and toxin gene profile, and being located in the same region of metropolitan Adelaide. Evaluation of the USA Pulse Net system found that PFGE was useful as an adjunct and not a replacement of epidemiological investigation. In the United States of America (USA), PFGE of STEC isolates has been used to confirm that clusters of cases are not related thus avoiding further costly epidemiological investigation, especially when typing is carried out in a timely manner.[8]

In the O157 cluster and nursing home outbreak, only people over 60 years of age were involved. STEC outbreaks in nursing homes have also been reported in Canada and the USA.[9,10] This emphasizes the need for high levels of food hygiene and infection control procedures in institutions caring for the elderly, who are at a higher risk for STEC infection. At the South Australian nursing home, the CDCB and local government officers reinforced infection control procedures, which included the need for regular hand washing, food handling procedures, environmental cleaning and correct linen handling procedures. Intensive surveillance of the nursing home continued for four weeks and residents continued to have stools screened for STEC toxin genes.

Acknowledgement

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Using the national guidelines to manage a meningococcal group C outbreak in a Brisbane boarding school – some discretionary judgements are needed

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Abstract

The management of an organisational outbreak of meningococcal disease using the national *Guidelines for the early clinical and public health management of meningococcal disease in Australia*[1] (the Guidelines), could be considered a relatively straightforward task. Nevertheless, discretional judgements are often still required by the outbreak control team, as no guidelines can fully cover every eventuality. The greatest challenges generated by this outbreak did not result from the magnitude of the intervention, but from the difficulties in defining the margins of the intervention in the face of heightened community and professional concern. Also Public Health decisions and communication strategies needed to be responsive to these concerns. *Commun Dis Intell* 2003;27:520–523.

Keywords: meningococcal disease, outbreak

Introduction

School–based clusters of meningococcal disease are uncommon. A United States of America study found that such clusters had been infrequently reported and risk factors for transmission in school settings were unclear.[2] In Australia, meningococcal serogroup C outbreaks have been reported in a university residential college in Sydney in 1997,[3] a Victorian school in 1999[4] and there have been five other clusters in north Queensland during 1990–1994[5] and one in Western Sydney in 1996.[6]

The Communicable Diseases Network Australia (CDNA) Guidelines[1] were developed to assist public health practitioners with the management of sporadic cases as well as outbreaks of invasive meningococcal disease. They also include excellent supporting material such as meningococcal fact sheets and sample letters. The Guidelines[1] were used extensively in defining and managing this outbreak, but the Outbreak Control Team (OCT) was required to make and defend discretionary judgements and develop new and amended resources, at several points in the process.

Description of the outbreak

On 5 August 2001, a 13-year-old student who attended a Brisbane boarding school, developed meningococcal septicaemia. The case died shortly after admission to hospital. The organism was later reported as a meningococcus serogroup C. All boarding students had individual bedrooms. As per the Guidelines,[1] the case was managed as a sporadic case and the Public Health Unit organised rifampicin chemoprophylaxis for identified close contacts, which consisted of the case's parents and 23 boarding school contacts who had spent time with the case in the school infirmary. In addition, all parents at the school were provided with a letter of advice and a meningococcal fact sheet.

On 15 August 2001, 10 days after the onset of symptoms in the first case, another case of meningococcal septicaemia occurred in a 16-year-old student who attended the same boarding school. The case deteriorated rapidly after admission to hospital and died. Although both cases had been boarders at the college, there was no evidence of social contact between the two students. One student was in the

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junior school and the other in the senior school. The boarding houses attended by both students were separate buildings with separate facilities such as dining rooms and recreation rooms. Therefore initially, until the availability of laboratory results, this case was also managed as a sporadic case. The Public Health Unit offered chemoprophylaxis to six household contacts and one identified close contact. Once again, all parents at the school received a letter informing them of the situation.

On 16 August 2001, the Queensland Health Scientific Services (QHSS) laboratory determined, by nucleic acid amplification of the cerebrospinal fluid sample from the second case, that the meningococcal organism from the second case, was a serogroup C. Restriction digestion of the amplified Por A gene, using Alu I, Hinf I, Hpa II, Rsa I and Hae III enzymes, showed that the subtypes were identical between the first and second cases. This result was available on 17 August 2001. The culture from the second case was not available until 20 August, when typing using Pulsed Field Gel Electrophoresis (enzymes Spe I, Bgl II and Nhe I) showed that the cultures were genetically indistinguishable from each other. The isolates were serosubtyped as C:NT:P1.15.

Outbreak management

An Outbreak Control Team was convened on 16 August 2001 to assist in the planning and management of the outbreak. Two serogroup C meningococcal cases in the school boarding population within a two week period, fitted the CDNA Guidelines[1] definition of an organisational outbreak. The Guidelines[1] recommend that members of the same group as the cases, should receive chemoprophylaxis and vaccination. In this situation the 'same group' would be defined as only the boarding school population. There were 450 boarders, 1,000 day students and approximately 380 staff, some of whom lived with their families on the college grounds. The OCT decided that restricting the chemoprophylaxis/vaccination intervention to just the boarding school community would increase the existing intense levels of anxiety in the remaining school community, caused by the sudden death of two students, to unmanageable levels. The second case had played in three football games in two different codes on the weekend prior to his illness and had also trained with a State representative team on the day prior to his illness. The football coaches had reported that it was common practice for team players to share saliva via water bottles and other means. The OCT decided that the program would be extended to members of all seven football teams who played with or against the case in the previous week. This issue is not addressed in the Guidelines.

The OCT declared an organisational outbreak and implemented the following actions:

- polyvalent ACWY meningococcal polysaccharide vaccine would be provided (the conjugated vaccine was not yet available in Australia);
- the antibiotic ciprofloxacin would be used for chemoprophylaxis. This decision was based on the fact that there were no known contraindications, all of the intervention group were over 12 years of age and a single 500 mg ciprofloxacin tablet given to each person prior to vaccination made it easy to supervise administration and to ensure compliance;
- laboratory surveillance would be strengthened; and
- increased clinical surveillance would be encouraged by informing every general practitioner, Emergency Department and Community Health Service on the Northside of Brisbane, requesting early notification of suspected cases and seeking their support for the proposed intervention.

The vaccination and chemoprophylaxis program commenced on Friday 16 August, with clinics being conducted on Saturday 17 August and Monday 19 August. Thirty-six Queensland Health staff from various public health service units and hospital districts throughout South East Queensland were brought in for the intervention. The school provided five nursing staff and three teaching staff to assist. A total of 1,840 persons were vaccinated and 1,768 persons were provided with chemoprophylaxis. Funding required for the vaccines, antibiotics and disposables (but not including labour costs) was in excess of $70,000.

School community acceptance of the outbreak intervention

No parents or person withheld their consent for vaccination, although a small number chose not to have the antibiotic. Recorded reactions to the vaccination/chemopropylaxis were minor. There were two students who fainted and two staff members who developed an itchy rash that resolved with antihistamines. In addition, the School Health Centre reviewed approximately 10 students/staff who presented with a range of symptoms such as headaches, fevers and rashes. One febrile student was admitted to hospital for observation and later discharged.

Managing the media and public concerns

From the beginning of the outbreak it was obvious that managing the media and public concerns would require substantial time and resources. The manager of the Queensland Health Communicable Diseases Unit took sole responsibility for media management during the intervention with the on-going support of the Public Health Services Senior Marketing and Communications Officer. From 6 to 25 August, communications officers responded to 166 media inquiries. There were approximately 530 electronic media reports in Queensland and approximately 150 stories in Queensland newspapers during this period. On 22 August, the 'outbreak' was number five in the National top ten of media issues with 241 stories being published. In response, Queensland Health set up an information hotline staffed by at least four officers full time over a one week period. There were over 3,300 calls to the hotline.

Enhanced surveillance

During the intervention the enhanced clinical and laboratory surveillance that had been initiated by the OCT identified two further cases.

On 18 August, three days after onset of illness in the second case, and after outbreak management had been implemented at the boarding school, a 13-year-old girl from an associated girls school developed meningococcal meningitis. A decision was made to continue to manage this as a sporadic case. Some time later the Public Health Unit was informed that the case was serogroup C, genetically indistinguishable from the other cases.

On 4 September, 17 days after the third case, an 8-year-old girl presented to a local hospital with a febrile illness. Blood cultures were collected and she was sent home. The blood cultures grew meningococcus, which was later confirmed as serogroup C, genetically indistinguishable from the other cases. Her parents worked at the boarding school and both had received antibiotics and vaccination 15 days earlier. This case was also managed as a sporadic case.

The guidelines do not specify how to respond to cases with genetically identical organisms occurring in cases in the same community, but outside the immediate contacts of the cases. The OCT decided not to extend the campaign as a result of these cases. No further cases of the outbreak genotype meningococcal group C disease were notified in the following months.

General discussion and difficulties experienced

The logistics of implementing such a large intervention required considerable planning and organisation of human and material resources. Nevertheless, the greatest challenges generated by this outbreak did not result from the magnitude of the intervention, but from the difficulties in defining the margins of the intervention in the face of heightened community and professional concern and pressure, and the occurrence of further cases with the same meningococcal genotype in the community. Specifically, there was intense pressure on the OCT to widen the intervention and to provide chemoprophylaxis and/or vaccination to many other associated individuals and groups. These issues were addressed by extensive use of the Guidelines,[1] consultation with the highest levels of expertise from across Queensland and Australia, establishing tight definitions of the eligible population and holding to those definitions.

General practitioners and hospital doctors greatly appreciated the early information about the outbreak, the intervention and the reasons for the public health decisions. As the intervention progressed, followup briefings to enable them to respond to emerging issues from their patients and the general community were provided. Providing such early and comprehensive information benefited all parties and ultimately reduced the workload on the public health staff.

Although the Public Health Unit had comprehensive information sheets on the disease and the chemoprophylaxis regimens (some from the National Guidelines), over time it became obvious that most of the questions from the general public were about why they too should not have the vaccine. The early availability of a fact sheet addressing this issue would have alleviated much of the community concern, and saved considerable time for the public health workforce. A fact sheet addressing this issue was developed during the intervention.

It is anticipated that the lessons learnt will assist us and others if similar situations occur in the future.

Acknowledgments

This public health response was made possible by the close cooperation between the local Public Health Unit, the Communicable Disease Unit, the laboratory staff of QHPSS, the District Nurses, Public Health Nurses, Public Health Medical Officers and other Public Health Service staff. A special acknowledgment goes to the families of the cases, the management of the school, the nursing and teaching staff of the school and the students and parents, whose assistance and cooperation enabled the successful implementation of the mass vaccination and chemoprophylaxis intervention. Thanks also to the staff throughout Queensland Health who covered the information hotline and other telephone calls associated with the outbreak, and to Dr John Sheridan and Dr Andrew Langley who proofread the drafts.

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Composition of Australian influenza vaccine for the 2004 season

In order to select virus strains for the manufacture of Influenza Vaccine for 2004 Season, a meeting of the Australian Influenza Vaccine Committee on Influenza Vaccines was convened on 10 October 2002.

Having considered information on international surveillance by the World Health Organization (WHO), and up-to-date epidemiology and strain characterisation presented at the meeting, the Committee considered that the WHO recommendations on the composition of vaccines for 2004 Southern Hemisphere Season should be followed.

A H1N1 strain:	A/New Caledonia/20/99(H1N1)-like strain A/New Caledonia/20/99 (IVR-116) is also recommended as a suitable vaccine strain.	15 µg HA per dose
A H3N2 strain:	A/Fujian/411/2002(H3N2)-like virus A/Kumamoto/102/2002 (IVR-135) and A/Wyoming/3/2003 (X-147) are also recommended as suitable vaccine strains.	15 µg HA per dose
B Strain:	B/Hong Kong/330/2001-like virus B/Brisbane/32/2202 is also recommended as a suitable vaccine strain.	15 µg HA per dose

Further details available on: http://www.tga.gov.au/docs/html/aivc2004.htm or http://www.influenzacentre.org/

Reporting of communicable disease conditions under surveillance by the APSU, 1 January to 30 June 2003

Compiled by Elizabeth Elliott, Donna Rose
Australian Paediatric Surveillance Unit

Background

The Australian Paediatric Surveillance Unit (APSU) was established in 1993 and is a unit of the Division of Paediatrics and Child Health, Royal Australasian College of Physicians. The activities of the APSU are funded in part by the Australian Government Department of Health and Ageing through the communicable diseases program. The APSU is a founding member of the International Network of Paediatric Surveillance Units (INoPSU). INoPSU now has 14 member units who employ a similar methodology.

The APSU conducts national active surveillance of rare diseases of childhood, including infectious and vaccine preventable diseases, genetic disorders, childhood injuries and mental health conditions. Surveillance through the APSU provides the only available method of national data collection for most of the childhood conditions studied.

The primary aim of the APSU is to document the epidemiology of the conditions under surveillance, their clinical features, current management and short-term outcome. The APSU's secondary aims are to provide a mechanism for national collaborative research and to disseminate data acquired by the Unit to inform best practice, appropriate prevention strategies and optimal health resource allocation.

Contributors to the APSU are clinicians known to be working in paediatrics and child health in Australia. In 2002 over 1,050 clinicians participated in the monthly surveillance of 14 conditions, with an overall response rate of 96 per cent.

As 100 per cent case ascertainment is unlikely to be achieved by any one surveillance scheme, rates reported below represent estimates of minimum incidence in the relevant population. Where available, additional data sources are used to supplement or verify case finding through the APSU. For further information please contact the APSU on telephone: 02 9845 2200 or email: apsu@chw.edu.au

The Table shows the confirmed cases of communicable diseases reported to the APSU between 1 January and 30 June 2003.

Acute flaccid paralysis

Heath Kelly, Bruce Thorley, Kerri Anne Brussen, Jayne Antony, Elizabeth Elliott, Anne Morris

Acute flaccid paralysis (AFP) surveillance in children under 15 years of age was initiated in 1995 to help meet the World Health Organization certification standards for poliomyelitis eradication. To the end of 2002 there were 262 confirmed cases of non-polio AFP. Based on these data, the reported incidence

Table. Confirmed cases of communicable diseases reported to the Australian Paediatric Surveillance Unit between 1 January and 30 June 2003*

Condition	Previous reporting period Jan–Dec 2002	Current reporting period Jan–Jun 2003*
Acute flaccid paralysis	30	14
Congenital cytomegalovirus		
confirmed (< 3 weeks of age)	9	6
suspected (3–52 weeks of age)	8	3
Congenital rubella	3†	2
Perinatal exposure to HIV	25	8
Neonatal herpes simplex virus infection	11	4
Hepatitis C virus infection	commenced 2003	6

* Surveillance data are provisional and subject to revision.

† Two imported cases i.e. children born to mothers who had rubella in Indonesia. One child was born in Indonesia, one child born in Australia. A third infant was born in Victoria in 2001, but was not notified to the APSU until 2002. The parents were Fijian, it is not known where the mother acquired her infection.

of non-polio AFP is 0.86 (95% CI 0.76– 0.97) per 100,000 children under 15 years. In 2002, the reporting of AFP was down on the preceding year with non-polio AFP 0.75 (95% CI 0.51– 1.08) per 100,000. As noted previously, Guillain-Barré syndrome was the most common cause of AFP (27% of confirmed cases), followed by transverse myelitis (17%) and trauma (13%).

Congenital cytomegalovirus infection

William Rawlinson, Daniel Trincado, Gillian Scott, Sian Munro, Pamela Palasanthiran, Mark Ferson, David Smith, Geoff Higgins, Michael Catton, Alistair McGregor, Dominic Dwyer, Alisson Kesson

Congenital cytomegalovirus infection (CMV) surveillance in children up to 12 months of age commenced through the APSU in 1999. Between January 1999 and December 2001 there were 25 confirmed cases of CMV, that is with CMV being isolated in blood, urine, saliva or tissue in the first three weeks of life. The estimated incidence of congenital CMV is 2.61 (95% CI 1.71– 3.83) per 100,000 live births. An additional eight cases of suspected CMV infection, in which the diagnosis was made between 3 weeks and 12 months of age, were identified in 2002.

Congenital rubella

Margaret Burgess, Jill Forrest, Cheryl Anne Jones, Peter McIntyre

Surveillance of newly diagnosed congenital rubella in children and adolescents under 16 commenced in 1993. Forty-five children with congenital rubella were identified through the APSU between May 1993 and December 2002. Twenty-nine of these children were born in Australia and 22 of these infants had defects attributable to congenital rubella. Several of these children had mothers who were born overseas and were not vaccinated. The estimated incidence of congenital rubella in children born in Australia is 1.20 (95% CI 0.80 –1.73) per 100,000 live births. The incidence of congenital rubella with defects is estimated to be 0.91 (95% CI 0.57 –1.38) per 100,000 live births. There have been two recent reports of congenital rubella infection in children born to Australian-born mothers in Queensland in 2003. These are the first such cases reported since 1999.[1]

HIV infection, AIDS and perinatal exposure to HIV

Ann McDonald, John Kaldor, Michelle Good, John Ziegler

This study monitors new cases of HIV/AIDS infection in children under 16 years and perinatal exposure to HIV. Perinatal exposure to HIV is now the most frequently reported source of HIV infection in Australian children. Between January 1997 and December 2002, 122 children with perinatal exposure to HIV were reported through the APSU and/or the National HIV/AIDS surveillance program. The estimated incidence of perinatal HIV exposure is 8.16 (95% CI 6.78 – 9.75) per 100,000 live births. HIV transmission during the perinatal period may be reduced from 25 per cent to less than two per cent among women whose HIV infection is diagnosed prior to delivery through the use of antiretroviral therapy, elective caesarean delivery and the avoidance of breast feeding.

Neonatal herpes simplex virus infection

Cheryl Anne Jones, David Isaacs, Peter McIntyre, Tony Cunningham, Suzanne Garland

Surveillance of herpes simplex virus (HSV) infection in children aged up to 28 days commenced in 1997. There were 54 confirmed cases of neonatal HSV infection in infants up to 28 days of age between January 1997 and December 2002. The estimated incidence is 3.61 (95% CI 2.71– 4.71) per 100,000 live births. Herpes simplex type 1 remains the predominant isolate causing neonatal disease in Australia.

Hepatitis C virus infection

John Kaldor, Cheryl Anne Jones, Elizabeth Elliott, Winita Hardikar, Alisson Kesson, Susan Polis, Catherine Mews

Surveillance of hepatitis C infection in children commenced in January 2003. APSU contributors are asked to report any child less than 15 years of age with:

- at least one confirmed positive anti-HCV antibody test performed at age greater than or equal to 18 months OR;
- a positive anti-HCV antibody test on a single occasion AND a positive test for HCV RNA (PCR or RT-PCR) on single occasion at any age greater than 1 month of age OR;
- a positive HCV RNA test (PCR or RT-PCR) on two separate occasions.

Six cases of hepatitis C virus infection were confirmed between January and June 2003.

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Prevention of opportunistic infections in immunosuppressed patients in the tropical Top End of the Northern Territory

Joshua S Davis,[1] Bart J Currie,[1,2,3] Dale A Fisher,[1] Sarah E Huffam,[1] Nicholas M Anstey,[1,2,3] Richard N Price,[1,2] Vicki L Krause,[4] Nathan Zweck,[4] Paul D Lawton,[1] Paul L Snelling,[1] Sid Selva-nayagam[1]

Abstract

The population of the Top End of the Northern Territory has a high incidence of several infections of particular significance in the immunosuppressed. The following protocol for evaluation and treatment of patients prior to immunosuppression was developed in order to reduce the incidence of serious opportunistic infections. The infections discussed are *Strongyloides stercoralis*, tuberculosis, scabies, chronic hepatitis B, melioidosis and other bacterial infections. We recommend that all patients planned to receive more than 0.5mg/kg/day of prednisolone for >14 days, or any more potent immunosuppressive drug, be evaluated and treated according to this protocol. Details of the rationale, evidence base, and proposed investigations and therapy for such patients are discussed. *Commun Dis Intell* 2003;27:526–532.

Keywords: immunosuppression, Strongyloides stercoralis*, tuberculosis, scabies, chronic hepatitis B, melioidosis*

Introduction

The Top End is unique, both in the infectious agents that are endemic there and in its population. This guideline has been developed with the people, geography and microbial ecology of the Top End in mind. It may also be useful for other parts of tropical Australia. It aims to supplement existing protocols and practices for defined patient groups, such as organ transplant recipients and chemotherapy patients. It also aims to draw attention to patients who are being immunosuppressed, but for whom no protocol exists regarding prevention of opportunistic infections.

This article focuses on recommendations for the prevention of disseminated strongyloidiasis (DS), tuberculosis, melioidosis and other bacterial sepsis, scabies hyperinfestation and activation of hepatitis B virus infection, which anecdotally have each presented as opportunistic complications in immunosuppressed patients in the Northern Territory over recent years. These conditions are generally more common in the Top End than elsewhere in Australia. The recommendations are justified by varying levels of evidence and represent a consensus guideline developed by local infectious diseases, renal, oncology and public health physicians.

This guideline is only intended to apply to people receiving significant immunosuppression. This is defined in Box 1.[1,2,3]

It does not include the following patients: inhaled corticosteroids, hydroxychloroquine, sulfasalazine, colchicine, gold, weekly methotrexate and oral courses of prednisone less than 14 days regardless of dose

Box 1. Eligible patients for the opportunistic infection prevention protocol

1. Anyone receiving ≥0.5mg/kg per day of prednisolone or the equivalent for >14 days.
2. Anyone currently receiving cyclosporin, cyclophosphamide, azathioprine, mycophenolate, tacrolimus or cancer chemotherapy.

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and where the frequency is less than six courses per year. Patients having ≥6 courses of prednisolone per year may also benefit from the protocol.

With systemically administered steroids, the risk of infection is related to the dose of steroid and the duration of therapy as demonstrated in a meta-analysis of 71 controlled trials.[3] The overall rate of infectious complications was 12.7 per cent in steroid-treated patients compared to 8.0 per cent in the control group (relative risk 1.6). The rate of infection was not increased in patients receiving less than 10 mg per day or a cumulative dose of less than 700 mg of prednisone (which is roughly equivalent to 0.5mg/kg for 20 days). Therefore a cut-off of prednisolone ≥0.5mg/kg/day for >14 days for this protocol is practical. This is supported by there having been to date no confirmed cases of disseminated strongyloidiasis in the Top End in patients on intermittent prednisolone therapy for respiratory and other conditions.

Each infection will be addressed in turn.

Strongyloides stercoralis

Strongyloides infestation is endemic in most remote communities of the Top End of the Northern Territory, particularly in the East Arnhem region. A stool microscopy prevalence study in 1997 at Galawin`ku (Elcho Island, Arnhem Land) showed strongyloides larvae in the stool in 15 per cent of 300 people.[4] In 1991/1992, at least 3.4 per cent (68/2000) of all admissions to Royal Darwin Hospital (RDH) had strongyloides larvae detected in stool.[5] During this 12-month period, another 98 cases of strongyloidiasis were detected in other Top End laboratories. Reliable prevalence data for other Top End communities is not available, but the incidence of symptomatic infection and stool positivity seems significantly lower outside East Arnhem. We encourage collection and analysis of stool prevalence data from Northern Territory communities outside East Arnhem to confirm this impression.

The authors are aware of six cases of disseminated strongyloidiasis (including 1 death) in immunosuppressed patients from the Top End over the last 10 years, with a range of 0–2 cases per year.[6] There may also be undiagnosed cases occurring, as overwhelming sepsis in the absence of a microbiological diagnosis is relatively common at RDH. The reported mortality of DS is up to 87 per cent.[7] Thus, detection and treatment of infestation prior to immunosuppression could avoid potentially fatal DS. However, in immunocompetent individuals with chronic asymptomatic infestation, even the best diagnostic methods may miss the presence of strongyloides. Eosinophilia is usually absent in immunosuppressed people with strongyloides hyperinfestation, although it is often present before immunosuppression begins. In a 1993 RDH survey, peripheral eosinophilia was only present in 57 per cent of immunocompetent patients with asymptomatic infection.[5]

Direct stool examination for strongyloides larvae has a sensitivity of approximately 30 per cent if one stool is examined[8] and 50 per cent for three stools.[9] One study claimed sensitivity of close to 100 per cent if seven stools are examined.[10] This is clearly impractical in our setting.

Culture of stool (as opposed to direct microscopic examination) improves sensitivity, but is labour intensive, and poses a small risk of laboratory-acquired infection. Agar-plate culture of a single stool specimen was approximately 90 per cent sensitive in one study.[8] This takes 2–3 days and may not be practical for screening large numbers of specimens.

The utility of serology varies widely depending on the exact nature of the test used.[7] Using an ELISA method, with crude extract of filariform larvae, improves sensitivity. Performance can be enhanced by pre-incubating the patient's serum with *Onchocerca* antigens to eliminate non-specific cross-reactions before testing. Under the above conditions, the assay was 88 per cent sensitive and >90 per cent specific for the detection of strongyloides infection in a non-endemic setting.[11] Serology may remain positive for months to years after a successfully treated infection, and may cross-react with other helminth infestations, notably *Ascaris lumbricoides*. The titre does not reliably fall with successful eradication.[12] RDH is using an ELISA with *Strongyloides rattii* as the antigen. We do not have good data about the sensitivity and specificity of this technique in our setting, and therefore the above-quoted published rates may not apply.

In summary the sensitivity of current diagnostic methods is not sufficiently high to be able to confidently exclude chronic asymptomatic infestation in a patient who is about to begin immunosuppression.

Disseminated strongyloidiasis can occasionally occur with mild levels of immunosuppression e.g. a 65-year-old man receiving 20 mg per day of prednisolone for severe chronic obstructive pulmonary disease (COPD) for 6 weeks.[1] However, most case reports relating to DS in patients on prednisolone, were with doses around 1 mg/kg/day.[2] There are also multiple case reports suggesting a strong association of disseminated strongyloidiasis with HTLV-I infection, and less so with HIV infection. However, this association has to date not been borne out in Central Australia where HTLV-I infection is endemic.

A recent review recommends empiric treatment (including those with negative serology and stool examinations) before organ transplantation in 'high-risk patients from endemic areas'.[13]

Ivermectin is very well tolerated. Repeated dosing has been used in the Northern Territory for crusted scabies for at least the last five years without any significant toxicity.[14] Large-scale studies with regular six-monthly dosing have been conducted in West Africa, also without significant drug toxicity detected.[15]

For the treatment of simple strongyloides infestation (in immunocompetent patients), a single dose of ivermectin seems equivalent to 2 doses of ivermectin, but is superior to three days of albendazole.[16,17,18] Disseminated strongyloidiasis, where the worm burden is massive, requires multiple doses of ivermectin, and suspected cases of DS should be discussed urgently with an infectious diseases specialist.

The concern for DS applies primarily to patients with solid organ transplants, chemotherapy for malignancy, immunosuppression for SLE and other autoimmune disorders, and patients with severe, steroid dependant COPD/asthma requiring multiple courses of higher dose prednisolone.

The recommended management procedures to prevent disseminated strongyloidiasis are shown in Box 2.

Tuberculosis

A significant proportion of the population of the Top End has latent infection with *Mycobacterium tuberculosis* (LTBI). Immunosuppressive medication greatly increases the chance of reactivation. In patients with HIV co-infection, the chance of developing active TB is approximately 10 per cent per year (compared with 10 per cent over 10–20 years for immunocompetent people).

This risk is similarly increased in patients taking immunosuppressive medications, although this risk is less well defined. Early treatment of LTBI with nine months of isoniazid greatly decreases the chance of reactivation (by around 90%) and should be strongly considered in a person with LTBI who is to begin immunosuppression. A short course alternative is the combination of rifampicin and pyrazinamide for two months, however there may be an unacceptably increased risk of adverse reactions to the medication with this latter regime.

Box 2. Management recommendations to prevent disseminated strongyloidiasis in eligible patients

1. Encourage the wearing of shoes to prevent infection or decrease worm burden through reduced exposure to soil-borne larvae.

2. Before immunosuppression (or at initial evaluation):

– test all eligible patients with serology, eosinophil count and stool microscopy and culture. Treat all patients from highly endemic areas (East Arnhem), regardless of the above results, with a single dose of oral ivermectin 200 mcg/kg. Pregnancy test is first required for all reproductive age women.

For patients with a positive stool microscopy or culture, give a second dose of ivermectin 7 days after the first dose. Repeat stool culture 7 days after the second dose. If still positive, discuss with the Infectious Diseases Unit.

– Outside of East Arnhem, only treat those with evidence of strongyloides infection.

3. With ongoing immunosuppression:

– In East Arnhem, repeat ivermectin every 3 months without investigation. Elsewhere, undertake serology, stool microscopy and culture for strongyloides and eosinophil count every 3 months and treat if positive.

4. Treat any immunosuppressed patient with unexplained pulmonary infiltrates, fever, abdominal pain or septic shock with ivermectin on a day 0,1 and 7,8 regimen.

Box 3 shows the management recommendations to prevent tuberculosis.

Box 3. Management recommendations to prevent tuberculosis in eligible patients

1. Ascertain past history of tuberculosis or latent tuberculosis infection.

– The relevant communicable diseases clinic or chest clinic should be contacted to ascertain if the patient already has a diagnosis of LTBI or partially treated TB. If there is no record of a Mantoux, one should be performed, before starting immunosuppression if possible, as immunosuppression (particularly corticosteroids) will significantly decrease response to the test. If immunosuppression must be commenced immediately, do a Mantoux on day one.

2. Baseline two-step Mantoux testing.

– The cutoff for a positive Mantoux prior to immunosuppression is 10 mm. If immunosuppression already exists, the cutoff is 5 mm.

– If the initial Mantoux result is negative (<10 mm or <5 mm as appropriate), a second Mantoux should be performed 1 to 3 weeks after the first in order to boost a false-negative first result to a true positive value (when a person has LTBI, but has acquired infection many years before, or has anergy to tuberculin).

– A positive Mantoux result on the initial or second test in the absence of active TB (on CXR and at clinical review) will require treatment of LTBI with a 9-month course of isoniazid (plus pyridoxine to decrease neurotoxicity) or a 2-month course of rifampicin and pyrazinamide.

3. Ongoing screening if baseline two-step Mantoux is negative.

– If the baseline two-step Mantoux test is negative, annual Mantoux screening for newly acquired LTBI should occur in those with continuing immunosuppression.

4. If LTBI or past partially treated TB is identified, treat for this as above.

Scabies

Scabies infestation is very common in many Top End communities, and poses a risk of secondary bacterial sepsis. Infected immunosuppressed patients may develop a severe form of scabies, crusted (Norwegian) scabies,[14] therefore it should be treated before immunosuppression. The mortality of crusted scabies in the Top End was up to 50 per cent within five years of diagnosis until recent improvements in scabies treatment and prevention and treatment of secondary sepsis.[19] Scabies infestation can be reliably detected by clinical examination.

Management recommendations to prevent scabies are shown in Box 4.

Box 4. Management recommendations to prevent scabies in eligible patients

1. Treat pyoderma with a single dose of intramuscular benzathine penicillin (Bicillin), 1.2 million units (900 mg).
2. Treat scabies with 5 per cent topical permethrin at days 0 and 7. All household contacts should also be treated.
3. If crusted scabies is present or suspected, hospital admission for eradication of infection should be organised prior to or coincident with the initiation of immunosuppression.

Hepatitis B virus

Chronic hepatitis B virus (HBV) infection is endemic in Top End communities with over 40 per cent of individuals in some communities having evidence of hepatitis B exposure. Endemicity, clinical impact and recommendations for follow up in immunocompetent patients from remote communities has been published. A guideline for follow up and management of these non-immunosuppressed patients from remote communities is in use[20] and should be used for patients on steroids alone, but not for those on more potent immunosuppression (Box 5). In a study at the RDH renal unit of patients undergoing renal replacement therapy, 73 of 122 Indigenous patients (59.8%) had evidence of hepatitis B virus exposure while 10 (8.2%) were HBsAg positive (Dr Nick Gray, Registrar in Renal Medicine, Royal Darwin Hospital, 2001, unpublished).

Box 5. Management recommendations to prevent reactivation of hepatitis B in eligible patients

- Any HBsAg positive patient in whom chemotherapy for cancer or potent immunosuppression is planned should be referred to the liver clinic for assessment, preferably prior to therapy.
- Non-immune patients should be vaccinated against hepatitis B prior to planned immunosuppression.

Rapidly progressive chronic active hepatitis may occur in chronically HBV-infected people who become immunosuppressed.[21] HBsAg positive patients undergoing renal transplantation almost invariably develop significant liver dysfunction with deaths from fulminant hepatic failure documented.[22] Antiviral therapy is of proven benefit in the renal transplantation setting, and it is now standard practice that antiviral therapy be used pre-emptively for patients who are HBsAg positive, irrespective of other markers of hepatitis B viraemia or liver enzyme levels.[23,24] Reactivation of hepatitis B in patients undergoing chemotherapy or potent immunosuppressive therapy has a mortality of 37 to 60 per cent.[25] Pre-emptive antiviral use is also now standard practice in liver transplant recipients, since studies in recent years have provided evidence of its efficacy and safety.[26,27]

For the prevention of reactivation of hepatitis B recommendations in this protocol, potent immunosuppression refers to chemotherapy for malignancy, organ transplantation, or potent therapy for autoimmune disease. This would include cyclophosphamide, azathioprine, cyclosporin, mycophenolate and leflunomide but **not** corticosteroids alone. Assessment will usually include a liver biopsy and initiation of lamivudine and possibly regular hepatitis B immunoglobulin. The other and most newly available antiviral treatment is adefovir dipivoxyl. At this stage, adefovir remains reserved for use in those developing the YMDD (lamivudine resistant) mutation of the reverse transcriptase gene, which occurs commonly e.g. up to 27 per cent of liver transplant recipients on lamivudine at 52 weeks.[26] The use of antiviral medications for hepatitis B is generally restricted to approved liver clinics.

Bacterial sepsis/melioidosis

Melioidosis is more common, more severe and more likely to cause death in people who are relatively immunosuppressed.[28] These data mainly apply to those with diabetes, heavy alcohol intake or chronic renal impairment, all of whom have subtle immune defects including poor neutrophil function. There have been cases of both acute melioidosis and relapsed melioidosis in people on therapeutic immunosuppression,[29] and thus it is probable that all significant therapeutic immunosuppression increases the probability of melioidosis occurring, and of it being more severe if it does occur.

Nocardia infection is uncommon, but well described in immunosuppressed people. Pulmonary and cerebral infections are likely to occur if an immunosuppressed person is infected, and are difficult to treat and have a high mortality.

Skin and systemic sepsis with *Staphylococcus aureus*, Group A streptococcus and *Streptococcus pneumoniae* (among others) are very common in the Top End. Opportunistic infections have been a significant cause of morbidity and mortality in renal transplant patients, with a reported odds ratio for all infectious complications of 30 compared with non-transplant patients.[30] The majority of these infections are with one of the three bacteria mentioned above. Bacterial sepsis with *S. aureus* and *Escherichia coli* were the commonest causes of death in Top End patients with SLE from 1984–90.[6] In these patients, staphylococcal and *E. coli* sepsis were particularly common in the setting of disease exacerbation and a significant increase in steroid dosage with or without other immunosuppressive agents.

A recent review reported, 'The use of low-dose trimethoprim-sulfamethoxazole in organ transplantation markedly reduces the risk of developing *Listeria* infection, *Pneumocystis carinii* pneumonia (PCP), toxoplasmosis, nocardiosis, and urinary tract infections'.[13] It is usual after solid organ transplantation to give prophylactic trimethoprim-sulfamethoxazole for the first 6–12 months post-transplant. Its efficacy has been confirmed in renal transplant patients in a randomised controlled trial which found a highly significant decrease in all bacterial infections, but not in PCP.[31] In our setting, we recommend continuing the prophylactic trimethoprim-sulfamethoxazole as long as potent immunosuppression continues, provided no attributable significant and unavoidable adverse medication reactions occur.

Prophylactic antibiotics are usually not needed with steroids alone. The exception to this rule is patients on particularly high doses (100 mg per day or more of prednisolone or equivalent). In the study on SLE in the Top End quoted previously, when steroid dose was intensified, the incidence of serious opportunistic infections increased significantly, so trimethoprim-sulfamethoxazole prophylaxis may be justified in this subgroup. This is for PCP prophylaxis, and also will decrease bacterial infections (including those from *Nocardia*, staphylococci, streptococci and gram negatives, including *Burkholderia pseudomallei*). The recommended dose is trimethoprim-sulfamethoxazole one double strength tablet daily (160 mg/800 mg).

Varicella zoster virus (VZV) vaccination prior to heavy immunosuppression[13] should be given prior to organ transplantation if the patient is not already immune, as recommended by the Infectious Diseases Society of America.[13] As it is a live vaccine, it should NOT be given to those who are already immunosuppressed.

Management recommendations to prevent melioidosis and bacterial sepsis are shown in Box 6.

Box 6. Management recommendations to prevent melioidosis and bacterial sepsis in eligible patients

Pneumococcal vaccination (23-valent pneumococcal polysaccharide) should be given, and other adult vaccinations made up to date, **before** planned immunosuppression (VZV, MMR, ADT, polio). Pneumococcal vaccination should be repeated every 5 years with ongoing immunosuppression.

– Prophylactic trimethoprim/sulfamethoxazole one double strength tablet (160 mg/800 mg) daily should be given to all patients receiving potent immunosuppression (as defined in Box 5 above) plus those on 100 mg per day or more of prednisolone or equivalent.

– In the wet season, patients should be encouraged to wear gardening gloves and footwear when coming into contact with mud or soil.

– Melioidosis serology should be performed on all patients in the Top End prior to immunosuppression. If positive (an indirect haemagglutination titre of ≥1:40), swabs for melioidosis culture should be taken from throat, rectum and any wounds. Urine and sputum (if any) should also be collected for melioidosis culture. If cultures are positive, full treatment is required (refer to Infectious Diseases Unit).

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A resurgence of congenital rubella in Australia?

Jill M Forrest,[1] Margaret Burgess,[2,3] Tim Donovan[4,5]

Abstract

Two infants with congenital rubella defects (congenital rubella syndrome) have been reported from Queensland in 2003, after an increase in rubella in that State in 2001–2002. The national Measles Control Campaign in 1998 aimed to give measles-mumps-rubella (MMR) vaccine to all unvaccinated preschoolers and a second dose to primary schoolchildren. Following the Campaign no children with congenital rubella defects were born to Australian-born mothers during the five years 1998 to 2002, according to reports to the Australian Paediatric Surveillance Unit. However, three imported cases occurred. Broad immunisation coverage and detection and vaccination of susceptible women of child-bearing age before they become pregnant are necessary to prevent further cases. *Commun Dis Intell* 2003;27:533–535.

Keywords: congenital rubella, Australia, rubella vaccination

Introduction

The 1998 Measles Control Campaign (MCC) raised the level of immunity to rubella as well as to measles, because measles-mumps-rubella (MMR) vaccine was used. Since the vaccine was given to both boys and girls, the large pool of rubella-susceptible male adolescents, a feature of the previous strategy of vaccinating teenage schoolgirls but not their male counterparts, is now reduced.[1]

The number of cases of congenital rubella infection in Australia, ascertained through the Australian Paediatric Surveillance Unit (APSU), a national network of over 1,000 paediatricians,[2] had been falling since 1995 (Table). After the MCC there was only one case in 1999 (mother infected late in pregnancy, no rubella defects in infant). For the next three years there were no local cases (infants whose mothers were infected in Australia) reported. There were two imported cases in 2001 and another in 2002, when mothers born overseas and infected there gave birth to affected children in Australia. It seemed that locally acquired infection causing congenital rubella syndrome was a thing of the past in Australia[3] until two infants were born with congenital rubella defects in Brisbane in 2003 (Figure), following an upsurge in rubella infection in Queensland in 2001–2002 (see accompanying report).

Figure. Congenital rubella syndrome in Australia, 1993 to 2003

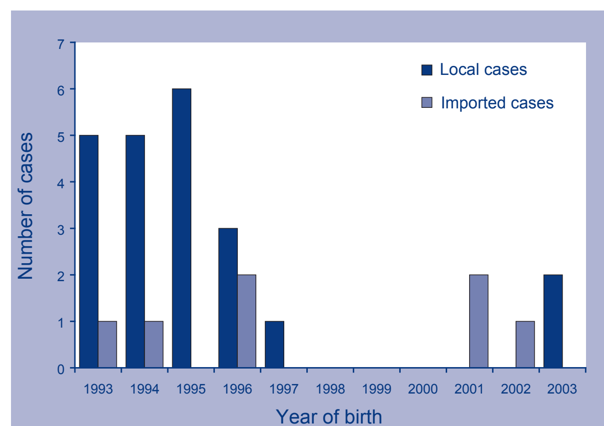

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Table. Cases of congenital rubella infection born between 1993 and 2003 and notified to the Australian Paediatric Surveillance Unit

Year of birth	Local cases (mother infected in Australia)					Imported cases§ (place child born)
	Total	With rubella defects	No rubella defects (week infected)	Mother		
				Vaccinated	Known to be born overseas*	
1993	6	5	1 (34)	3	1	1 (Kenya)
1994	7	5	2 (19, 27)	1	1	1 (Nauru)
1995	7	6	1 (10)	1	4	0
1996	4	3	1 (6†)	2	0	2 (NSW, Fiji)
1997	1	1		0	0	
1998	–	–		–	–	
1999	1	0	1 (35)	0	0	
2000	–	–		–	–	
2001	–	–		–	–	2 (Indonesia, Vic)
2002	–	–		–	–	1 (WA)
2003	2	2		0	0	
Total	28	22 (4 deaths)‡	6	7	6	7 (1 death)

* Not always known whether mother born overseas, if her nationality not Australian.

† Contact only, no clinical rubella in mother.

‡ 1 additional infant born in 1994 died from sudden infant death syndrome.

§ Infected outside Australia and mother born outside Australia.

Case reports

Case 1

In April 2003 an 18-year-old Caucasian Australian-born mother delivered her second child at 38 weeks' gestation. This male infant was small for gestational age (birth weight 2,220 grams) and had a head circumference on the 10th percentile (32.2 cm). Postnatally, his rubella IgM was positive and he had neonatal thrombocytopenia requiring platelet transfusion. Later, brainstem evoked responses indicated severe bilateral sensorineural deafness. This mother had a rubella contact (an adolescent female) at 9 weeks' gestation, and serologically confirmed rubella with rash at 10 weeks. She had missed the rubella schoolgirl vaccination program because of illness.

Case 2

In May 2003 a 21-year-old Caucasian Australian-born mother delivered her first child, a son, whose birth weight was 3,570 grams at 39 weeks' gestation. He had thrombocytopenia, cerebral calcification and ventriculomegaly, and severe bilateral sensorineural deafness. The mother had a rubella contact at her workplace at 9 weeks' gestation, followed one week later by a serologically confirmed rubella-like illness without rash. It is not known whether she was vaccinated against rubella at school.

Notifications of congenital rubella to the Australian Paediatric Surveillance Unit

Since January 1993, 35 infants born with definite congenital rubella infection have been notified to the APSU (Table). Of these, 29 infants had rubella defects and six were infected but without defects at the time of notification. Twenty-eight of the 35 mothers were infected in Australia. Of these 28, 15 infants were born in New South Wales, eight in Queensland, three in Victoria and two in the Australian Capital Territory. The other seven cases were imported. Although the mothers came from and were infected in Kenya, Nauru, Mauritius, Fiji (2) and Indonesia (2), three of the seven infants were born in Australia (New South Wales, Victoria and Western Australia).

Of the 28 local cases, nine mothers had nationalities other than Australian, and six were known to have been born overseas. When the seven imported cases are included, a total of 16 of the 35 mothers (46%) had a nationality other than Australian, and 11 of the 16 came from countries without rubella vaccination programs.

One-quarter (7 of 28) of the local cases gave a history of rubella vaccination in the past.

Discussion

How did these two young mothers fail to receive full and effective rubella vaccination? One mother had missed the schoolgirl rubella vaccination because she was sick. Perhaps there is a need to follow-up girls who miss their adolescent rubella vaccination. There was no documentation of rubella vaccination for the second mother. Both mothers appear to have been exposed to rubella through infected adults. Rubella in Australia is predominantly a disease occurring in young adults, particularly men, who were not eligible for an adolescent dose of rubella vaccine. This cohort of predominantly non-immune males will continue to provide an exposure risk to pregnant women who have missed vaccination for rubella. Immigrants also contribute to this risk as in 2002 only 124 (58%) of the 214 countries reporting to the World Health Organization had introduced rubella vaccination into their national immunisation programs.[4] Therefore helping Australia's neighbouring countries establish rubella vaccination programs could reduce congenital rubella and also the number of our imported cases.

A recent report from the Mercy Hospital for Women, Melbourne, has shown that since rubella immunisation commenced in Australia in 1971, there has been a significant reduction in rubella seronegativity in women of child-bearing age in Melbourne from around nine per cent in 1976 to three per cent in 1995. However, susceptibility has remained at this level since, due to high rates of seronegativity in women born in developing countries.[5] This finding further emphasises the need for vaccination programs targeting immigrants to Australia.[6]

There is no room for complacency in Australia. The MCC was a great success; however, the subsequent offer of MMR vaccination to young adults awaits evaluation, but is thought not to have reached many of the targeted under 30-year-olds. Cuba was able to eliminate rubella and congenital rubella syndrome using two mass vaccination campaigns targeting women aged 18–30 years in 1985 and children aged 1–14 years in 1986. The last case of congenital rubella syndrome was reported in 1989, and the last rubella case in 1995.[7] Finland reported elimination of indigenous measles and rubella in 2000.[8] In 1999 the United States of America reported that congenital rubella syndrome was near elimination with an average of six cases annually of whom 42 per cent were imported and a high proportion of the locally acquired infections were in foreign-born mothers.[9] In the United Kingdom, where there is an active surveillance system comparable to our own, using the British Paediatric Surveillance Unit, a small number of locally acquired cases continue to occur with a similar proportion of imported cases and foreign-born mothers, to Australia and the United States of America.[10,11]

The birth of a baby with congenital rubella is both a personal and a community tragedy. We must continue to immunise children, to identify and immunise vaccine failures and other susceptible women before they become pregnant, and to screen pregnant women so they can be vaccinated after delivery. Effective surveillance of rubella and congenital rubella syndrome is also needed. Continued vigilance will be the price of freedom from congenital rubella.

Acknowledgements

We gratefully acknowledge the contribution of the APSU and the Australian doctors who report to it, without whom enhanced surveillance of congenital rubella in Australia would not be possible. The National Centre for Immunisation Research and Surveillance is supported by the Commonwealth Department of Health and Ageing, the NSW Department of Health and the Children's Hospital at Westmead.

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Festschrift for Professor Margaret Burgess AO

5-6 February 2004

A two-day program has been organised in honour of Professor Margaret Burgess' retirement. Guest speakers include Professor Felicity Cutts (UK) and Professor Stanley Plotkin (USA). Topics covered include 'Vaccines for the 21st Century' and 'Congenital and neonatal infections'.

Venue

The Children's Hospital, Westmead, NSW, Australia and The Children's Medical Research Institute, Westmead, NSW Australia.

Registration fee $55 GST inclusive covers lunch, morning and afternoon breaks.

Further enquiries

The program and registration form are available from the NCIRS Website at: http://www.ncirs.usyd.edu.au/ in 'publications'.

Alternatively contact Jan Michniewicz, National Centre for Immunisation Research & Surveillance.

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RSVP: 31 December 2003

Rubella in Australia: can we explain two recent cases of congenital rubella syndrome?

Heather F Gidding,[1,2] Margaret Young,[3] Robyn Pugh,[4] Margaret Burgess[5,6]

Introduction

Vaccination has lead to a considerable reduction in rubella and congenital rubella syndrome (CRS) in Australia. In 2002, national notification rates for rubella were the lowest on record and there were no notified cases of CRS between 1997 and 2002—in stark contrast to the pre-vaccination era when there were an average of 120 cases of CRS reported annually. It is a concern therefore that two cases of CRS have been reported from Queensland in 2003.[1] To investigate possible reasons why these two cases occurred, we reviewed recent surveillance data about rubella incidence, immunity and vaccination coverage.

Methods

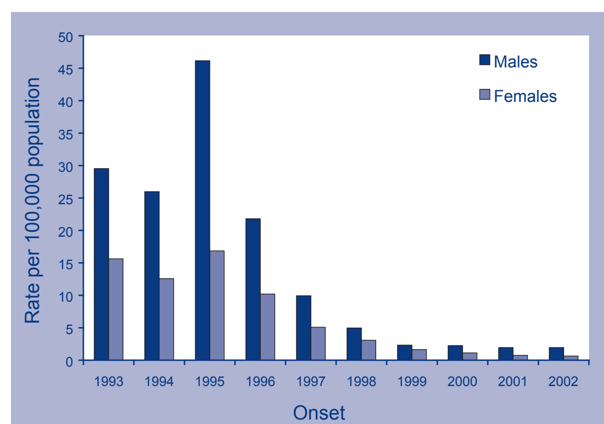
We reviewed notifications of rubella available from the National Notifiable Diseases Surveillance System and the Queensland Notifiable Conditions System as of 24 September 2003. Coverage with the first and second dose of the measles-mumps-rubella (MMR) vaccine due at one and four years of age was reviewed using data from the Australian Childhood Immunisation Register (ACIR). We used the first national serosurvey results to estimate levels of immunity to rubella for persons aged 1–59 years, women of child-bearing age (aged 15–45 years in 2003) and the corresponding cohort of males.[2] To obtain the most recent estimates of immunity we used sera collected in 1999 for 1–18 year olds and sera collected in 1996–1998 for 19–59 year olds. For each of the analyses we identified recent trends and sought any significant differences between Queensland and the rest of Australia.

Results

Rubella notifications

Nationally, the rubella notification rate for both males and females has been declining since 1995 (Figure 1) with preliminary data for 2003 indicating that this trend is continuing. All jurisdictions have shown dramatic rate reductions following outbreaks in the early part of the 1990s, and this downward trend has continued in all states and territories except Queensland in 2000–2002 (Figure 2).

Figure 1. Rubella notification rates, Australia, 1993 to 2002, by sex and year of onset



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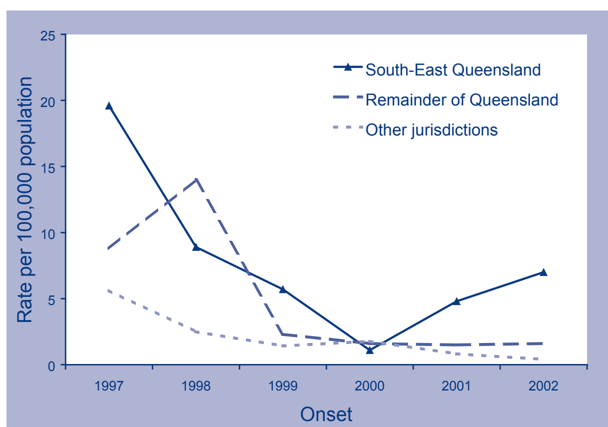
In 2000, Queensland had their lowest notification rates for rubella on record and even had lower rates than other jurisdictions (Figure 2). This is in contrast to most years prior to 2000, when Queensland had the highest notification rates of any jurisdiction. Following the record low rates in 2000, a sustained increase in rubella notifications occurred in Queensland from mid-2001 to late 2002, such that in 2002 (when the two mothers of the CRS cases would have been infected) 75 per cent of Australia's notifications were from Queensland. Most of the increase was in the Moreton and Brisbane Statistical Divisions, which form the south-east corner of Queensland (Figure 2). These two divisions combined accounted for 89 per cent (170/190) of Queensland's rubella notifications in 2002. Since 2002 however, notifications in Queensland have declined, with only 27 cases notified to the end of June 2003.

Figure 2. Rubella notification rates, Australia, 1997 to 2002, by jurisdiction and year of onset

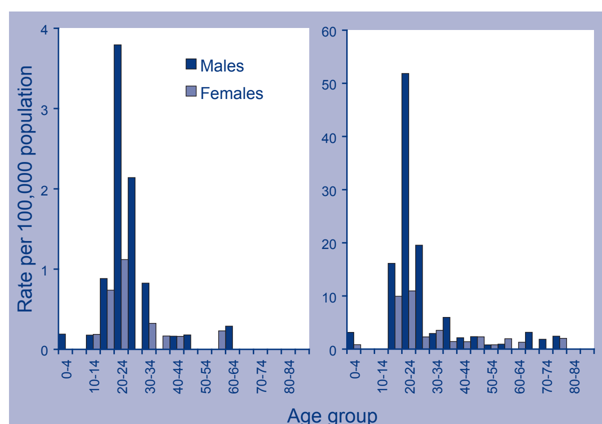


Although Queensland accounted for most of the notified cases of rubella in 2002, the age/sex distribution was the same as elsewhere in Australia (Figure 3). Rates were highest in males aged 15–29 years and females aged 15–24 years. Children aged under 15 years had low rates. Since the Measles Control Campaign (MCC),[3] the proportion of reported rubella cases in young adults has increased across Australia, resulting in an increase in the median age of notified rubella infections for both males and females (Figure 4).

Vaccination coverage

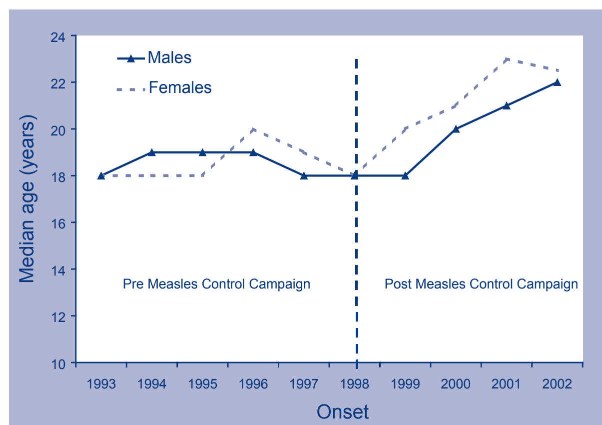
Vaccination coverage data from the ACIR by jurisdiction indicates that levels for Queensland for both the first and second dose of MMR vaccine have always been above or similar to the average for Australia as a whole. According to the ACIR, Queensland achieved 90 per cent coverage with MMR at 24 months in 1998 and coverage rates for this milestone now approach 95 per cent across the State. As with other jurisdictions, coverage for the second dose at 72 months increased steadily after changes to the childhood schedule in 1998 and now approaches 85 per cent. Statistical Divisions with slightly lower than average childhood vaccination coverage do not correspond with those that have had high rubella notification rates.

Figure 3. Rubella notification rates, Australia, 2002, by jurisdiction (a: jurisdictions other than Queensland; b: Queensland), and age group and sex*



* Note differing scales on x axis.

Figure 4. Median age of notified rubella cases, Australia, 1993 to 2002, by sex and year of onset



Immunity in the first serosurvey

Using the most recently available results from the first serosurvey, immunity for 1–59 year olds in Queensland (90.3%) was similar to, but slightly lower than, the national average (91.8%). A similar pattern was seen for women of child bearing age (Queensland 92.8%, Australia 95.4%). However, immunity for these groups at a population level in both Queensland and Australia is above that thought to be required for herd immunity (82–87%).[4] In contrast, men aged 15–45 years were found to have much lower levels of immunity (Queensland 85.2%, Australia 87.2%). Such levels may be insufficient to prevent ongoing transmission in young adult males. Unfortunately the Young Adult MMR Campaign conducted in 2001[5] is unlikely to have significantly improved immunity levels; in Queensland, it is estimated that only about five per cent of the eligible 18 to 30 year old cohort received a dose of MMR vaccine (K Peterson, State Immunisation Coordinator, Queensland Health, personal communication).

Discussion

The epidemiology of rubella has changed since the MCC was conducted in 1998.[3] Notification rates for children aged less than 15 years have declined dramatically due to firstly, the mass vaccination of primary school aged children as part of the MCC; secondly, lowering of the age for the second dose of MMR from age 10–16 years to age 4–5 years (and later 4 years); and finally, continued improvement in coverage with the first dose of MMR vaccine. This has resulted in the lowest overall notification rate on record for Australia in 2002.

Improved immunity in children and lower rates overall have led to an increase in the median age of rubella infection. This is of concern because it puts susceptible young females of child-bearing age at increased risk of infection. In addition, there remains a cohort of susceptible young adult males who have missed being vaccinated as part of previous young adult or schoolgirl only programs and are too old to have been eligible for vaccination as infants. As the serosurvey results show, this cohort probably has insufficient immunity to prevent ongoing transmission and can therefore act as a reservoir to infect susceptible women of child-bearing age. Both the increased median age of infection and ongoing circulation of rubella in young adult males helps to explain why cases of CRS can still occur despite record low rates overall.

What is more difficult to explain is why increased notification rates of rubella and reported cases of CRS are confined to Queensland, and south-east Queensland in particular. National serosurveillance and childhood vaccination coverage data indicate that Queensland has similar levels of immunity to other jurisdictions. However, the serosurveillance data do indicate there is sub-optimal immunity in young adult males and that a proportion of women of child-bearing age are still susceptible. It would appear that the population of susceptible adults in the densely populated and rapidly expanding areas of south-east Queensland is large enough to sustain transmission of rubella. This may not be the case in less densely populated areas which were found to have similar levels of immunity to South East Queensland.

Another possible explanation is that Queensland, especially South East Queensland, has more complete rubella and CRS surveillance data than other jurisdictions due to increased awareness and reporting of these diseases. Since national surveillance began, Queensland has usually had the highest annual notification rate of all states and territories and has reported proportionally more cases of CRS than any other jurisdiction except New South Wales.[1] In addition, some Public Health Units in south-east Queensland alerted general practitioners and other clinicians in their area to increases in rubella notifications in early 2002. However, better surveillance alone is unlikely to explain such a large difference in notification rates.

Even if we don't fully understand the reasons why rubella notification rates increased in Queensland during 2001 and 2002, the notification of two cases of CRS serves as a warning for the whole of Australia. We need to maintain high coverage with the first dose of MMR vaccine and improve uptake of the second dose. Given rubella transmission is still occurring amongst young adults, maintenance of programs to detect and vaccinate non-immune females of child-bearing age is essential, through screening of females planning pregnancy and through antenatal/postnatal programs. High quality surveillance data are also required to determine whether immunity is sufficient to prevent further cases of rubella and CRS. If rubella continues to circulate in young adults, we may need to consider another adult vaccination program using mass vaccination strategies such as those successfully employed in the Americas.[6]

Acknowledgements

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Identification of *Photorhabdus asymbiotica* in cases of human infection

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Photorhabdus asymbiotica is a potential cause of severe soft tissue and systemic infection in Australia. The clinical and laboratory features have been described in a recent publication.[1] Recognition of this unusual pathogen presents a challenge for clinical microbiology laboratories. It is a bioluminescent gram-negative bacillus and is a member of the *Enterobacteriaceae*. It produces a thin zone of annular haemolysis on trypt ic soy agar containing either 5 per cent sheep or horse blood and tends to swarm.

Some isolates produce a yellow pigment and all are faintly luminescent in total darkness. This species is not yet included in the databases of commercial bacterial identification systems. Use of the MicroScan Walkaway (Dade Behring Inc., Sacramento, CA), Vitek (bioMérieux, Hazlewood, MO) or API 20E (bioMérieux, Marcy l'Etoile, France) will result in incorrect identification. The results obtained with these three systems for six Australian isolates of *P. asymbiotica* are shown in the Table.

It is possible that infection due to this species is under-reported due to incorrect laboratory identification. Knowledge of the epidemiology of infection due to *P. asymbiotica* is incomplete. For example, it has only ever been isolated from clinical specimens and no reservoir or source has been identified. We wish to raise awareness of this infection among clinicians and laboratory workers in the hope of improving case identification. We would be grateful to receive information and isolates from other cases of this condition.

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Table. Misidentification of Australian isolates of *P. asymbiotica* by commercial Vitek GNI, Microscan Walkaway Neg BP combo 11, Microscan Walkaway Rap Neg BP 5A and API 20E panels

Year	Location	Vitek GNI		Microscan Walkaway Neg BP combo 11		Microscan Walkaway Rap Neg BP 5A		API 20E	
		Bionumber	Misidentification	Bionumber	Misidentification	Bionumber	Misidentification	Bionumber	Misidentification
1998	Melbourne	6022100020	Unidentified	0004060-0	*P. oryzihabitans*	011377142202-000	*S. putrefaciens*	000000045	Non fermenter spp.
1998	Wangaratta	6002000020	*Providencia stuartii*	0000060-0	*P. oryzihabitans*	011377162222-400	*P. aeruginosa*	000000045	Non fermenter spp.
1998	Murwillumbah	6002100020	*Shigella dysenteriae*	0000064-0	*A. lwoffii*	011377142222-400	*S.putrefaciens*	000000045	Non fermenter spp.
1999	Gold Coast	60221000000	*Flavobacterium* spp.	0000060-0	*P. oryzihabitans*	011377102202-000	*S. putrefaciens*	000000045	Non fermenter spp.
2001	Gladstone	60201000000	*Flavobacterium* spp.	4000000-2	*Shigella* sp.	011377100002-000	*S. putrefaciens*	000000045	Non fermenter spp.
2003	Beaudesert	60221000000	*Flavobacterium* spp.	0004060-0	*P. oryzihabitans*	011377102202-000	*S. putrefaciens*	000000045	Non fermenter spp.

Surveillance for neuraminidase inhibitor resistance in human influenza viruses from Australia

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Abstract

Two hundred and forty-five human influenza A and B viruses isolated in Australia between 1996 and 2003 were tested for their sensitivity to the NA inhibitor drugs, zanamivir and oseltamivir using a fluorescence-based neuraminidase inhibition assay. Based on mean IC_{50} values, influenza A viruses (with neuraminidase subtypes N1 and N2) were more sensitive to both the NA inhibitors than were influenza B strains. Influenza A viruses with a N1 subtype and influenza B strains both demonstrated a greater sensitivity to zanamivir than to oseltamivir carboxylate, whereas influenza A strains with a N2 subtype were more susceptible to oseltamivir carboxylate. A comparison of IC_{50} values for viruses isolated before and after the release of the NA inhibitors in Australia, found there was no significant difference in the sensitivity of strains to either neuraminidase inhibitor and none of the isolates tested showed clinically significant resistance. *Commun Dis Intell* 2003;27:542–547.

Keywords: influenza, neuraminidase inhibitors, oseltamivir carboxylate, zanamivir, Australia

Introduction

Two new antiviral agents, zanamivir and oseltamivir, have recently become available for the treatment and prophylaxis of influenza.[1,2] These compounds were designed to interfere with the activity of the neuraminidase enzyme of the influenza virus based on knowledge of the three-dimensional crystal structure of the enzyme, determined by Australian scientists, and of the chemistry of its interaction with cell surface receptors.[3]

Neuraminidase plays a crucial role in the release of new virions from the infected cell[1] and the structure of the enzyme active site is highly conserved across the known types of influenza A and B which are responsible for epidemic and, for influenza A, pandemic human disease.[4] Consequently, the neuraminidase inhibitors offer considerable promise as broadly active anti-influenza drugs. The rapid replication of influenza, which may precede onset of symptoms in the infected individual, and the difficulty in making an accurate diagnosis of influenza in time for the compounds to have maximum benefit, have limited their use for the treatment of established infection. Nevertheless, they represent an important addition to the options for prevention and treatment of influenza in situations such as institutional outbreaks, for immunocompromised individuals or where vaccines are unavailable. The NA inhibitors are likely to be of value for the treatment and prevention of influenza in the event of a future pandemic when it is expected that there will be little, if any, vaccine available initially, particularly for prophylaxis of health care workers and others providing essential services.

Influenza viruses have an exceptional capacity for adaptive change both through a high rate of mutation and their ability to undergo genetic reassortment.[5] While this is most commonly seen as antigenic variation, the rapid evolution of resistance to earlier anti-influenza drugs, the ion channel inhibitors amantadine and rimantadine,[6] illustrates the potential that these viruses have to respond to both immunological and other evolutionary pressures. Viruses resistant to the neuraminidase inhibitors can be generated *in vitro* by multiple passage in the presence of the drugs, however many of these strains demonstrate a significantly reduced infectivity in animal models.[7] *In vivo* resistance to zanamivir has only been recorded in a single persistently infected immunodeficient individual,[8] whereas viruses with reduced drug sensitivity have been recorded in 0.4 per cent of paediatric and four per cent of adult patients treated with oseltamivir.[9] To date there is no evidence of transmission and persistence of resistant viruses. Nevertheless, in view of the capricious nature of influenza and experience with development of drug resistance by

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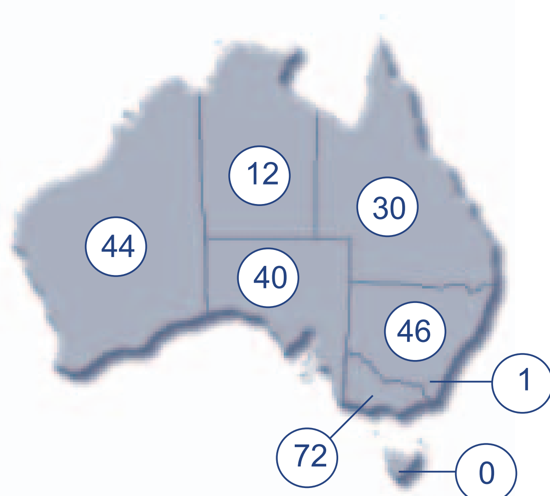
other viruses it seems prudent to maintain an active surveillance for drug sensitivity in clinical isolates. A previous study of strains collected globally between 1996 and 1999 investigated the susceptibility of viruses to the NA inhibitors.[10] However, because oseltamivir had not yet been released in 1999, and zanamivir had only just become available, the study was unable to determine if the sensitivity of influenza viruses had changed significantly as a result of the introduction of these drugs. Here we report the results of a study conducted on Australian influenza viruses isolated from 1996, prior to the release of zanamivir (1999) and oseltamivir (2001), through 2003.

Methods

Influenza isolates

Two hundred and forty-five influenza A and B viruses (Figure 1) were selected from over 4,000 strains collected through the WHO global influenza surveillance program between the years 1996 and 2003, and were tested for their sensitivity to the NA inhibitor drugs zanamivir and oseltamivir carboxylate. The majority of the viruses were isolates from hospital patients, with a smaller number derived from general practitioner surveillance. Where possible, similar numbers of each NA type (B, N1 and N2) were tested annually with similar overall numbers for each year of isolation (1996–2003) and from all geographic regions of Australia (Figure 1). Due to low numbers of influenza A(H1N1) and B viruses circulating in Australia in some years, and the absence of uniform virological surveillance this was not fully achieved. All viruses were cultured in Madin-Darby canine kidney (MDCK) cells at the WHO Collaborating Centre for Reference and Research on Influenza, Melbourne.

Figure 1. Distribution of isolates selected for NA inhibitor drug sensitivity testing



NA inhibitor drugs

The NA inhibitors zanamivir and oseltamivir carboxylate were used in the NA inhibition assays. Zanamivir was used directly from the blister packaging of Relenza (5 mg zanamivir and 20 mg lactose) as distributed through pharmacies. Oseltamivir carboxylate (GS 4071) is the active form of the prodrug oseltamivir phosphate (tradename Tamiflu), and was kindly supplied by Professor Noel Roberts, Roche Products, Welwyn Garden City, United Kingdom.

NA inhibition assay

To determine the sensitivity of the influenza viruses to the NA inhibitor compounds a fluorescence-based NA inhibition assay was used. The assay was based on the release of the fluorescent product 4-methylumbelliferone from the substrate 2-(4-methylumbelliferyl)-a-D-N-acetylneuraminic acid (MUNANA) as a measure of NA activity.[11] The protocol followed a previously published method,[12] except that the inhibitor/virus mix was incubated for 45 minutes rather than 30 minutes, and the MUNANA/inhibitor/virus mix was incubated for 60 minutes at 37° C with no shaking, rather than 15 minutes at 37° C with shaking. Briefly, to determine the drug concentration required to inhibit 50 per cent of the NA activity (IC_{50}), each virus was mixed with a range of concentrations of each drug (0.01 nM to 10,000 nM). Following an incubation period, the fluorescence substrate MUNANA is added to the virus/inhibitor mix and then incubated at 37° C for 60 minutes. After this time the reaction was stopped and the level of NA inhibition at different drug concentrations was quantified and an IC_{50} value for each virus calculated using a logistic curve fit program kindly provided by Dr Trevor Rae, Roche Products, Welwyn Garden City, United Kingdom. One NA inhibitor sensitive and two NA inhibitor resistant control strains were included in each assay for reference purposes. A known susceptible isolate wba-1 A(H1N9), and two known resistant strains xw-2/3 A(H1N9) (E119G mutation) and yn-1 A(H1N9) (R292K mutation) were kindly provided by Jennifer McKimm-Breschkin, CSIRO, Parkville, Australia, and used as controls in each assay for reference purposes.

Statistical analysis

Paired t-tests were used to compare the IC_{50} values of the two NA inhibitors for each virus type. To compare the sensitivity of the three NA types to the two NA inhibitors it was first necessary to adjust for year to year differences in the number of each NA type tested, by using year as a covariate. Each of the three comparisons was then analysed using a Tukey's HSD test. Changes in NA inhibitor sensitivity which can predict clinical resistance have yet to be determined, however it has been proposed that a shift of >10-fold may be an appropriate predictor.[13] In this study a more conservative approach was taken where by viruses with a >5-fold reduction in sensitivity compared to the mean, were considered outliers and investigated further.

Results

Mean IC_{50} values ± standard deviation (S.D.) of both zanamivir and oseltamivir carboxylate for influenza A viruses (grouped by NA type N1 and N2) and influenza B viruses are shown in the Table. Strains from each NA type demonstrated a different level of sensitivity to the two NA inhibitors. Generally, influenza A viruses containing a N1 NA and influenza B viruses were better inhibited by zanamivir than by oseltamivir carboxylate ($p = <0.001$), while influenza A viruses with a N2 NA type were better inhibited by oseltamivir carboxylate than by zanamivir ($p = <0.0001$) (Table).

The NA inhibitors were also shown to differentially inhibit the three NA types to different degrees. Mean IC_{50} values of zanamivir were similar for N2, and B strains, but significantly lower for N1 viruses ($p <0.05$). While the mean IC_{50} values of oseltamivir carboxylate were similar for N1 and N2 strains, but significantly higher for influenza B viruses ($p <0.05$) (Table).

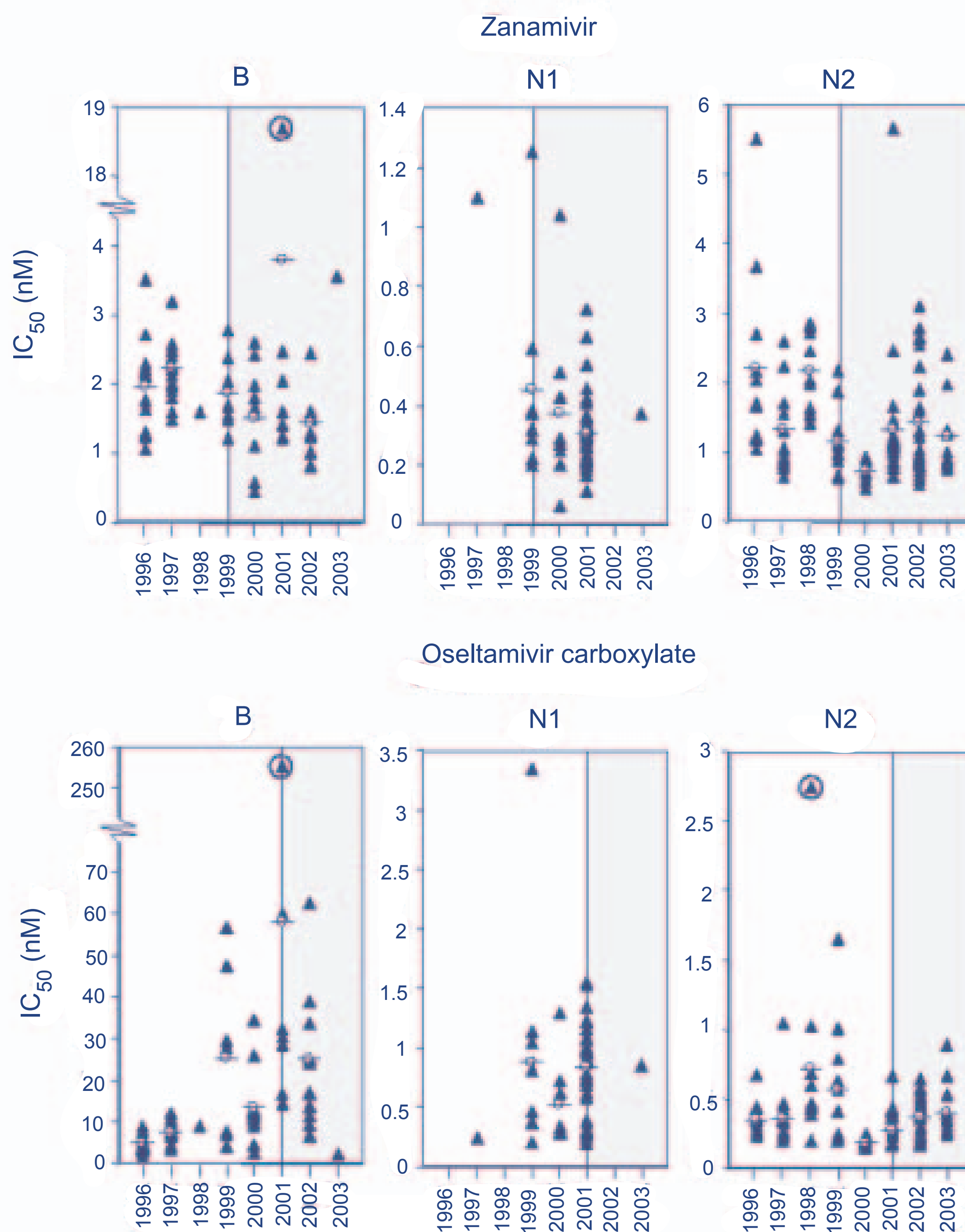
To investigate whether there has been any significant change in NA inhibitor sensitivity since the introduction of the drugs into Australia, IC_{50} values of viruses were compared based on their isolation date. Figure 2 illustrates the individual and mean IC_{50} values of viruses isolated in the years prior to and following the release of the two NA inhibitors. The mean inhibition level of Australian viruses for each drug before its introduction compared with after its introduction was not significantly different for either NA type or NA inhibitor tested ($p = <0.05$).

Of the 245 influenza viruses tested, only two demonstrated a fivefold or greater reduction in sensitivity to either of the NA inhibitors compared with the mean IC_{50} value for the relevant NA type (Figure 2). The first of these viruses was an N2 strain isolated in 1998, prior to the release of either zanamivir or oseltamivir carboxylate. This strain demonstrated an IC_{50} with oseltamivir carboxylate (2.75 nM) that was approximately sevenfold higher than the mean for all N2 viruses with this inhibitor (0.38 nM), although with zanamivir the IC_{50} for the isolate was not significantly different from the mean (1.97 nM vs Mean IC_{50} N2 = 1.39nM). The other Australian virus to demonstrate a reduction in NA inhibitor sensitivity was an influenza B virus isolated in 2001 from an eight-month-old female infant who was not undergoing NA inhibitor treatment. This strain was shown to have an oseltamivir carboxylate IC_{50} value of 256.7 nM, approximately 13-fold greater than the mean IC_{50} for type B strains (19.25 nM), and a zanamivir IC_{50} value of 18.8 nM, approximately 9-fold higher than the mean (2.05 nM). Further passages of this isolate demonstrated an equally reduced sensitivity to the NA inhibitors.

Table. Mean IC_{50} (nM) ± standard deviation of zanamivir and oseltamivir carboxylate for influenza viruses of each NA type

	B n = 65	N1 n = 55	N2 n = 125
Zanamivir	2.05 ± 2.21	0.36 ± 0.22	1.39 ± 0.86
Oseltamivir carboxylate	19.25 ± 33.22	0.81 ± 0.52	0.38 ± 0.30

Figure 2. Scatter plots of individual IC_{50} values (▲) of zanamivir and oseltamivir carboxylate for influenza viruses from each NA type/subtype and for each year of isolation between 1996 and 2003



Mean IC_{50} values (⊖) for each year are included where there were more than one virus tested for that period. Viruses with IC_{50} values that are fivefold or greater than the mean IC_{50} (▲ circled).

Blue shaded area indicates the years following the introduction of the particular NA inhibitor.

Discussion

Due to the rapid evolution of resistance in influenza viruses following treatment with the previous class of anti-influenza drugs the M2 ion channel inhibitors, it was thought necessary, following the release of the NA inhibitors, to monitor influenza isolates for the development of resistance to zanaivir and oseltamivir carboxylate. Here we showed that Australian viruses demonstrated a high level of sensitivity to the two licensed NA inhibitors throughout the study period which were similar to those reported for strains collected globally by the Neuraminidase Inhibitor Susceptibility Network[10] and for viruses from Canada.[14] While the mean oseltamivir carboxylate IC_{50} for type B viruses from this study (19.25 nM) is higher than previously reported for strains collected globally (12.46 nM),[10] much of this difference can be explained by the inclusion of the outlying 2001 type B isolate (oseltamivir carboxylate IC_{50} of 256.7 nM) in our data set. Removal of the IC_{50} value for this virus reduced the overall mean to 15.54 nM.

Zanamivir became available for use in Australia in 1999 followed by oseltamivir in 2001. The overall IC_{50} values of Australian viruses to the two drugs has not changed significantly since their introduction, nor is there evidence of increased numbers of individual viruses with decreased sensitivity to either NA inhibitor. From 245 influenza isolates tested only two Australian viruses were identified in this study as outliers with IC_{50} values showing a 7- to 13-fold reduction in sensitivity to the NA inhibitors. These reductions in sensitivity are very low compared to resistant viruses reported in the literature. Strains isolated from patients following oseltamivir treatment have been reported to demonstrate a fold reduction in sensitivity of between 50 and >80,000 compared to the sensitive wildtype strains.[15] However, many of the viruses that have a high level of resistance to the NA inhibitors have also demonstrated a significant reduction in infectivity in mice and ferret models.[8,16,17] To date only one resistant clinical isolate has been reported following zanamivir treatment, an influenza B isolate from an immunocompromised child.[8] This isolate demonstrated a 100-fold reduction in sensitivity to zanamivir.[18]

For surveillance purposes it is important to define the reduction in sensitivity of a virus that would be expected to result in the clinical failure of the NA inhibitors. Based on the high concentrations of NA inhibitor drug that can be achieved following administration (zanamivir concentrations have been measured above 3,000 nM in sputum six hours post-inhalation)[19] even the least sensitive virus from this study should be fully inhibited by either zanamivir or oseltamivir treatment *in vivo*.

The results of this study and others[10,14] indicate that resistance to the NA inhibitors is uncommon. However, it does remain prudent to continue to conduct sensitivity testing of viruses, particularly if the use of the NA inhibitors increases in the community. For this purpose the NA inhibition method used in this study has been shown to be ideal for the surveillance for NA inhibitor resistant influenza viruses, particularly as it has been reported to be more predictive of *in vivo* susceptibility than cell-based assays.[20] Targeted surveillance directed to the isolation and testing of strains from normal or immuno-compromised individuals undergoing treatment with the NA inhibitors may allow a more focussed and thorough assessment of the potential for influenza viruses to develop clinically significant resistance to these compounds.

Acknowledgements

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The National Immunisation Committee

History

The National Immunisation Committee (NIC) was established as a sub-committee of the Australian Health Ministers Advisory Council (AHMAC) in 1993 to oversee the implementation of the 1993 National Health and Medical Research Council National Immunisation Strategy and to provide advice to AHMAC on immunisation and vaccine preventable disease issues. A realignment of AHMAC sub-committees repositioned the NIC under the auspices of the National Public Health Partnership in early 2000 and a further realignment in 2002 resulted in the NIC reporting to National Public Health Partnership through the Communicable Diseases Network Australia.

The original membership of state, territory and Australian government immunisation program managers has been broadened over the years to include representatives of general practice and Indigenous persons. The common thread among members is their years of experience and a commitment to the development and implementation of sound and sustainable immunisation policies and programs in Australia. The excellent rapport within the group has facilitated the sharing of ideas and local initiatives, and collaborative development of strategies and programs.

Role

The NIC is the peak group responsible for overseeing the development, implementation and delivery of the National Immunisation Program.

The NIC's current terms of reference are:

- provide leadership and take responsibility for policy development, implementation and review of the National Immunisation Program;
- consult and negotiate with stakeholders on the development of national immunisation priorities, strategies and service delivery;
- establish task-oriented, time-limited working groups as required;
- collaborate with other peak immunisation related committees including the Australian Technical Advisory Group on Immunisation, the Communicable Diseases Network Australia, and the Australian Childhood Immunisation Register Management Committee, on issues pertaining to immunisation policy development and program implementation;
- promote collaboration between local, state, national and international organisations to inform national immunisation policy; and
- report to the AHMAC through the Communicable Diseases Network Australia, and National Public Health Partnership on immunisation policies and programs.

Membership

Current members of the NIC include representatives of:

- Australian government;
- state and territory governments;
- general practitioners; and
- an Aboriginal and Torres Strait Islander representative.

Mr Greg Sam has been the long term Chair of the NIC, having taken over the position in 1997. Following his recent departure from the Australian Government Department of Health and Ageing, Ms Lesley Podesta, also from the Department of Health and Ageing, has taken over the role of Chair of the NIC. The Deputy Chair of the NIC is Dr Rosemary Lester from the Victorian Department of Human Services.

The National Immunisation Program (or Immunise Australia Program) is a joint program between the Australian and state and territory governments which aims to reduce morbidity and mortality associated with vaccine preventable diseases through the improvement in national immunisation coverage rates.

Achievements

The NIC played a major role in increasing the level of immunisation coverage in Australia. In 1989–90, the percentage of Australian children fully immunised at 12 months of age was 53 per cent.[1] In contrast, the coverage rate for children aged 12–15 months of age calculated at 30 June 2003 was 91.7 per cent.[2]

In the first few years, the NIC was instrumental in achieving a nationally co-ordinated childhood immunisation program in Australia including: the adoption of the first national immunisation schedule, implementation of the Australian Childhood Immunisation Register, and development of a national pricing arrangement for vaccines. More recently, the NIC has successfully managed the rapid expansion of the national immunisation program.

The NIC has taken a lead role in the implementation of the Immunise Australia Program, including the Seven Point Plan and a range of related initiatives such as hepatitis B vaccination of infants and adolescents, influenza vaccination for older Australians, measles, mumps, rubella vaccination for school children and young adults, Q fever vaccination of those at occupational risk and the National Meningococcal C Vaccination Program.

Measles

The Measles Control Campaign was one of the largest and most ambitious mass immunisation campaigns ever undertaken in Australia since the polio vaccination program of 1950s, with the NIC having a lead role in the campaign's implementation. The campaign was aimed at providing a second dose of measles-mumps-rubella (MMR) vaccine to all primary school children in Australia and resulted in a significant increase in levels of protection against measles among preschool and primary school age children.

Conducted from 3 August to 6 November 1998, the campaign vaccinated around 1.7 million or 96 per cent of school children aged 5–12 years. More than 1.3 million of these children were vaccinated in the school program in almost 8,800 schools in all states and territories. A serosurvey was conducted after the campaign and showed that 94 per cent of children aged 6–12 years were immune to measles, an increase from 84 per cent before the campaign. It was estimated that the campaign averted 17,500 cases of measles.[3]

A comprehensive report on the campaign was published in 2000. A copy of the report is available from: http://www.immunise.health.gov.au/beatmeasles.pdf

In 2001, the Australian Government announced that it would fund a Young Adult Measles Program. The program was aimed at providing free measles-mumps-rubella vaccine to young adults aged from 18 to 30 years of age. Again, the NIC was instrumental in the implementation of this program.

Young Australian adults are at a greater risk of measles infection because they are too old to have been recipients of the two dose MMR vaccination program for 10–16 year olds introduced in 1994, and generally too young to have been exposed to wild virus prior to the introduction of a measles vaccine into Australia. This high susceptibility is further exacerbated by the large numbers of young adults travelling to countries where measles is endemic and circulating freely in the community. As well as being at higher risk of acquiring measles, young adults are also at higher risk of complications from the disease.

Evidence to date suggests that this cohort has proven to be a difficult group to target for an immunisation program despite targeted information campaigns. The National Centre for Immunisation Research and Surveillance commenced a national serosurvey in late 2002 to measure the success of this Program. The results of this serosurvey are expected to be available towards the end of 2003.

Q fever

The National Q Fever Management Program was the first occupation-based immunisation program with which the NIC has been involved. This program aims to reduce the incidence of Q fever in rural Australia through a targeted screening and vaccination program to those considered to be at high risk. (The Northern Territory is not taking part in the program.)

The National Q Fever Management Program was rolled-out in two phases. Phase 1 of the program commenced in 2001 and was industry-focussed, targeting abattoir workers, those contracted to abattoirs, and sheep shearers. Phase 2 of the program commenced in 2002 and targeted sheep, dairy and beef cattle farmers, their employees and unpaid family members working on farms. An evaluation of the National Q Fever Management Program is due to commence towards the end of 2003.

Meningococcal C

The National Meningococcal C Vaccination Program commenced in January 2003 and is the largest immunisation program with which the NIC has been involved. The program, costing $298 million over four years, sees almost 6 million young Australians who are turning 1 to 19 years of age in 2003, eligible for free meningococcal C vaccine over four years.

Meningococcal C vaccination was added to the National Immunisation Program for children turning 12 months of age from 1 January 2003. As part of the catch-up component of the program, children turning one to five years of age in 2003 have been able to access free meningococcal C vaccine from their general practitioner and school vaccination clinics have commenced for senior high school students aged from 15 to 19 years in all states and territories. Several jurisdictions have already completed school-based vaccination of senior high school students and are now conducting school-based clinics for remaining high school and primary school students. Most jurisdictions anticipate completing school-based vaccination clinics in the second half of 2004.

As was seen in the Measles Control Campaign, school-based vaccination clinics have proven to be the most effective method of achieving high coverage rates and the best public health outcome when vaccinating large groups within the community.

To further raise community awareness about meningococcal disease in general and the National Meningococcal C Vaccination Program in particular, national press advertising ran from May to September 2003.

The catch-up component of the program initially provided access to free meningococcal C vaccine for those who fall into the higher risk groups—children aged one to five years (via general practitioners and other immunisation providers) and senior high school students aged 15 to 19 years (via school-based vaccination clinics).

Throughout these challenges, the NIC has maintained a strong commitment to continuous improvement and best practice in immunisation programs in Australia.

The NIC has also published a broad range of educational resources for both immunisation service providers and consumers, and is represented on a broad range of related immunisation and vaccine preventable disease committees. The NIC meets by teleconference each month and face-to-face three times per year in February, June and October.

Current issues

Issues currently on NIC's agenda include:

- development of strategies to implement changes to the National Immunisation Program. In September 2003, the Australian Government announced changes to the National Immunisation Program. These were:
  - a new single dose of diphtheria-tetanus-pertussis (DTPa) vaccine for adolescents at 15 to 17 years of age to be funded in place of the previous adult diphtheria-tetanus vaccine;
  - expansion of the National Childhood Pneumococcal Vaccination Program to include additional groups within the medical risk factor group; and
  - removal of the dose of diphtheria-tetanus-pertussis (DTPa) at 18 months of age. This has been removed in line with technical advice from the Australian Technical Advisory Group on Immunisation that the dose is now considered unnecessary because of the high levels of immunity from the primary course of vaccinations at 2, 4 and 6 months of age.
- ongoing management of the National Meningococcal C Vaccination Program;
- assisting with provider education on the recently approved *Australian Immunisation Handbook*, 8th edition;
- consultation and negotiation with stakeholders on the development of national immunisations priorities, strategies and service delivery; and
- provision of advice on other immunisation related matters including adverse events following immunisation (AEFI).

The future challenge for the NIC is to sustain recent successes in the decline of vaccine preventable diseases and parallel increases in immunisation coverage and to target hard to reach segments of the population to enable further improvements in the control of vaccine preventable disease in Australia.

For further information, please contact the National Immunisation Committee Secretariat via email to: NIC@health.gov.au

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Communicable diseases surveillance

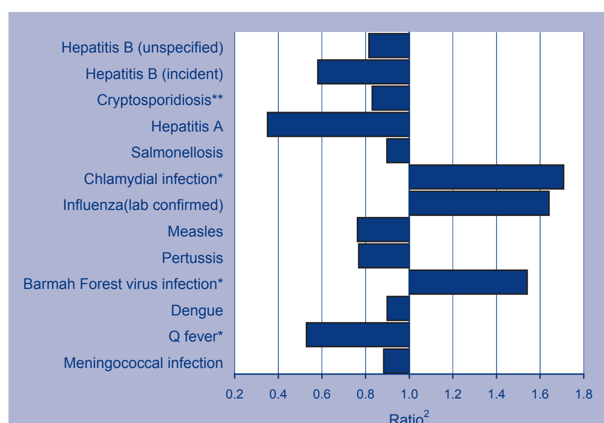
Highlights for 3rd quarter, 2003

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 disease notifications with an onset in the third quarter of 2003, compared with the 5-year mean of the same period. Disease notifications outside the 5-year mean plus or minus two standard deviations are marked with an asterisk. Barmah Forest virus and chlamydial infection notifications exceeded the five-year mean plus two standard deviations while notifications of Q fever was below two standard deviations of the 5-year mean. The number of cryptosporidiosis notifications, a condition notifiable since 2001, was lowest compared with the same quarter in the last two years. The rest of the notifiable diseases were within the expected range of the historical data.

Figure 1. Selected[1] diseases from the National Notifiable Diseases Surveillance System, comparison of provisional totals for the period 1 July to 30 September 2003 with historical data[2]



1. Selected diseases are chosen each quarter according to current activity.
2. Ratio of current quarter total to mean of corresponding quarter for the previous five years.

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

† Notifications above or below the 2-year mean for the same period.

Bloodborne diseases

Hepatitis B

There were 58 incident cases of hepatitis B infection notified in the third quarter of the year; a notification rate of 1.2 cases per 100,000 population. The notification rate of incident hepatitis B has been stable at 1–2 cases per 100,000 population since 1998. The number of cases of hepatitis B unspecified that were notified during the third quarter was 1,581, representing a notification rate of 32.2 cases per 100,000 population. This is 29 per cent lower than the rate reported in 2002 (44 cases per 100,000 population) which was the highest since 1998.

Gastrointestinal diseases

Salmonellosis

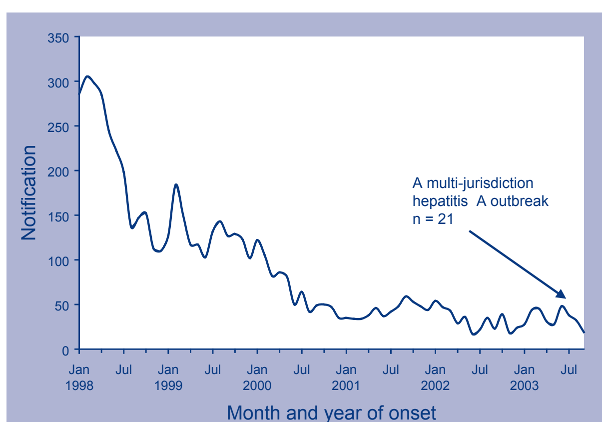
A total of 987 cases of salmonellosis (a notification rate of 20 cases per 100,000 population) were notified to the National Notifiable Diseases Surveillance System (NNDSS) during the third quarter of 2003. This represents a drop of 39 per cent from the previous quarter nationally, which is consistent with the seasonal pattern of salmonellosis notification, peaking in the first quarter of the year and declining during the third quarter. Compared with the same quarter of 2002, there was a 14 per cent decrease in the number of notifications of salmonellosis nationally. The Australian Capital Territory and the Northern Territory were the exception with a 30 per cent and a 77 per cent increase, respectively, compared with the same quarter of 2002.

Hepatitis A: notifications and an update on a multi-jurisdictional outbreak investigation

There were 85 (1.7 per 100,000 population) cases of hepatitis A reported to the NNDSS with an onset in the third quarter of 2003; representing a drop of 17 per cent from the previous quarter. Twenty-five per cent of cases notified during the previous quarter were linked to a hepatitis A outbreak that occurred at an interstate gathering in the Northern Territory. The outbreak, which affected 21 persons including two hospitalisations, lasted from 18 May to 5 June 2003. The Department of Health and Human Services, Tasmania led the investigation into the outbreak. Sally Munnoch, Epidemiologist at the Department of Health and Human Services, Tasmania, reported that the investigation into the outbreak concluded that there was epidemiological evidence for an association between the consumption of coleslaw at the gathering and illness. The investigation could not establish how the contamination of the coleslaw occurred, whether it was via a contaminated ingredient, or from an infected food handler. No microbiological evidence, either from environmental or food samples, were found.

Hepatitis A notification in the third quarter was 65 per cent lower than the historical 5-year mean (Figures 1 and 2). Notifications of hepatitis A have steadily decreased between 1998 and 2002 from 13.3 to 2 cases per 100,000 population. At 1.7 cases per 100,000 population, this quarter represents a further decline of notifications of hepatitis A.

Figure 2. Notifications of hepatitis A infections, Australia, 1998 to September 2003



Vaccine preventable diseases

Measles

Twenty-five cases of measles, nine in New South Wales, 10 in South Australia, four in Queensland and one each in the Northern Territory and Victoria, were reported in the third quarter of 2003. No cases of measles were reported from Tasmania, the Australian Capital Territory or Western Australia for the third consecutive quarter.

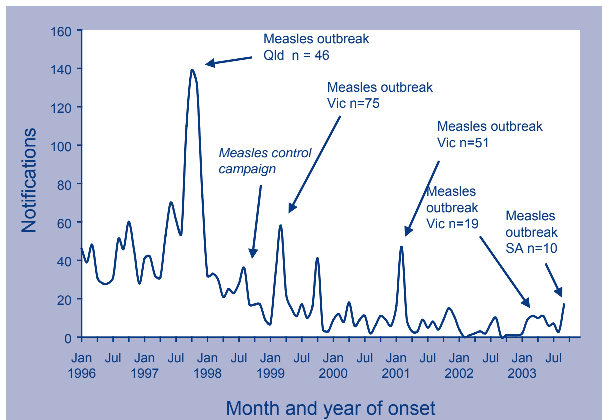
Seven of the nine cases in New South Wales were part of a cluster, reported in the previous quarter, and linked to a 29-year-old male index case suspected to have been infected during recent travel in Nepal. Of the seven linked cases two persons were immunised, two were partially immunised, one had a history of measles infection and no information was available for the remaining cases.

In South Australia the 10 cases of measles, six male and four female (median age 22.5 years, range 11 to 32 years), were linked to an index case with recent travel to New Zealand. Within a month of the onset of illness in the index case (31 August 2003), a fourth generation of transmission of the infection was identified. Those affected included four unvaccinated (two of whom were children aged 11 and 13 years), four partially vaccinated and two of unknown vaccination status. Celia Cooper, from the Department of Human Services, South Australia, said that to control the spread of the infection, public health authorities traced 2,000 potential contacts of identified cases. Other public health measures in response to the outbreak included issuing health alerts to general practitioners and infection control practitioners at metropolitan and rural hospitals, media releases, and alerting interstate public health authorities where identified cases had visited.

The four measles cases in Queensland (age range 23 to 32 years) were from the Whitsundays and were linked to an index case, an Italian tourist. All four cases and the index case were unimmunised for measles.

Figure 3 shows the trend in measles notifications received by NNDSS since 1996. The trend shows a gradual decline in notifications marked with periodic spikes representing outbreaks. The majority of these outbreaks were linked to either visitors or Australians with a recent history of travel,[1,2,3]

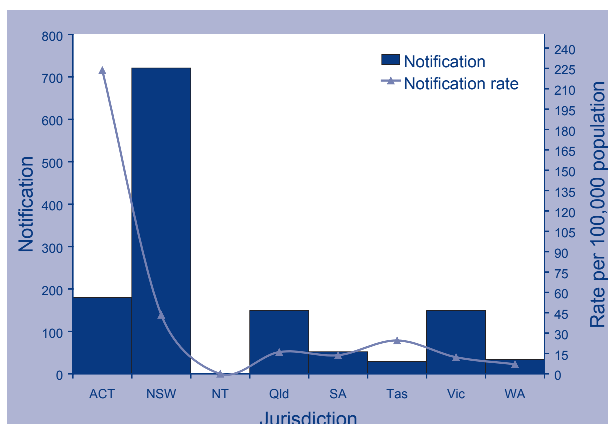
Figure 3. Notifications and reported outbreaks of measles, Australia, 1996 to September 2003



Pertussis

There were 1,314 cases of pertussis notified this quarter, a notification rate of 27 cases per 100,000 population. Although the number of notifications represents a rise of 58 per cent on the previous quarter, it was within the range of the historical data (Figure 1). All jurisdictions, with the exception of Western Australia and the Northern Territory, experienced increases from the last quarter. The Australian Capital Territory and Tasmania experienced the highest percentage increase from the previous quarter (253% and 142% respectively). In the Australian Capital Territory a number of pertussis outbreaks were reported during this quarter, and the notification rate was eight times the national level (Figure 4).

Figure 4. Notifications and rate per 100,000 population of pertussis, Australia, July to September 2003, by jurisdiction



Influenza

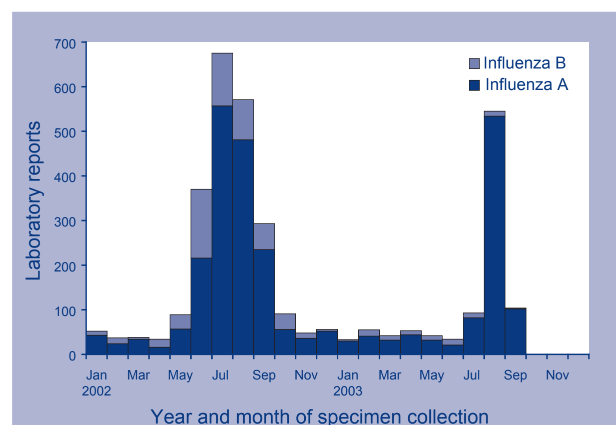
The influenza season peaked in the third quarter of the year with 2,967 cases of laboratory-confirmed cases reported to the NNDSS. This year's influenza season was characterised by:

(a) A rapid rise in influenza activity in August when 47 per cent of cases for the year to date were reported. Nevertheless, notification was 15 per cent lower than the same period in 2002.

(b) The rate of clinical presentations of influenza-like illness (ILI) per 1,000 consultations per week during the peak period was higher in 2003 (24 cases per 1,000 consultations per week) than in the peak period of 2002 (18 cases per 1,000 consultations per week). The peak ILI periods in 2003 and in 2002 were during the week ending 24 August and during the week ending on 20 July, respectively.

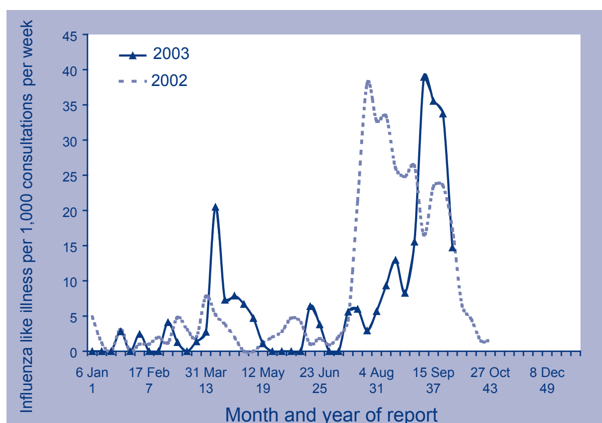
(c) Reports received by the Laboratory Virology and Serology Reporting Scheme (LabVISE) show that there were significantly fewer influenza B viruses detected in 2003 season compared with 2002. The ratio of influenza A to B was 30:1 during the third quarter, and 11:1 for the year to date. This was the highest A:B ratio seen since 1998 and significantly higher than the previous year when the A:B ratio was 5:1 in the third quarter of the year, and 3:1 for the year to date in September (Figure 5).

Figure 5. Laboratory reports of influenza A and B to LabVISE, Australia, 1 January 2002 to 30 September 2003, by month of specimen collection



Influenza activity in the tropical north of Australia peaked during the week ending on 8 September. The rate of clinical presentation of ILI for the third quarter of 2003 was 22 cases per 1,000 consultations per week, higher than the rate of 12 ILI per 1,000 consultations per week reported in the same period of 2002 (Figure 6). The peak ILI rate in 2003 was 38 cases per 1,000 consultations per week, higher than the peak ILI rate in 2002 (33 ILI per 1,000 consultations per week, during the week ending on 11 August).

Figure 6. Consultation rates of influenza-like illness reported to the Northern Territory Influenza Surveillance, Australia, 2002 and 2003, by week



Vectorborne diseases

Dengue

There were 32 cases of dengue reported to NNDSS in the third quarter of 2003, an 88 per cent decline compared to the previous quarter. There has been considerable public health activity to control the disease in Queensland where 85 per cent of notifications in the previous quarter originated. In this state, notifications dropped by 92 per cent from the previous quarter. Compared to the same quarter of 2002, the over all number of notifications was 18 per cent lower, but in Queensland and Victoria was higher by 70 per cent and 100 per cent respectively.

Barmah Forest virus

The number of notifications for Barmah Forest virus during the third quarter of 2003 was 163, a rate of 15 cases per 100,000 population. The number of notifications represents a 79 per cent drop from the previous quarter, consistent with the seasonal pattern. However, compared to the historical data (Figure 1), the number of notifications surpasses the expected number for the same period. The highest increase in notifications from the same period of 2002 occurred in New South Wales and Queensland—increase by 16 per cent and 119 per cent respectively.

Zoonoses

Q fever

In the third quarter of 2003, 79 cases of Q fever were reported to NNDSS; a 33 per cent drop from the previous quarter. In comparison to the historical data, the reported number of Q fever notification was lower than the expected range.

Q fever has long been associated with work in the Australian stock industry, and abattoir workers have been an occupational group at a high risk of infection. Since October 2000, as part of a Commonwealth funded program, abattoir workers and shearers have been eligible for free vaccination against Q fever. In a second phase of the program, which commenced in October 2001, other workers in the beef, sheep and dairy industries may also be vaccinated. Complementing the program, a register of the immune status of individual workers has been maintained on behalf of the livestock industry, Work Cover groups, and state and Commonwealth Departments of Health.

Figure 7 illustrates the trends in Q fever notifications between 1992 and 2003 for New South Wales, Queensland and Victoria, and national totals. The changes in notifications of Q fever may be the result of a combination of control program activities and a natural variability in the disease prevalence.

Other bacterial infections

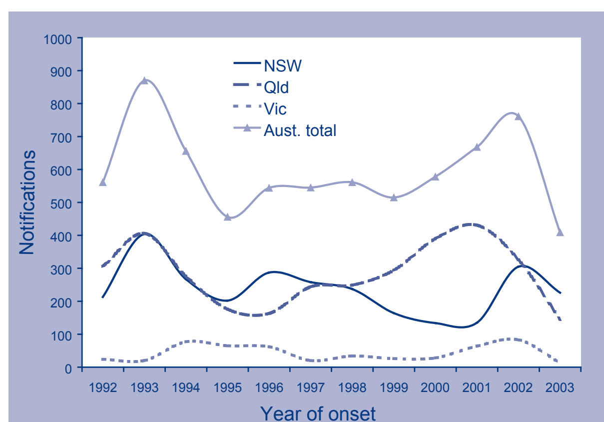
Meningococcal infections

There were 202 cases of meningococcal infection notified in the third quarter, an increase of 96 per cent on the last quarter. Meningococcal infection reaches its seasonal peak in the third quarter of the year (spring). The number of cases in the third quarter 2003 were 19 per cent less from the same period last year, and 12 per cent lower than the historical mean.

Fifty-two per cent of notifications of meningococcal infection were serotype B, 31 per cent C and 17 per cent unknown or other serotypes. Fifteen deaths from meningococcal infection were reported, five due to serotype B, nine due to serotype C, and one due to other serotypes.

For the year to date 404 cases of meningococcal infections, resulting in 25 deaths, were reported to the Communicable Diseases Network Australia. Of the 25 deaths, 28 per cent were due to serotype B, 56 per cent due to serotype C and 16 per cent due to unknown serotype. Nationally, the ratio for serotypes B to C for the year to date was 1.7:1.

Figure 7. Q fever notifications, Australia and, Queensland, New South Wales and Victoria, 1992 to September 2003



With thanks to:

Craig Davies, Queensland Health,

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Mark Bartlett, New South Wales Health Department

Sally Munnoch, Department of Health and Human Services, Tasmania

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Tables

A summary of diseases currently being reported by each jurisdiction is provided in Table 1. There were 24,533 notifications to the National Notifiable Diseases Surveillance System (NNDSS) with a notification date between 1 July and 30 September 2003 (Table 2). The notification rate of diseases per 100,000 population for each State or Territory is presented in Table 3.

There were 6,830 reports received by the Virology and Serology Laboratory Reporting Scheme (LabVISE) in the reporting period, 1 July to 30 September 2003 (Tables 4 and 5).

Table 1. Reporting of notifiable diseases by jurisdiction

Disease	Data received from:
Bloodborne diseases	
Hepatitis B (incident)	All jurisdictions
Hepatitis B (unspecified)	All jurisdictions except NT
Hepatitis C (incident)	All jurisdictions except Qld
Hepatitis C (unspecified)	All jurisdictions
Hepatitis D	All jurisdictions
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
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 jurisdictions

Disease	Data received from:
Vaccine preventable diseases	
Diphtheria	All jurisdictions
Haemophilus influenzae type b	All jurisdictions
Influenza	All jurisdictions
Measles	All jurisdictions
Mumps	All jurisdictions
Pertussis	All jurisdictions
Pneumococcal disease	All jurisdictions
Poliomyelitis	All jurisdictions
Rubella	All jurisdictions
Tetanus	All jurisdictions
Vectorborne diseases	
Arbovirus infection NEC	All jurisdictions
Barmah Forest virus infection	All jurisdictions
Dengue	All jurisdictions
Japanese encephalitis	All jurisdictions
Kunjin	All jurisdictions except ACT*
Malaria	All jurisdictions
Murray Valley encephalitis	All jurisdictions except ACT*
Ross River virus infection	All jurisdictions
Zoonoses	
Anthrax	All jurisdictions
Australian bat lyssavirus	All jurisdictions
Brucellosis	All jurisdictions
Leptospirosis	All jurisdictions
Ornithosis	All jurisdictions
Other lyssaviruses (NEC)	All jurisdictions
Q fever	All jurisdictions
Other bacterial infections	
Legionellosis	All jurisdictions
Leprosy	All jurisdictions
Meningococcal infection	All jurisdictions
Tuberculosis	All jurisdictions

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

Table 2. Notifications of diseases received by State and Territory health authorities in the period 1 July to 30 September 2003, by date of notification*

Disease	State or territory								Total 3rd quarter 2003[1]	Total 2nd quarter 2003	Total 3rd quarter 2002	Last 5 years mean 3rd quarter	Year to date 2003	Last 5 years YTD mean	Ratio[†]
	ACT	NSW	NT	Qld	SA	Tas	Vic	WA							
Bloodborne diseases															
Hepatitis B (incident)	0	12	0	11	4	2	14	15	58	90	104	100	240	282	0.6
Hepatitis B (unspecified)	14	762	NN	213	53	20	398	121	1,581	1,673	1,759	1,910	4,786	5,508	0.8
Hepatitis C (incident)	4	30	0	0	14	3	25	29	105	109	95	109	337	334	1.0
Hepatitis C (unspecified)	71	1,837	70	640	145	94	941	287	4,085	3,747	4,050	4,636	11,557	13,944	0.9
Hepatitis D	0	2	0	0	0	0	6	0	8	5	6	5	18	13	1.6
Hepatitis (NEC)	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0.0
Gastrointestinal diseases															
Botulism	0	0	0	0	0	0	0	0	0	1		0	1	2	0.0
Campylobacteriosis[2]	81	0	51	820	546	144	1,173	454	3,269	3,519	3,367	3,530	11,273	9,961	0.9
Cryptosporidiosis[‡]	0	26	1	35	18	11	69	26	186	310	236	172	1,014	1,607	NA
Haemolytic uraemic syndrome	0	0	0	1	2	0	0	0	3	2	4	3	10	9	1.2
Hepatitis A	1	16	7	5	3	2	22	29	85	108	81	254	310	937	0.3
Hepatitis E	0	4	0	0	0	0	1	0	5	1	4	2	8	8	2.1
Listeriosis	0	6	0	1	0	1	2	1	11	20	11	13	51	47	0.8
Salmonellosis	13	233	80	300	68	25	169	99	987	1,620	1,156	1,105	5,366	5,455	0.9
Shigellosis	0	8	23	13	8	2	9	27	90	106	110	114	349	416	0.8
SLTEC,VTEC[3]	0	0	0	3	5	0	0	1	9	12	14	8	40	31	1.1
Typhoid	0	4	0	1	1	0	3	4	13	6	16	15	38	54	0.9
Quarantinable diseases															
Cholera	0	0	0	0	0	0	0	0	0	1	3	2	1	3	0.0
Plague	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
Viral haemorrhagic fever	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

Table 2. Notifications of diseases received by State and Territory health authorities in the period 1 July to 30 September 2003, by date of notification,* continued

Disease	State or territory								Total 3rd quarter 2003	Total 2nd quarter 2003	Total 3rd quarter 2002	Last 5 years mean 3rd quarter	Year to date 2003	Last 5 years YTD mean	Ratio†
	ACT	NSW	NT	Qld	SA	Tas	Vic	WA							
Sexually transimissible diseases															
Chlamydial (NEC)	130	1,898	377	1,894	453	170	1,647	914	7,483	7,411	6,318	4,440	22,219	12,962	1.7
Donovanosis	0	0	1	0	0	0	0	0	1	3	3	6	11	19	0.2
Gonococcal infection[4]	12	261	326	238	49	1	286	371	1,544	1,694	1,589	1,435	4,996	4,475	1.1
Syphilis	2	201	77	69	4	3	88	12	456	489	531	469	1,466	1,337	1.0
Syphilis - congenital	0	1	3	0	0	0	0	0	4	3	2	2	9	6	1.8
Vaccine preventable disease															
Diptheria	0	0	0	0	0	0	0	0	0	0	0	0	0	1	0.0
Haemophilus influenzae type b	0	1	0	3	0	1	1	0	6	8	3	7	16	25	0.8
Influenza (laboratory confirmed)‡	4	692	110	759	263	7	624	525	2,984	136	2,618	738	3,218	937	NA
Measles	0	9	1	4	10	0	1	0	25	27	17	35	76	130	0.8
Mumps	1	6	0	2	4	0	1	3	17	13	20	42	50	124	0.4
Pertussis	180	721	0	149	52	29	149	34	1,314	832	1,157	1,710	3,047	4,314	0.8
Pneumococcal disease (invasive)‡	11	282	23	225	45	11	128	48	773	558	892	398	1,629	780	NA
Poliomyelitis	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0.0
Rubella	0	3	0	0	0	1	0	1	5	12	79	123	54	294	0.0
Rubella - congenital	0	0	0	0	0	0	0	0	0	0	0	1	0	1	0.0
Tetanus	0	0	0	0	0	0	1	0	1			2	3	3	0.5
Vectorborne diseases															
Arbovirus infection NEC	0	2	0	16	0	0	0	0	18	17	2	5	55	42	3.3
Barmah Forest virus infection	0	57	1	103	0	0	0	2	163	762	106	106	1,204	630	1.5
Dengue	1	7	0	17	1	0	4	2	32	262	39	36	640	201	0.9
Japanese encephalitis‡	0	0	0	0	0	0	0	0	0	0	0	0	0	3	NA
Kunjin virus‡	0	0	0	0	0	0	0	0	0	5	0	0	17	2	NA
Malaria	5	24	5	55	6	4	16	11	126	157	83	160	463	559	0.8
Murray Valley encephalitis‡	0	0	0	0	0	0	0	0	0	0	0	1	0	5	0.0
Ross River virus infection	0	34	0	160	2	0	0	16	212	2,351	101	186	3,146	2,845	1.1

Table 2. Notifications of diseases received by State and Territory health authorities in the period 1 July to 30 September 2003, by date of notification,* continued

Disease	ACT	NSW	NT	Qld	SA	Tas	Vic	WA	Total 3rd quarter 2003	Total 2nd quarter 2003	Total 3rd quarter 2002	Last 5 years mean 3rd quarter	Year to date 2003	Last 5 years YTD mean	Ratio†
Zoonoses															
Anthrax‡	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	NA
Brucellosis	0	0	0	3	0	0	1	0	4	7	8	11	15	25	0.4
Leptospirosis	0	8	0	10	0	0	0	2	20	28	18	35	95	181	0.6
Other lyssavirus (NEC)‡	0	0	0	0	0	0	0	0	0	0	0	0	0	0	NA
Ornithosis	0	24	2	0	0	0	28	0	54	37	96	35	117	87	1.5
Q fever	0	50	0	23	2	0	1	3	79	118	174	149	405	463	0.5
Other bacterial infections															
Legionellosis	0	10	0	13	18	0	12	14	67	87	79	58	240	243	1.2
Leprosy	0	0	0	0	0	0	0	0	0	1	1	1	3	3	0.0
Meningococcal infection	3	78	4	43	10	3	44	18	203	104	249	229	395	465	0.9
Tuberculosis	1	60	7	12	12	1	80	16	189	175	225	257	578	765	0.7
Total	516	6,567	1,169	5,617	1,727	510	5,507	2,920	24,533	26,627	25,426	22,652	79,566	70,531	1.2

1. Totals comprise data from all States and Territories. Cumulative figures are subject to retrospective revision so there may be discrepancies between the number of new notifications and the increment in the cumulative figure from the previous period.
2. Not reported for NSW because it is only notifiable as 'foodborne disease' or 'gastroenteritis in an institution'.
3. Infections with Shiga-like toxin (verotoxin) producing *E. coli* (SLTEC/VTEC).
4. Northern Territory, Qld, SA , Vic and WA: includes gonococcal neonatal ophthalmia.

* Date of notification = a composite of three dates: (i) the true onset date from a clinician, if available, (ii) the date the laboratory test was ordered, or (iii) the date reported to the public health unit.

† Ratio = ratio of current month total to mean of last 5 years calculated as described above.

‡ Notifiable from January 2001 only.

NA Not calculated as only notifiable for under 5 years.

NDR No data received.

NN. Not Notifiable

NEC Not Elsewhere Classified.

- Elsewhere Classified.

Table 3. Notification rates of diseases by State or Territory, 1 July to 30 September 2003. (Rate per 100,000 population)

Disease[1]	State or territory								Australia
	ACT	NSW	NT	Qld	SA	Tas	Vic	WA	
Bloodborne diseases									
Hepatitis B (incident)	0.0	0.7	0.0	1.2	1.1	1.7	1.1	3.1	1.2
Hepatitis B (unspecified)	17.4	45.9	NN	23.0	13.9	16.9	32.7	25.1	32.5
Hepatitis C (incident)	5.0	1.8	NN	NN	3.7	2.5	2.1	6.0	2.7
Hepatitis C (unspecified)	88.2	110.7	141.4	69.1	38.2	79.5	77.2	59.6	83.1
Hepatitis D	0.0	0.1	0.0	0.0	0.0	0.0	0.5	0.0	0.2
Hepatitis (NEC)	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
Gastrointestinal diseases									
Botulism	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
Campylobacteriosis[2]	100.7	NN	103.0	88.5	143.7	121.8	96.3	94.2	100.4
Cryptosporidiosis	0.0	1.6	2.0	3.8	4.7	9.3	5.7	5.4	3.8
Haemolytic uraemic syndrome	0.0	0.0	0.0	0.1	0.5	0.0	0.0	0.0	0.1
Hepatitis A	1.2	1.0	14.1	0.5	0.8	1.7	1.8	6.0	1.7
Hepatitis E	0.0	0.2	0.0	0.0	0.0	0.0	0.1	0.0	0.1
Listeriosis	0.0	0.4	0.0	0.1	0.0	0.8	0.2	0.2	0.2
Salmonellosis	16.2	14.0	161.6	32.4	17.9	21.2	13.9	20.5	20.1
Shigellosis	0.0	0.5	46.5	1.4	2.1	1.7	0.7	5.6	1.8
SLTEC,VTEC[3]	0.0	0.0	0.0	0.3	1.3	0.0	0.0	0.2	0.2
Typhoid	0.0	0.2	0.0	0.1	0.3	0.0	0.2	0.8	0.3
Quarantinable diseases									
Cholera	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
Plague	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
Rabies	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
Viral haemorrhagic fever	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
Yellow fever	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
Sexually transmissible diseases									
Chlamydial infection	161.6	114.3	761.6	204.4	119.2	143.8	135.2	189.7	152.2
Donovanosis	0.0	0.0	2.0	0.0	0.0	0.0	0.0	0.0	0.0
Gonococcal infection[4]	14.9	15.7	658.5	25.7	12.9	0.8	23.5	77.0	31.4
Syphilis	2.5	12.1	155.5	7.4	1.1	2.5	7.2	2.5	9.3
Syphilis - congenital	0.0	0.1	6.1	0.0	0.0	0.0	0.0	0.0	0.1
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	0.3	0.0	0.8	0.1	0.0	0.1
Influenza(laboratory confirmed)	5.0	41.7	222.2	81.9	69.2	5.9	51.2	109.0	60.7
Measles	0.0	0.5	2.0	0.4	2.6	0.0	0.1	0.0	0.5
Mumps	1.2	0.4	0.0	0.2	1.1	0.0	0.1	0.6	0.3
Pertussis	223.7	43.4	0.0	16.1	13.7	24.5	12.2	7.1	26.7
Pneumococcal disease	13.7	17.0	46.5	24.3	11.8	9.3	10.5	10.0	15.7
Poliomyelitis	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
Rubella	0.0	0.2	0.0	0.0	0.0	0.8	0.0	0.2	0.1
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	0.1	0.0	0.0

Table 3. Notification rates of diseases by State or Territory, 1 July to 30 September 2003. (Rate per 100,000 population) , continued

Disease[1]	State or territory								Australia
	ACT	NSW	NT	Qld	SA	Tas	Vic	WA	
Vectorborne diseases									
Arbovirus infection NEC	0.0	0.1	0.0	1.7	0.0	0.0	0.0	0.0	0.4
Barmah Forest virus infection	0.0	3.4	2.0	11.1	0.0	0.0	0.0	0.4	3.3
Dengue	1.2	0.4	0.0	1.8	0.3	0.0	0.3	0.4	0.7
Japanese encephalitis	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
Kunjin virus	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
Malaria	6.2	1.4	10.1	5.9	1.6	3.4	1.3	2.3	2.6
Murray Valley encephalitis	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
Ross River virus infection	0.0	2.0	0.0	17.3	0.5	0.0	0.0	3.3	4.3
Zoonoses									
Anthrax	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
Australian bat lyssavirus	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
Brucellosis	0.0	0.0	0.0	0.3	0.0	0.0	0.1	0.0	0.1
Leptospirosis	0.0	0.5	0.0	1.1	0.0	0.0	0.0	0.4	0.4
Other lyssavirus (NEC)	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
Ornithosis	0.0	1.4	4.0	0.0	0.0	0.0	2.3	0.0	1.1
Q fever	0.0	3.0	0.0	2.5	0.5	0.0	0.1	0.6	1.6
Other bacterial infections									
Legionellosis	0.0	0.6	0.0	1.4	4.7	0.0	1.0	2.9	1.4
Leprosy	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
Meningococcal infection	3.7	4.7	8.1	4.6	2.6	2.5	3.6	3.7	4.1
Tuberculosis	1.2	3.6	14.1	1.3	3.2	0.8	6.6	3.3	3.8

1. Rates are subject to retrospective revision.
2. Not reported for New South Wales because it is only notifiable as ‘foodborne disease’ or ‘gastroenteritis in an institution’.
3. Infections with Shiga-like toxin (verotoxin) producing *E. coli* (SLTEC/VTEC).
4. Northern Territory, Queensland, South Australia, Victoria and Western Australia: includes gonococcal neonatal ophthalmia.
5. Includes congenital syphilis.
6. Includes congenital rubella.

NN Not Notifiable.

NEC Not Elsewhere Classified.

–- Elsewhere Classified.

Table 4. Virology and serology laboratory reports by State or Territory[1] for the reporting period 1 July to 30 September 2003, and total reports for the year[2]

	State or territory								This period 2003	This period 2002	Year to date 2003[3]	Year to date 2002
	ACT	NSW	NT	Qld	SA	Tas	Vic	WA				
Measles, mumps, rubella												
Measles virus	-	4	1	1	1	-	1	-	8	6	48	15
Rubella virus	-	1	-	2	1	-	2	1	7	31	19	80
Hepatitis viruses												
Hepatitis A virus	-	-	-	4	3	-	2	19	28	15	64	54
Hepatitis D virus	-	-	-	-	-	-	6	1	7	3	15	6
Arboviruses												
Ross River virus	-	4	-	51	4	-	1	11	71	29	1,183	377
Barmah Forest virus	-	5	-	27	-	-	1	1	34	42	375	171
Dengue type 1	-	-	-	-	-	-	-	1	1	-	4	1
Dengue type 2	-	-	-	-	-	-	-	1	1	-	2	1
Dengue not typed	-	-	-	-	-	-	-	5	5	11	27	155
Flavivirus (unspecified)	-	-	-	4	-	-	-	-	4	9	108	37
Adenoviruses												
Adenovirus type 1	-	3	-	-	-	-	-	-	3	-	4	-
Adenovirus type 7	-	-	-	-	1	-	-	-	1	-	1	5
Adenovirus type 40	-	-	-	-	-	-	-	8	8	9	28	30
Adenovirus not typed/ pending	-	60	-	31	121	-	10	30	252	322	704	691
Herpes viruses												
Herpes virus type 6	-	-	-	-	-	-	2	-	2	-	5	-
Cytomegalovirus	8	51	1	23	66	1	9	1	160	311	670	849
Varicella-zoster virus	3	41	4	222	74	-	23	89	456	438	1,249	1,334
Epstein-Barr virus	-	12	21	172	190	1	8	59	463	433	1,302	1,316
Other DNA viruses												
Molluscum contagiosum	-	-	-	-	-	-	-	1	1	6	11	18
Contagious pustular dermatitis (Orf virus)	-	-	-	-	-	-	-	1	1	2	3	2
Poxvirus group not typed	-	-	-	-	-	-	1	-	1	1	2	5
Parvovirus	-	1	-	16	1	-	22	21	61	77	166	251
Picornavirus family												
Coxsackievirus A9	-	9	-	-	1	-	-	-	10	1	21	2
Coxsackievirus A16	-	1	-	-	-	-	-	-	1	1	5	3
Echovirus type 4	-	1	-	-	-	-	-	-	1	-	2	-
Echovirus type 6	-	1	-	-	-	-	-	-	1	4	8	60
Echovirus type 9	-	2	-	-	-	-	-	-	2	2	11	16
Poliovirus type 1 (uncharacterised)	-	3	-	-	-	-	-	-	3	8	29	22
Poliovirus type 2 (uncharacterised)	-	3	-	-	-	-	-	-	3	4	7	12
Poliovirus type 3 (uncharacterised)	-	3	-	-	-	-	-	-	3	2	4	4
Poliovirus not typed/ pending	-	-	-	-	-	-	1	-	1	1	3	1
Rhinovirus (all types)	1	69	-	-	7	-	-	46	123	156	374	350
Enterovirus not typed/ pending	-	4	1	4	1	1	1	18	30	140	117	406
Picornavirus not typed	-	-	-	-	-	-	1	-	1	-	6	12

Table 4. Virology and serology laboratory reports by State or Territory[1] for the reporting period 1 July to 30 September 2003, and total reports for the year[2]

	State or territory								This period 2003	This period 2002	Year to date 2003[3]	Year to date 2002
	ACT	NSW	NT	Qld	SA	Tas	Vic	WA				
Ortho/paramyxoviruses												
Influenza A virus	-	484	20	159	407	-	95	224	1,389	1,274	1,593	1,664
Influenza B virus	-	3	-	1	21	-	1	7	33	266	92	496
Parainfluenza virus type 1	-	2	-	1	4	-	-	-	7	76	32	259
Parainfluenza virus type 2	-	4	-	1	10	-	-	2	17	24	66	69
Parainfluenza virus type 3	-	51	-	9	77	-	3	51	191	205	388	355
Respiratory syncytial virus	-	297	-	96	275	37	54	86	845	1,665	1,521	2,771
Other RNA viruses												
HTLV-1	-	-	-	-	1	-	-	1	2	2	10	3
Rotavirus	2	196	-	-	47	2	31	54	332	982	472	1,368
Calicivirus	-	-	3	-	-	-	-	20	23	-	103	8
Norwalk agent	-	-	-	-	-	-	29	-	29	119	70	256
Other												
Chlamydia trachomatis not typed	5	146	4	370	269	11	13	230	1,048	1,041	3,366	2,920
Chlamydia pneumoniae	-	-	1	-	-	-	-	-	1	8	11	15
Chlamydia psittaci	-	1	-	-	-	-	44	-	45	18	87	46
Chlamydia species	-	-	-	-	-	-	1	-	1	1	1	3
Mycoplasma pneumoniae	1	68	3	172	108	10	79	17	458	414	890	997
Mycoplasma hominis	-	4	-	-	-	-	-	-	4	-	9	2
Coxiella burnetii (Q fever)	3	5	-	11	23	-	3	2	47	80	140	192
Rickettsia tsutsugamushi	-	-	-	-	-	-	-	1	1	-	2	-
Streptococcus group A	-	1	-	84	-	-	6	-	91	207	362	424
Yersinia enterocolitica	-	5	-	-	-	-	-	-	5	4	9	8
Brucella species	-	-	-	3	-	-	-	-	3	1	5	4
Bordetella pertussis	1	18	-	12	39	8	26	1	105	224	358	788
Legionella pneumophila	-	1	-	-	3	-	56	-	60	32	113	78
Legionella longbeachae	1	2	-	-	6	-	8	9	26	26	54	48
Legionella species	-	-	-	-	-	-	6	-	6	8	10	13
Cryptococcus species	-	-	-	2	6	-	-	-	8	11	20	25
Leptospira hardjo	-	-	-	-	-	-	-	1	1	-	1	-
Leptospira species	-	1	-	5	4	-	-	-	10	2	21	18
Treponema pallidum	-	22	-	127	121	-	-	-	270	409	944	1,099
Entamoeba histolytica	-	-	-	-	-	-	1	3	4	7	10	22
Toxoplasma gondii	-	7	-	2	-	-	2	-	11	8	32	23
Echinococcus granulosus	-	-	-	-	3	-	-	-	3	5	14	25
Total	25	1,596	59	1,612	1,895	71	549	1,023	6,830	9,183	17,383	20,285

1. State or Territory of postcode, if reported, otherwise State or Territory of reporting laboratory.
2. From January 2000 data presented are for reports with report dates in the current period. Previously reports included all data received in that period.
3. Totals comprise data from all laboratories. Cumulative figures are subject to retrospective revision, so there may be discrepancies between the number of new notifications and the increment in the cumulative figure from the previous period.

– No data received this period

Table 5. Virology and serology reports by laboratories for the reporting period 1 July to 30 September 2003*

State or territory	Laboratory	July 2003	August 2003	September 2003	Total this period
Australian Capital Territory	The Canberra Hospital	1	-	-	1
New South Wales	Institute of Clinical Pathology and Medical Research, Westmead	155	128	75	358
	New Children's Hospital, Westmead	179	305	186	670
	Repatriation General Hospital, Concord	-	-	-	0
	Royal Prince Alfred Hospital, Camperdown	12	-	-	12
	South West Area Pathology Service, Liverpool	163	252	96	511
Queensland	Queensland Medical Laboratory, West End	486	596	607	1,689
	Townsville General Hospital	-	-	-	0
South Australia	Institute of Medical and Veterinary Science, Adelaide	509	630	755	1,894
Tasmania	Northern Tasmanian Pathology Service, Launceston	20	30	20	70
	Royal Hobart Hospital, Hobart	-	-	-	0
Victoria	Monash Medical Centre, Melbourne	44	62	28	134
	Royal Children's Hospital, Melbourne	71	-	25	96
	Victorian Infectious Diseases Reference Laboratory, Fairfield	80	131	105	316
Western Australia	PathCentre Virology, Perth	360	24	600	984
	Princess Margaret Hospital, Perth	-	-	-	0
	Western Diagnostic Pathology	33	17	45	95
Total		2,113	2,175	2,542	6,830

* The complete list of laboratories reporting for the 12 months, January to December 2003, will appear in every report regardless of whether reports were received in this reporting period. Reports are not always received from all laboratories.

– Nil reports

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 2003, 13 conditions are being monitored, five of which are related to communicable diseases. These include influenza, gastroenteritis, antibiotic prescription for acute cough, varicella and shingles. Definitions of these conditions were published in Commun Dis Intell *2003;27:125–126.*

Data from 1 July to 30 September 2003 are shown as the rate per 1,000 consultations in Figures 8, 9 and 10.

Figure 8. Consultation rates for influenza-like illness, ASPREN, 1 July to 30 September 2003, by week of report

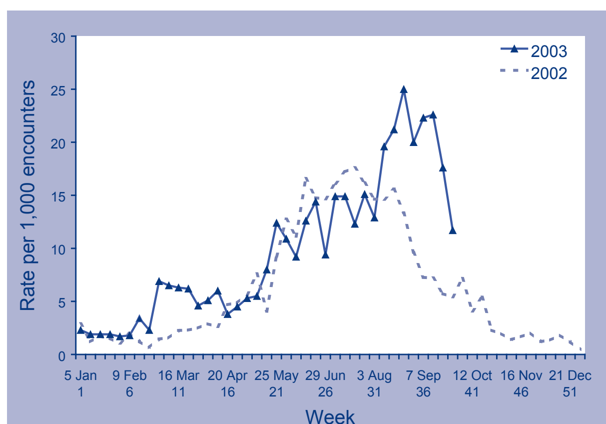


Figure 9. Consultation rates for gastroenteritis, ASPREN, 1 July to 30 September 2003, by week of report

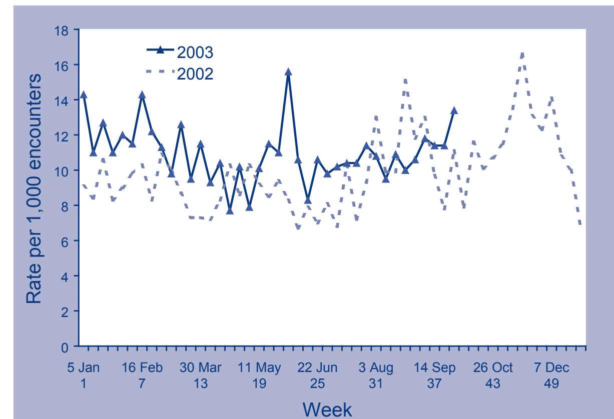
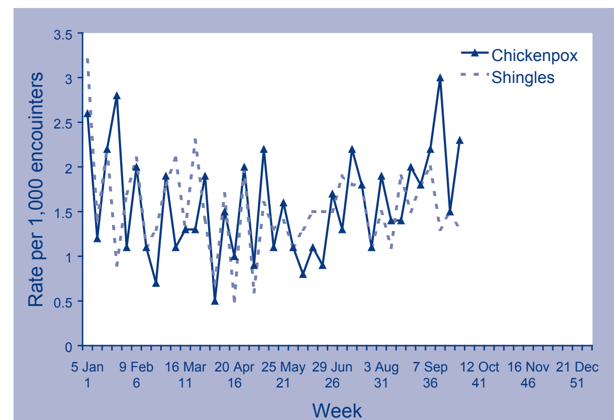


Figure 10. Consultation rates for varicella, ASPREN, 1 July to 30 September 2003, by week of report



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 *2003;27:128.*

Reporting period 1 April to 30 June 2003

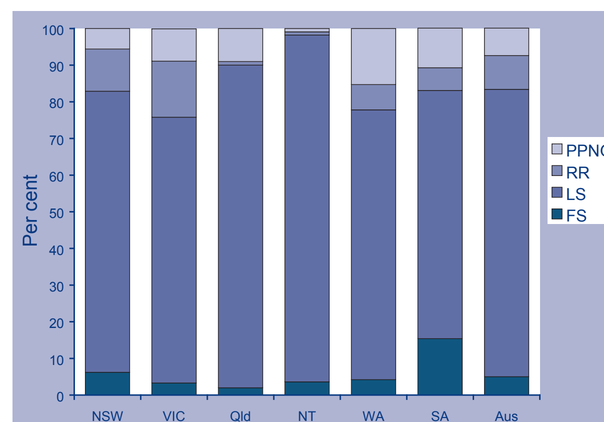
The AGSP laboratories received a total of 980 isolates in the second quarter of 2003 of which 962 remained viable for susceptibility testing. This number approximates the 1,000 strains examined in the same period in 2002. About 32 per cent of this total was from New South Wales, 28 per cent from Victoria, 14 per cent from Queensland, 12 per cent from the Northern Territory and seven per cent from Western Australia and South Australia. Isolates from other centres were few. Numbers examined decreased in New South Wales and Western Australia by about 25 per cent, but increased in Victoria by approximately 50 per cent and substantially in South Australia when compared with data in the same period in 2002. The number of strains from Queensland and Northern Territory examined was similar to last year

Penicillins

In this quarter about 16.6 per cent of all isolates were penicillin resistant by one or more mechanisms—7.5 per cent penicillinase producing *Neisseria gonorrhoeae* (PPNG) and 9.1 per cent by chromosomal mechanisms (CMRNG). The number and proportion of PPNG was little changed from the same period in 2002, but the number of CMRNG decreased from 100 to 88 isolates. The proportion of all strains resistant to the penicillins by any mechanism ranged from 1.8 per cent in the Northern Territory to 24.2 per cent in Victoria.

Figure 11 shows the proportions of gonococci fully sensitive (MIC ≤0.03 mg/L), less sensitive (MIC 0.06–1 mg/L), relatively resistant (MIC ≥1 mg/L) or penicillinase producing aggregated for Australia and by state and territory. The small number of strains from the Australian Capital Territory and Tasmania are aggregated in national data. A high proportion those strains classified as PPNG or else resistant by chromosomal mechanisms fail to respond to treatment with penicillins (penicillin, amoxycillin, ampicillin) and early generation cephalosporins.

Figure 11. Categorisation of gonococci isolated in Australia, 1 April to 30 June 2003, by penicillin susceptibility and region



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

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

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

PPNG Penicillinase producing *Neisseria gonorrhoeae.*

The number of PPNG isolated across Australia (n=72) was little different from the corresponding period in 2002 (n=77). The highest proportion of PPNG was found in isolates from Western Australia (15.3 per cent). PPNG were present in all jurisdictions. Slightly more isolates were resistant to the penicillins by separate chromosomal mechanisms (n=88). CMRNG were especially prominent in Victoria (15.3% of isolates) and New South Wales (11.5%). Only a single CMRNG was detected in the Northern Territory.

Ceftriaxone

Three isolates with decreased susceptibility to ceftriaxone were identified in New South Wales and one each in South Australia and Queensland.

Spectinomycin

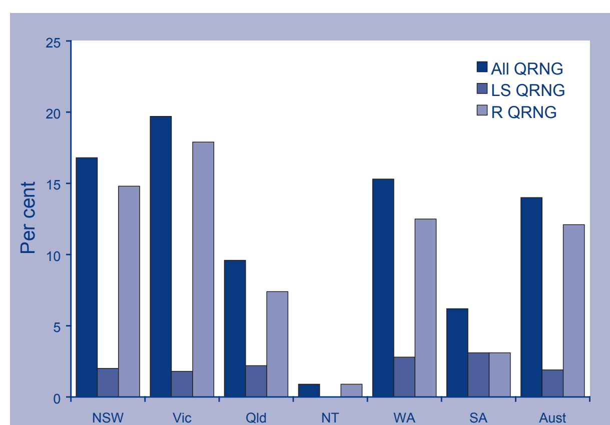
All isolates were susceptible to this injectable agent.

Quinolone antibiotics

The total number (135) and proportion (14%) of all quinolone resistant *N. gonorrhoeae* (QRNG) was slightly higher that seen in the second quarter of 2002 (122 isolates, 12%). The majority of QRNG (117 of 135, 82%) continued to exhibit higher level resistance. Quinolone resistant *N. gonorrhoeae* are defined as those isolates with an MIC to ciprofloxacin equal to or greater than 0.06 mg/L. QRNG are further subdivided into less sensitive (ciprofloxacin MICs 0.06–0.5 mg/L) or resistant (MIC ≥1 mg/L) groups.

QRNG were again widely distributed. The highest numbers were found in Victoria (54) and New South Wales (52) with the highest rate (20%) in Victoria (Figure 12). QRNG rates above five per cent were maintained in all centres except the Northern Territory (0.9%). Details of geographic acquisition of QRNG were available in only 40 instances. Local contact (26) was twice as common as overseas contact (14) indicating that a substantial degree of domestic transmission continues. MICs ranged up to 16 mg/L.

Figure 12. The distribution of quinolone resistant isolates of *Neisseria gonorrhoeae* in Australia, 1 April to 30 June 2003, 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 (92) and proportion (9.5%) of high level tetracycline resistance (TRNG) isolates were lower than in the second quarter of 2002. TRNG represented between five per cent (South Australia) and 22.2 per cent (Western Australia) of all isolates. TRNG was not found in isolates from the Northern Territory.

Reference

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

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 2003;27:57.

HIV and AIDS diagnoses and deaths following AIDS reported for 1 April to 30 June 2003, as reported to 30 September 2003, are included in this issue of Communicable Diseases Intelligence (Tables 6 and 7).

Table 6. New diagnoses of HIV infection, new diagnoses of AIDS, and deaths following AIDS occurring in the period 1 April to 30 June 2003, 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 2003	This period 2002	Year to date 2003	Year to date 2002
HIV diagnoses	Female	0	6	0	4	1	0	6	4	21	17	42	49
	Male	2	88	2	26	11	0	51	12	192	158	388	347
	Sex not reported	0	2	0	0	0	0	0	0	2	0	3	1
	Total[1]	2	96	2	30	12	0	57	16	215	175	433	399
AIDS diagnoses	Female	0	0	0	2	0	0	0	0	2	0	5	7
	Male	0	5	0	0	1	0	2	1	9	41	39	97
	Total[1]	0	5	0	2	1	0	2	1	11	41	45	105
AIDS deaths	Female	0	0	0	0	1	0	0	0	1	0	5	2
	Male	0	2	0	1	0	0	2	0	5	17	21	31
	Total[1]	0	2	0	1	1	0	2	0	6	17	26	33

1. Totals include people whose sex was reported as transgender.

Table 7. Cumulative diagnoses of HIV infection, AIDS, and deaths following AIDS since the introduction of HIV antibody testing to 30 June 2003 and reported, by sex and state or territory

	Sex	State or territory								
		ACT	NSW	NT	Qld	SA	Tas	Vic	WA	Australia
HIV diagnoses	Female	28	705	14	202	79	7	281	154	1,470
	Male	240	12,145	119	2,325	765	85	4,495	1,039	21,213
	Not reported	0	236	0	0	0	0	24	0	260
	Total[1]	268	13,112	133	2,535	844	92	4,818	1,199	23,001
AIDS diagnoses	Female	9	213	0	56	30	4	87	33	432
	Male	90	4,943	38	932	377	47	1,791	397	8,615
	Total[1]	99	5,170	38	990	407	51	1,887	432	9,074
AIDS deaths	Female	4	125	0	38	20	2	57	22	268
	Male	71	3,402	26	611	252	31	1,334	273	6,000
	Total[1]	75	3,536	26	651	272	33	1,398	296	6,287

1. Totals include people whose sex was reported as transgender.

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 April and 30 June 2002, at 24 months of age for the cohort born between 1 April and 30 June 2001, and at 6 years of age for the cohort born between 1 April and 30 June 1997 according to the Australian Standard Vaccination Schedule.

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

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

Immunisation coverage for 'fully immunised' children at 12 months for Australia has increased from the last quarter by 0.5 percentage points to 91.7 per cent (Table 8). There was very little change in 'fully immunised' coverage by state or territory. The Northern Territory showed the biggest change (–1.8%). Four jurisdictions had changes in coverage greater than 0.8 per cent for individual vaccines: Victoria, with increases in coverage for diphtheria, tetanus, pertussis (DTP) (+1.1%), and poliomyelitis (OPV) (+1.1%); Queensland, with increases in coverage for DTP (+0.9%), and OPV (+0.9%); the Australian Capital Territory (the ACT) with increases in coverage for *Haemophilus influenzae* type b (Hib) (+1.0%) and hepatitis B (hep B) (+1.6%); and the Northern Territory, with decreases in coverage for Hib (–2.0%) and hep B (–0.9%).

Coverage measured by 'fully immunised' at 24 months of age for Australia decreased marginally from the last quarter by 0.1 percentage point to 89.2 per cent (Table 9). Coverage for individual vaccines for Australia basically remained unchanged with DTP still 3–4 percentage points lower than other vaccines for this age group. This difference was due to the greater number of DTP doses required to be considered up-to-date at 24 months of age. The only important jurisdictional changes in coverage at 24 months of age occurred in the Australian Capital Territory, with a decrease in DTP (–2.0%), MMR (–1.0%) and 'fully immunised' (–1.8%) coverage, and a 1.3 per cent increase in polio coverage.

Table 10 shows immunisation coverage estimates for 'fully immunised' and for individual vaccines at six years of age for Australia and by state or territory. 'Fully immunised' coverage at six years of age for Australia increased by 0.8 percentage points from the previous quarter to 83.1 per cent with significant increases in the Australian Capital Territory (+2.7%) and South Australia (+2.0%). Encouragingly, coverage for all individual vaccines at six years of age increased in all states and territories with some substantial increases in some jurisdictions. There were significant increases in measles, mumps and rubella (MMR) coverage in the Australian Capital Territory (+3.2%), the Northern Territory (+2.4%) and South Australia (+1.7%), and similar increases in coverage for DTP in the same three jurisdictions. Coverage for individual vaccines assessed at six years, is now over 85 per cent in a number of different jurisdictions, and close to 85 per cent in most jurisdictions. Whilst it is still a way off from the coverage target of 90 per cent, it is encouraging to see gains being made in coverage for children in this age group. Assuming there is no differential reporting of immunisations to the ACIR by providers for children of different ages, it seems likely that these increases in coverage are a result of an increase in uptake of immunisation at six years of age.

Table 8. Proportion of children immunised at 1 year of age, preliminary results by disease and State for the birth cohort 1 April to 30 June 2002; assessment date 30 September 2003

Vaccine	State or territory								
	ACT	NSW	NT	Qld	SA	Tas	Vic	WA	Australia
Number of children	1,012	20,945	890	12,409	4,199	1,408	14,991	6,068	61,922
Diphtheria, tetanus, pertussis (%)	92.9	92.9	92.3	92.9	92.8	92.6	93.8	91.1	92.9
Poliomyelitis (%)	92.8	92.9	92.0	92.8	92.7	92.5	93.7	90.9	92.8
Haemophilus influenzae type b (%)	94.8	94.5	94.8	94.9	95.1	95.2	95.4	93.9	94.8
Hepatitis B (%)	95.9	95.6	96.4	95.3	95.6	95.2	95.2	93.7	95.3
Fully immunised (%)	91.6	91.5	89.9	92.1	91.6	92.1	92.6	89.7	91.7
Change in fully immunised since last quarter (%)	+0.1	+0.5	-1.8	+1.0	+0.2	+0.2	+0.9	-0.2	+0.5

Figure 13 shows the trends in vaccination coverage from the first ACIR-derived published coverage estimates in 1997 to the current estimates. There is a clear trend of increasing vaccination coverage over time for children aged 12 months, 24 months and six years, although the rate of increase has slowed over the past two years, especially for children in the 12 and 24 month age groups.

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

Figure 13. Trends in vaccination coverage, Australia, 1997 to 2003, by age cohorts

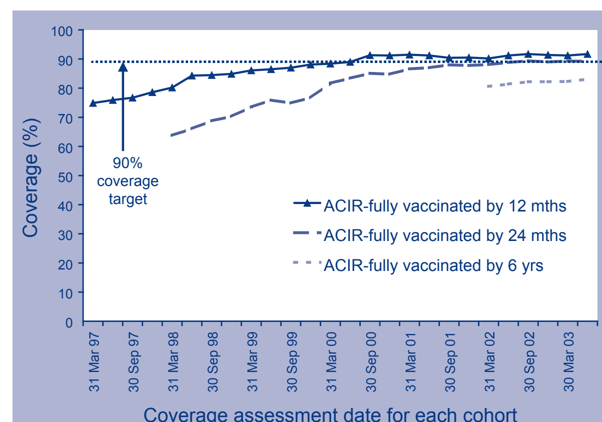


Table 9. Proportion of children immunised at 2 years of age, preliminary results by disease and State for the birth cohort 1 April to 30 June 2001; assessment date 30 September 2003[1]

Vaccine	State or territory								
	ACT	NSW	NT	Qld	SA	Tas	Vic	WA	Australia
Total number of children	960	21,152	900	13,029	4,348	1,414	14,822	6,107	62,732
Diphtheria, tetanus, pertussis (%)	88.4	90.8	90.1	92.1	91.9	93.9	91.8	89.6	91.3
Poliomyelitis (%)	94.7	95.0	96.7	95.0	95.8	96.5	95.2	94.5	95.1
Haemophilus influenzae type b (%)	93.1	93.6	95.1	94.2	94.6	95.7	94.3	92.8	94.0
Measles, mumps, rubella (%)	92.4	93.7	95.4	94.5	94.7	95.3	94.5	93.2	94.1
Hepatitis B(%)	94.8	95.7	98.0	95.6	96.0	97.2	96.1	95.2	95.8
Fully immunised (%)[2]	85.1	88.5	89.4	90.0	90.2	93.1	89.9	87.4	89.2
Change in fully immunised since last quarter (%)	-1.8	+0.0	+0.5	+0.1	-0.3	-0.5	-0.6	+0.4	-0.1

1. The 12 months age data for this cohort was published in *Commun Dis Intell* 2002;26:627.
2. These data relating to 2-year-old children should be considered as preliminary. The proportions shown as 'fully immunised' appear low when compared with the proportions for individual vaccines. This is at least partly due to poor identification of children on immunisation encounter forms.

Table 10. Proportion of children immunised at 6 years of age, preliminary results by disease and State for the birth cohort 1 April to 30 June 1997; assessment date 30 September 2003

Vaccine	State or territory								
	ACT	NSW	NT	Qld	SA	Tas	Vic	WA	Australia
Total number of children	1,044	21,975	809	13,391	4,686	1,557	15,832	6,443	65,737
Diphtheria, tetanus, pertussis (%)	84.7	84.7	84.3	84.1	85.0	84.3	87.1	82.8	85.0
Poliomyelitis (%)	84.7	84.7	85.3	84.1	85.3	84.5	87.3	82.9	85.1
Measles, mumps, rubella (%)	85.0	83.4	85.4	83.9	84.4	83.2	87.0	82.7	84.4
Fully immunised (%)[1]	83.1	82.1	81.6	82.4	83.3	82.3	85.8	81.1	83.1
Change in fully immunised since last quarter (%)	+2.8	+0.9	+0.5	+0.4	+2.0	+0.1	+0.5	+0.5	+0.8

1. These data relating to 6-year-old children should be considered as preliminary. The proportions shown as 'fully immunised' appear low when compared with the proportions for individual vaccines. This is at least partly due to poor identification of children on immunisation encounter forms.

National Enteric Pathogens Surveillance System

The National Enteric Pathogens Surveillance System (NEPSS) collects, analyses and disseminates data on human enteric bacterial infections diagnosed in Australia. These pathogens include Salmonella, E. coli, Vibrio, Yersinia, Plesiomonas, Aeromonas *and* Campylobacter. Communicable Diseases Intelligence *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.

Reported by Joan Powling (NEPSS Co-ordinator) and Mark Veitch (Public Health Physician), Microbiological Diagnostic Unit — Public Health Laboratory, Department of Microbiology and Immunology, University of Melbourne. NEPSS can be contacted at the above address or by telephone: +61 3 8344 5701, facsimile: +61 3 9625 2689. For more information see Commun Dis Intell *2003;27:129.*

Reports to the National Enteric Pathogens Surveillance System of Salmonella infection for the period 1 July to 30 September 2003 are included in Tables 11 and 12. Data include cases reported and entered by 14 October 2003. Counts are preliminary, and subject to adjustment after completion of typing and reporting of further cases to NEPSS.

Third quarter 2003

The total number of reports to NEPSS of human *Salmonella* infection declined to 879 in the third quarter of 2003, 42 per cent less than the second quarter of 2003. The incidence of human salmonellosis is lowest in the third quarter of each year. Case counts to 14 October 2003 are approximately 90 per cent of the expected final counts for the quarter.

During the third quarter of 2003, the 25 most common *Salmonella* types in Australia accounted for 504 (57%) of all reported human *Salmonella* infections.

Seventeen of the 25 most common *Salmonella* infections in the second quarter of 2003 were amongst the 25 most commonly reported in the previous quarter.

Although counts of *S.* Typhimurium phage types 135, 9 and 170 and *S.* Infantis declined compared with the previous quarter, they remained among the six most common salmonellae in the nation and were mostly reported from the eastern mainland states.

S. Typhimurium phage type 170 was the fourth most commonly reported *Salmonella* in Australia in the third quarter of 2003. Reports of this phage type continue to exceed historical averages. There were a further five reports of the similar phage type, *S.* Typhimurium phage type 108.

Reports of *S.* Typhimurium phage type U290 have increased progressively since 2001.

Acknowledgement

We thank scientists, diagnostic and reference laboratories, State and Territory health departments, and the Australian Government Department of Health and Ageing for their contributions to NEPSS.

Table 11. Reports to the National Enteric Pathogens Surveillance System of *Salmonella* isolated from humans during the period 1 July to 30 September 2003, as reported to 14 October 2003

	ACT	NSW	NT	Qld	SA	Tas	Vic	WA	Australia
Total all *Salmonella* for quarter	14	216	62	240	58	24	177	88	879
Total contributing *Salmonella* types	12	78	31	90	36	14	76	48	183

Table 12. Top 25 *Salmonella* types identified in Australian States and Territories, 1 July to 30 September 2003

National rank	*Salmonella* type	State or territory								Total 3rd quarter 2003	Last 10 years mean 3rd quarter	Year to date 2003	Year to date 2002
		ACT	NSW	NT	Qld	SA	Tas	Vic	WA				
1	*S.* Typhimurium 135	0	23	2	7	1	1	11	8	53	75	558	526
2	*S.* Typhimurium 9	2	14	2	4	2	2	14	3	43	74	337	503
3	*S.* Saintpaul	2	8	4	21	2	0	3	3	43	43	231	313
4	*S.* Typhimurium 170	1	23	0	4	1	4	6	0	39	16	338	320
5	*S.* Typhimurium U290	1	11	0	1	0	1	19	1	34	3	111	80
6	*S.* Infantis	1	16	1	2	3	0	7	0	30	19	163	85
7	*S.* Typhimurium 197	0	6	0	12	0	0	3	0	21	3	130	48
8	*S.* Typhimurium RDNC	0	6	0	2	3	0	4	5	20	17	51	49
9	*S.* Muenchen	0	1	5	4	0	1	3	6	20	16	108	101
10	*S.* Chester	0	1	5	6	1	0	0	6	19	20	173	133
11	*S.* Birkenhead	0	5	0	12	0	0	0	1	18	21	142	194
12	*S.* Virchow 8	0	1	1	14	0	0	1	0	17	16	127	253
13	*S.* Hvittingfoss	0	1	1	10	0	0	3	0	15	8	74	131
14	*S.* Adelaide	0	2	4	1	4	0	0	2	13	8	26	29
15	*S.* Oranienburg	0	3	0	0	0	0	0	9	12	7	42	24
16	*S.* Typhimurium 126	0	1	0	2	0	3	2	3	11	18	53	172
17	*S.* Agona	0	1	0	6	1	0	1	2	11	13	53	65
18	*S.* Stanley	0	1	0	4	0	0	6	0	11	12	34	42
19	*S.* Aberdeen	0	0	1	10	0	0	0	0	11	12	66	114
20	*S.* Typhimurium 12	0	2	0	3	3	0	3	0	11	3	73	53
21	*S.* Bovismorbificans 32	0	3	0	2	0	0	6	0	11	2	16	4
22	*S.* Typhimurium 6 var 1	1	6	0	2	1	0	1	0	11	1	21	6
23	*S.* Typhimurium 4	0	3	0	0	6	0	1	0	10	6	54	49
24	*S.* Ball	0	0	10	0	0	0	0	0	10	5	39	47
25	*S.* Zanzibar	0	0	0	6	1	0	3	0	10	5	34	17

Overseas briefs

World Health Organization

This material has been summarised from information on the World Health Organization Internet site. A link to this site can be found under 'Other Australian and international communicable diseases sites' on the Communicable Diseases Australia homepage.

Cholera in Liberia – update 6

(30 September 2003)

The cholera outbreak in Monrovia remains a public health concern. As of 21 September 2003 the total number of cases since the beginning of the year was 18,038 cases with 17,561 cases occurring since the beginning of the epidemic in June. The case fatality rate is low (below 1%) in the cholera treatment centres run by Médecins sans Frontières France and Belgium and by MERLIN. Many non-governmental organisations have opened oral rehydration corners to increase the coverage of the population. The caseload seems to be decreasing in some areas, probably as the result of the widespread chlorination of wells undertaken during the last four weeks, by the World Health Organization and UNICEF. In Central Monrovia, the low access to safe water and sanitation remains critical and limits the effectiveness of control measures. An additional concern is the increase in bloody diarrhoea cases (141 cases reported between 15–21 September) which might be due to shigellosis. Laboratory tests are currently underway.

ProMED-mail

This material has been summarised from information provided by ProMED-mail (http://ww.promedmail.org). A link to this site can be found under 'Other Australian and international communicable diseases sites' on the Communicable Diseases Australia homepage.

Dengue/dengue haemorrhagic fever update

India (New Delhi)

Source: Times of India, 24 September 2003 (edited)

There are a growing number of dengue cases in New Delhi with about 130 to 24 September 2003. Officials of the Delhi government's health department blame this year's heavy rainfall for the spurt in dengue cases. In 1996, over 10,000 people were affected by dengue and 423 died. This year, only one person has died, but the number of cases so far is almost three times that of 2002.

Creation of breeding conditions for mosquitoes is an offence under municipal laws and is liable to prosecution and fine. Over 590,000 houses have been visited this year and 23,110 houses were found to be harbouring *Aedes* mosquitoes. As many as 17,248 persons have been served legal notices while 6,111 persons have been prosecuted.

Indonesia (North Sumatra)

Source: Antara News Agency, 24 September 2003 (edited)

From January up to September 2003, 14 people have died of dengue fever in North Sumatra, a provincial health official said. The victims were part of a total of 344 people affected by the disease during the period. In Medan city alone, 220 people contracted the disease. However, compared to the situation in other provinces, the number of dengue-fever victims in North Sumatra during the period was low. In North Sumatra, dengue fever tended to spread at the beginning of the rainy season, namely between August and December.

Brazil

Source: National Foundation for Health, Brazil, online, accessed 28 September 2003

As of week 34, 271,161 cases of dengue have been notified, 259,800 of them in the first six months, a reduction of 65 per cent compared with the first six months of 2002. As of 28 September 2003, 7,487 cases have been notified for July and August 2003, but 13 states have only very preliminary figures. The north east region has the most cases notified, 139,529, followed by the south east with 77,281 cases. Currently dengue virus serotypes 1, 2, and 3 are active in 22 states. Five hundred and seventy five cases of dengue haemorrhagic fever (DHF) have been confirmed in 20 states. This represents only 21 per cent of the total DHF cases in 2002. The dengue death rate for the whole country currently stands at about 6.4 per cent.

Colombia

Source: El Tiempo – Oriente, Colombia, 24 September 2003 (edited)

Thirteen confirmed and 52 suspected cases of DHF and 135 cases of classic dengue fever have been reported so far in 2003. During 2002, 82 cases of DHF and 64 cases of classic dengue fever were reported. In September 2002 only three cases of DHF were diagnosed, but so far this month, 10 cases have already been reported.

Bovine spongiform encephalopathy update 2003

Source: BSE in Europe, http://home.hetnet.nl/~mad.cow/, updated 26 September 2003 (edited)

Table 1 shows the number of cases of bovine spongiform encephalopathy reported in the past three years.

Most countries show decreased incidence of recorded bovine spongiform encephalopathy cases, compared to 2002. The exceptions are Portugal, Spain (with a relatively high number of cases), as well as Japan and Poland.

Table 1. Cases of bovine spongiform encephalopathy, 2001 to 26 September 2003, and total since 1987, by country

Country	2001	2002	2003 to date	Total since 1987
UK	1,175	1,104	409	183,510
Austria	1	0	0	1
Belgium	46	38	11	114
Canada	0	0	1	1
Czech Republic	2	2	1	5
Denmark	6	3	2	13
Finland	1	0	0	1
France	274	239	104	852
Germany	125	106	38	281
Greece	1	0	0	1
Ireland	246	333	134	1,310
Israel	0	1	0	1
Italy	50	36	23	111
Japan	3	2	2	7
Liechtenstein	0	0	0	2
Luxembourg	0	1	0	2
Netherlands	20	24	12	64
Portugal	110	86	89	814
Poland	0	4	4	8
Slovakia	5	6	2	13
Slovenia	1	1	1	3
Spain	82	127	113	324
Switzerland	42	24	16	447

Influenza update 2003

Source: MMWR Morb Mortal Wkly Rep 2003;52;911–913, 26 September 2003 (edited)

United States and worldwide: influenza activity update – May to September 2003

During the period May to September 2003, influenza A (H3N2) viruses circulated worldwide and were associated with mild to moderate levels of disease activity. Influenza A(H1)* and B viruses were reported less frequently. The influenza A(H1N2) strain appears to have resulted from the reassortment of the genes of the circulating influenza A(H1N1) and A(H3N2) subtypes.

United States of America

In the United States of America, influenza surveillance is conducted by a network comprising four components, including approximately 900 sentinel health-care providers who regularly report data on patient visits for influenza-like illness (ILI) and approximately 120 US-based World Health Organization (WHO) and National Respiratory and Enteric Virus Surveillance System (NREVSS) collaborating laboratories that report the number of respiratory specimens tested and the number and type of influenza viruses identified during October to mid-May. During the period 18 May to 13 September 2003, the weekly percentage of patient visits to sentinel providers for ILI ranged from 0.5–0.9 per cent, and WHO and NREVSS collaborating laboratories tested 9,145 respiratory specimens, of which 68 (0.7%) were positive. Of the positive results, 31 (45.6%) were influenza A(H3N2) viruses, 25 (36.8%) were influenza type-B viruses, 7 (10.3%) were influenza A(H1) viruses, and 5 (7.0%) were influenza A viruses that were not subtyped. Influenza A viruses were reported each week during mid-May to mid-August. Influenza B viruses were reported for five consecutive weeks during mid-May to mid-June and during the week ending 2 August 2003.

* A(H1) includes both the A(H1N1) and A(H1N2) influenza virus types.

Worldwide

During the period May to July 2003, influenza A(H3N2) viruses predominated in Africa (Madagascar and South Africa). In Asia, influenza A(H3N2) viruses predominated in Hong Kong and Thailand and were reported in Bangladesh, China, Guam, Indonesia, Japan, and Singapore. In Oceania (Australia, New Caledonia, and New Zealand), influenza A(H3N2) viruses predominated and were associated with widespread activity in Australia and New Zealand. In Latin America, influenza A(H3N2) viruses predominated in Brazil, Chile, and Uruguay. Influenza A(H3N2) viruses also circulated widely in Argentina and were isolated in El Salvador, French Guiana, Paraguay, and Peru.

During May to August 2003, sporadic cases of influenza A(H3N2) infection were reported in North America (Canada and Mexico) and Europe (Latvia, Norway, and the United Kingdom). Influenza A(H1) viruses predominated in Argentina and also were reported from Brazil, Chile, French Guiana, Iceland, New Zealand, Peru, South Africa, Trinidad and Tobago, the United Kingdom, and Uruguay. In Africa, influenza B viruses were reported in May (Morocco) and July (South Africa). A small number of influenza B viruses were identified in Asia (Bangladesh, Hong Kong, Japan, and Thailand), South America (Argentina, Brazil, Peru, and Uruguay), and Australia. During May, influenza B viruses were reported in Canada, Latvia, Mexico, and the United Kingdom.

Severe acute respiratory syndrome worldwide: etiology

Confirmation of the identity of the severe acute respiratory syndrome agent

Kuiken T, Fouchier RAM, Schutten M, Rimmelzwaan GF, van Amerongen G, van Riel D, et al. *Newly discovered coronavirus as the primary cause of severe acute respiratory syndrome. http://image.thelancet.com/extras/03art6318web.pdf*

The summary of the paper reads as follows:

'The worldwide outbreak of severe acute respiratory syndrome (SARS) is associated with a newly discovered coronavirus, SARS-associated coronavirus (SARS-CoV). We carried out clinical and experimental studies to assess the role of this virus in the causation of SARS. We tested clinical and postmortem samples from 436 SARS patients in six countries for infection with SARS-CoV, human metapneumovirus, and other respiratory pathogens. We infected four cynomolgus macaques (*Macaca fascicularis*) with SARS-CoV in an attempt to replicate SARS and did necropsies on day six after infection. SARS-CoV infection was diagnosed in 329 (75%) of 436 patients fitting the case definition of SARS; human metapneumovirus was diagnosed in 41 (12%) of 335, and other respiratory pathogens were diagnosed only sporadically. SARS-CoV was, therefore, the most likely causal agent of SARS. The four SARS-CoV-infected macaques excreted SARS-CoV from nose, mouth, and pharynx from two days after infection. Three of four macaques developed diffuse alveolar damage similar to that in SARS patients and characterised by epithelial necrosis, serosanguineous exudate, formation of hyaline membranes, type 2 pneumocyte hyperplasia, and the presence of syncytia.

SARS-CoV was detected in pneumonic areas by virus isolation and RT-PCR, and was localised to alveolar epithelial cells and syncytia by immunohistochemistry and transmission electron microscopy. We conclude that replication in SARS-CoV-infected macaques of pneumonia similar to that in human beings with SARS, combined with the high prevalence of SARS-CoV infection in SARS patients, fulfil the criteria required to prove that SARS-CoV is the primary cause of SARS.'

SARS: Singapore laboratory acquired case

Source: WHO CSR website. http://www.who.int/csr/don/2003_09_10/en/, 10 September 2003 (edited)

Singapore had a laboratory-confirmed case of SARS coronavirus (CoV) infection. This single case was in a 27-year-old postgraduate medical student who worked in a virology laboratory in Singapore. The patient developed fever, was hospitalised and isolated, and his fever has now resolved. Contact tracing is continuing, but it has not identified any secondary cases arising from this infection.

It is well recognised that most viruses can cause a range of illness from mild to severe. The goal of WHO's SARS guidance in the post-outbreak period is to rapidly identify threats to global public health. Consequently, WHO's case definitions are designed to provide early warning and give health officials direction for responding to a new outbreak of SARS. The Singapore case was mild, isolated, and has not produced secondary cases, and therefore is not regarded as a public health concern.

Report of review panel on new SARS case and biosafety

Source: Ministry of Health Singapore, Press Release 23 September 2003

An 11-member Review Panel, comprising local and external experts, was tasked by the Ministry of Health to review (a) epidemiologic data of the recent SARS case and (b) the biosafety requirements and practices at Singapore's BSL3 laboratories. The Panel has completed its investigation and submitted its report to the Ministry.

Epidemiologic investigation

From the results of its investigations, the Panel has concluded that the patient most likely acquired the infection in the Environmental Health Institute laboratory where he had worked. Inappropriate laboratory procedures and a cross-contamination of West Nile virus samples with SARS coronavirus in the laboratory led to the infection of the doctoral student. No evidence could be found of any other source of infection. The Panel's conclusion is further supported by the results of the genome sequencings on the laboratory strain of SARS coronavirus and that of the patient's. Both genome sequences were found to be closely related. The Panel also established that there was no evidence of secondary transmission and this was an isolated case of SARS.

Biosafety standards

The Panel also reviewed the three existing BSL3 laboratories and found a large range of biosafety structures and practices. The Panel recommended that a national legislative framework for ensuring international standards in biosafety in laboratories be established. Where gaps in biosafety standards were identified, the Panel has made specific recommendations to rectify them. The details are in their report.

The full report of the Panel is available at: http://www.moh.gov.sg/sars/pdf/Report_SARS_Biosafety.pdf

Measles – Marshall Islands

Source: Morb Mortal Wkly Rep 2003;52:888–889, 19 September 2003 (edited)

During the period 13 July to 13 September 2003, a total of 647 clinically diagnosed measles cases were reported on Majuro Atoll in the Republic of the Marshall Islands (RMI); this is the first measles outbreak reported in RMI since 1988. An additional 74 suspected measles cases are under investigation. Of the 647 cases, 15 (2%) are laboratory confirmed, either by serology, polymerase chain reaction, or viral culture. The age of patients ranged from two weeks to 43 years (median: 12 years); 479 (74%) patients were aged <20 years. The overall measles incidence on Majuro Atoll (estimated 2003 population: 25,097) was 26 cases per 1,000 population. The incidence was highest among infants aged <1 year (160/1,000 population), followed by children aged one to four years (40/1,000). A total of 58 people with measles were admitted to hospital; three patients died.

To stop measles transmission, the Ministry of Health in RMI recommended measles, mumps, and rubella vaccine (MMR) for all infants aged 6 to 11 months and all people aged 1 to 40 years who did not have documented proof of measles immunity. As of 13 September 2003, 98 per cent of those aged six months to 40 years had documentation of receipt of at least one dose of MMR. Campaign activities that delivered 16,913 doses included; (1) vaccinating health care and public health workers; (2) vaccinating children at nine vaccination posts across the atoll; (3) delaying the start of the school year until school children were vaccinated, and requiring documentation of vaccination for school entry; and (4) conducting neighbourhood and house-to-house vaccination in areas where adequate coverage was not reached.

To prevent spread from Majuro Atoll, vaccination campaigns were conducted in other atolls and islands in RMI. The Ministry of Health suspended travel of sea vessels and airlines from Majuro Atoll until vaccination campaigns had been completed in other atolls and islands, and required proof of MMR vaccination for all travellers leaving Majuro Atoll for other atolls or islands or for international destinations. Spread to other areas in the Pacific and to the United States of America has been limited; five measles cases in Hawaii, three in Guam, one in Palau, and one in California are believed to be linked to this epidemic.

The source of importation of the measles virus to Majuro Atoll has not yet been determined, but the H1 genotype found in this outbreak is common in Asia, and the specific strain has been reported recently in measles cases from Japan and China. The Advisory Committee on Immunisation Practices recommends that all international travellers be immune to measles because it is endemic or epidemic in many parts of the world, including developed countries. People aged <40 years who are travelling to RMI during the next 60 days should be aware that RMI requires documentation of measles immunity for all departing passengers on international flights. The documentation must fulfil the same age-specific requirements used in the vaccination campaign.

Antimicrobial growth promoters, livestock – Denmark

Source: Eurosurveillance Weekly 2003;7, 4 September (edited)

An international review panel has concluded that Denmark's termination of the use of antimicrobial growth promoters seems to have achieved its desired public health goal, and that countries with similar animal production conditions could see similar benefits if they follow suit. The World Health Organization (WHO) convened the independent international panel in November 2002. The panel evaluated the impact that withdrawal of antimicrobial growth promoters in Denmark has made on the efficiency of food animal production, animal health, food safety, and consumer prices. Denmark's termination program is consistent with WHO global principles, which call on governments to adopt a proactive approach to reduce the need for antimicrobials in animals and ensure their prudent use.

Antimicrobial growth promoters were withdrawn both as a public health measure and to ensure consumer confidence in Denmark, in 1999. The concern was that resistance to these antimicrobials in the food animal reservoir would lead to clinical problems in humans. The quantity of antimicrobials used in food animals in Denmark has declined 54 per cent from peak use between 1994 and 2001.

Before the program began, most pigs and broiler chickens in Denmark were given antimicrobials, such as avilamycin, avoparcin, tylosin, and virginiamycin, for most of their lives. After withdrawal, average use declined to 0.4 days in broiler chickens (with life span of around 42 days) and 7.9 days in pigs (with life span of around 170 days). Pork production in Denmark has continued to increase, and effects on poultry production were small.

Data from the Danish Integrated Antimicrobial Resistance Monitoring and Research Programme showed that ending the use of the above antimicrobials has greatly reduced the reservoir of resistant *Enterococcus faecium* in the food animal reservoir, thus reducing the reservoir of resistance genes. For example, resistance to avilamycin, avoparcin, and streptogramins in *Enterococcus faecium* isolates from broiler chickens declined from 60 to 80 per cent before withdrawal of antimicrobials to only 5 to 35 per cent after. The panel considered that the threat of antimicrobial resistance to human health has been reduced.

The panel concluded that use of antimicrobials for the sole purpose of growth promotion can be ended in countries that have similar conditions to Denmark, that is, where animal farming methods are intensive, and animals have a relatively high health status, and where a high level of infrastructure and capacity to monitor antimicrobial use and resistance exist. In July 2003, the European Parliament adopted a regulation on feed additives that completes a ban on antibiotic growth promoters in feed, and the European Union's Scientific Steering Committee has recommended that the use of these antimicrobials be progressively phased out.

CJD (new var.) – United Kingdom: update 2003

Source: UK Department of Health, CJD/BSE monthly statistics, 1 September 2003, (edited)

On 1 September 2003 the Department of Health issued the latest information about the numbers of known cases of Creutzfeldt-Jakob disease. This included cases of variant Creutzfeldt-Jakob disease—the form of the disease thought to be linked to bovine spongiform encephalopathy. Table 2 shows the known cases.

Summary of vCJD cases

Deaths: from definite vCJD (confirmed): 99

from probable vCJD (without neuropathological confirmation): 33

from probable vCJD (neuropathological confirmation pending): 4

from definite or probable vCJD (as above): 136

Probable vCJD cases still alive: 4

Total number of definite or probable vCJD (dead and alive): 140

The death toll continues to rise but overall the data are consistent with the continued decline in vCJD incidence reported during the preceding six months.

Table 2. Cases of Creutzfeldt-Jakob disease reported in the United Kingdom, 1990 to 2003

Year	Referrals	Sporadic	Iatrogenic	Familial	GSS	vCJD	Total
1990	53	28	5	0	0	–	33
1991	75	32	1	3	0	–	36
1992	96	45	2	5	1	–	53
1993	78	37	4	3	2	–	46
1994	118	53	1	4	3	–	61
1995	87	35	4	2	3	3	47
1996	134	40	4	2	4	10	60
1997	161	60	6	4	1	10	81
1998	154	63	3	4	1	18	89
1999	170	62	6	2	0	15	85
2000	178	49	1	2	1	28	81
2001	179	56	3	2	2	20	83
2002	163	72	0	4	1	17	94
2003*	107	35	4	1	0	15	55
Total	1,753	667	44	38	19	136	904

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