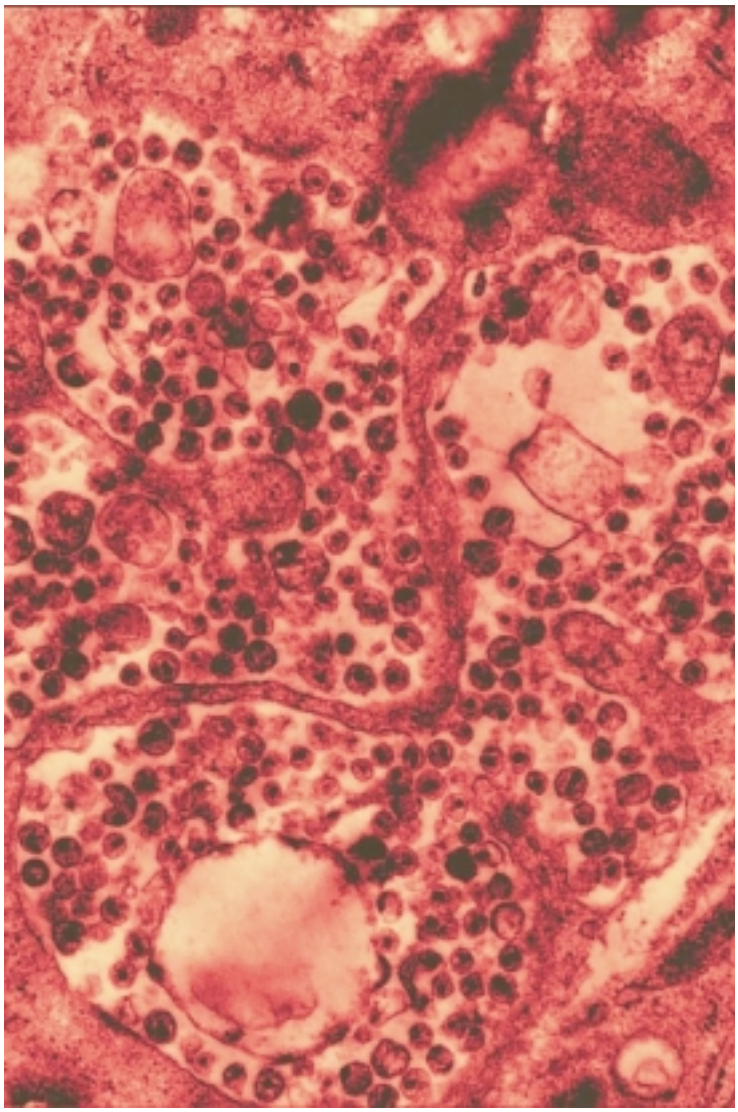
Communicable Diseases Intelligence

Quarterly report



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Front cover photograph: HIV

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Editorial: Development of Australia's response to bovine spongiform encephalopathy and variant Creutzfeldt-Jakob disease

Fiona J Brooke[1]

On 18 July 2001, the Australian government announced details of a permanent system of categorisation and certification designed to ensure that all beef products entering Australia for human consumption are free from the dangers of bovine spongiform encephalopathy (BSE). Details of the certification system will be published in a later edition of *Communicable Diseases Intelligence*.

The certification regime is the latest measure announced with the objective of ensuring Australia remains free of animal transmissible spongiform encephalopathies (TSEs) and to reduce the risk of variant Creutzfeldt-Jakob disease (vCJD) amongst the human population.

Australia has had two historical incidents of animal TSEs. In 1952 an imported herd of sheep was found to have scrapie. The herd was quarantined without any further spread of the infectious agent. In 1991, a cheetah in a zoo in Broome was discovered to have feline spongiform encephalopathy. This incident was attributed to exposure to infected meat and bone meal (MBM), before the animal was imported into Australia.

In 1966 Australia banned any further importation of MBM, except from New Zealand, to reduce the risk of foot and mouth disease. The ban also had the effect of protecting Australian cattle from any subsequent exposure to the BSE epidemic that emerged in the United Kingdom (UK) in the 1980s. Subsequently, Australia imposed restrictions on the importation of live cattle, semen and embryos from the UK. When BSE spread into mainland Europe in the 1990s, these restrictions were extended to all known BSE-affected countries. Australia is also fully compliant with the animal testing requirements of the Office International des Epizooties (OIE).

Variant CJD, first recognised in 1996 and attributed to eating BSE infected beef and beef product, has been a major tragedy for many families in the UK and Europe. With the announcement of a possible case of vCJD in Hong Kong, it can only be a matter of time before other countries with high numbers of travellers to and from the UK and continental Europe, such as Australia, begin reporting cases. Australians are amongst the most travelled people in the world.

There is still major uncertainty about many aspects of BSE and vCJD — particularly about the science, the control measures and the effectiveness of public health responses.

We do not know if vCJD is transmissible through human blood and blood products. The preliminary report by Houston et al in the *Lancet* in August 2000 helped prompt Australia to defer blood donors who had spent 6 months or more in the UK. In the meantime, Australia continues to monitor blood donor deferral policies globally, while awaiting confirmation of the Houston finding.

There are now emerging reports of possible treatment regimes. At this time it is far too early to tell how effective these treatments may be, or indeed whether they will be effective in the long term. Australia will continue to monitor the situation with great interest.

If the ultimate size of the UK BSE epidemic (e.g. over the next 40 years) were known, an assessment of the risk to other exposed populations could be given. Knowledge of the epidemic size would allow calculation of the expected number of vCJD cases for a given number of contaminated animals in the food chain. It is likely to be 5-10 years before the size of the UK epidemics can be estimated with any more precision. With BSE epidemics still emerging in other European countries, an accurate quantification of risk for humans cannot be given.

Control measures introduced in the UK, and subsequently into other BSE-affected and at-risk countries, have reduced the entry of BSE-contaminated material into the human food chain. Nevertheless, there are continuing uncertainties.

- BSE is asymptomatic for most of its course and the point at which various tissues become potentially infectious has not been established.
- The minimum size of an infectious dose for humans and the cumulative dose response relationship is unknown.
- It is not known whether all mechanisms of transmission have been identified, especially as some cases of BSE continue to be detected in cattle born after the imposition of control measures in the UK.
- The lympho-reticular system is recognised as the site of early replication of prions, although the infectivity of these tissues in the early stages of infectivity is still under study.
- The precise amount, destination and end-use of exports of possibly infective meat and bone meal is still uncertain.
- The degree of compliance with control measures, particularly in countries at an early stage of the BSE epidemic, remains unclear.
- Many of the countries that are recorded as having received potentially infective MBM are not fully compliant with OIE requirements and thus may not be in a position to detect an emerging BSE epidemic.

1. Fiona J Brooke, Manager, TSE Task Force, National Centre for Disease Control, Communicable Diseases and Environmental Health Branch, E-mail: Fiona.Brooke@health.gov.au

There are still about 1,000 new cases of BSE detected each year in the UK, despite the MBM ban having been in force and closely monitored for compliance for some years.

Previously, BSE could only be diagnosed at autopsy. Pre-symptomatic diagnosis is now possible by immunochemical testing of brain tissue at the time of slaughter. However, there are still no reliable tests available to detect BSE early in the incubation period. The tests currently approved for use in cattle down to the age of 30 months in the European Union, have been evaluated by comparison with inoculation experiments in mice. Although there are reports of positive results in younger animals, the testing regimes have still to be validated at younger ages. The tests for diagnosis of BSE, currently in use throughout Europe, are now being validated to ensure they are equally sensitive and specific in Australian cattle breeds.

There are still no validated tests available to detect BSE-contamination in processed foods and meat products at the end of the human food chain. Therefore, the traditional method of ascertaining the level and extent of threat from an imported foodborne disease to the Australian population — namely by testing at the point of entry — cannot be applied.

As BSE has continued to spread throughout the UK and Europe, the risk from non-food exposures has also come under increasing examination. In 2000, Australia's Chief Medical Officer, Professor Richard Smallwood, convened a group of experts to examine the safety of some vaccines derived from master seeds which include bovine products — some of which are from BSE affected countries. That group reported in November 2000 that these vaccines should remain on the market as they posed negligible, risk from BSE contamination.

The Therapeutic Goods Administration (TGA) is taking a proactive approach to ensure that any potential risk of exposure to BSE through medicines and medical devices is minimised. In line with other international regulatory agencies, the TGA is continuing to require that animal derived ingredients used in the manufacture of new products submitted for approval, should be sourced from BSE-free countries. Where this is not possible, evidence needs to be provided as to the product's safety from BSE risk. TGA is also continuing an extensive review of existing products to identify and remove any potential risks of exposure to BSE.

As the magnitude of potential exposure to BSE became more apparent, Australia's National Health and Medical Research Council established a Special Expert Advisory Committee on Transmissible Spongiform Encephalopathies (SECTSE) to provide independent scientific advice to government on the animal and human risks for Australia.

Since its establishment, SECTSE has been examining a number of areas of potential risk to the Australian population and animal industries. The committee has also advised on a proposal to undertake a comprehensive survey of blood donors which will describe travel profiles of blood donors for use in risk assessments and policy development. The survey should be completed by the end of 2001.

SECTSE is also keeping itself fully informed of risk assessments being undertaken within the Department of Health and Aged Care, the Australia New Zealand Food Authority and the Department of Agriculture, Fisheries and Forestry — Australia (AFFA).

CJD is not currently a notifiable disease in Australia. Since 1993, the Commonwealth has funded the Australian National CJD Case Registry, based at the Department of Pathology in the University of Melbourne, to undertake national surveillance of human TSEs, provide diagnostic assistance for physicians and advise on infection control issues. In 2000, this role was broadened to include surveillance and diagnostic testing for vCJD.

Contingency planning is an important aspect of any national response to an emerging disease threat. AFFA has contingency plans in place should a case of BSE be detected in Australia. A vCJD response plan is also close to finalisation should a case of vCJD be detected in Australia.

Australia's Infection Control Guidelines are also being reviewed and will be made widely available upon publication. SECTSE is examining the need for additional guidance on infection control issues for vCJD, and will be issuing those separately.

Editorial: The United Nations General Assembly special session on HIV/AIDS: 'Global crisis – global action'

Paul Lehmann,
HIV/AIDS Section, Population Health Division, Department of Health and Aged Care

The 26th Special Session of the General Assembly of the United Nations, held in New York from 25 to 27 June 2001, represents the high water mark of global political commitment to the fight against HIV/AIDS. It was also the first time in its 54-year history that the General Assembly of the United Nations had convened to discuss HIV/AIDS as a public health issue. More than 20 years since the onslaught of the HIV/AIDS pandemic and after 60 million HIV infections, the world community now has a plan of action for combatting the pandemic.

In calling for the Special Session, the United Nations' objective was to secure a global response to HIV/AIDS through the adoption of a Declaration of Commitment. This Declaration of Commitment would then be used to identify priorities for national, regional and international action and as a yardstick for the Joint United Nations Program on HIV/AIDS (UNAIDS) policy and programs. Importantly, the occasion also served as an opportunity to endorse the Secretary-General's proposed Global Fund for HIV/AIDS and Health.

Australia's delegation to the Special Session was lead by the Minister for Health Michael Wooldridge, and included: the Australian Ambassador to the United Nations, Ms Penny Wensley; the Commonwealth Chief Medical Officer, Professor Richard Smallwood; Chair of the Australian National Council on AIDS, Hepatitis C and Related Diseases (ANCAHRD); Mr Chris Puplick; President of the Australian Federation of AIDS Organisations (AFAO), Mr Bill Whittaker; and representatives from the Population Health Division, Department of Health and Aged Care and the Australian Agency for International Development (AusAID).

Australia played a crucial role in turning the idea of an agreed statement of commitment on HIV/AIDS into a reality. In the months preceding the Special Session in June, Australia's Ambassador to the United Nations, Penny Wensley, co-chaired alongside Ambassador Ka of Senegal, a series of formal and informal meetings convened to draft the Declaration of Commitment. Technical assistance for the process was ably provided by Dr Peter Piot, Executive Director of UNAIDS, and his colleagues.

In the view of many seasoned United Nations (UN) observers, the process of agreement on the wording of text in the Declaration proved to be a particularly arduous and complex one. HIV/AIDS has manifested itself differently in almost every country and region of the world and therefore defies a simple global response. Real national differences often meant that countries came to discuss the issue of HIV/AIDS from vastly different perspectives.

Australia has confronted the sensitive issues connected to HIV/AIDS transmission in the context of a public health approach to the virus. For many other countries, particularly those with strict and widely observed moral codes, open discussion of HIV transmission risk factors, such as those associated with male to male sex, injecting drug use and commercial sex work, was never going to be easy.

Negotiations were further complicated by differences of opinion on issues such as access to HIV/AIDS treatments, and the inevitable nexus between the global response to HIV/AIDS and other global issues such as poverty, underdevelopment, conflict and respect for human rights.

Highlighting the difficulties in reaching agreement on the wording of text was the fact that final agreement was not reached until after the commencement of the Special Session itself, despite a series of preliminary meetings earlier in the year and round-the-clock efforts over the weekend preceding the Session.

As negotiations drew to a close it became clear, however, that responding to HIV/AIDS at the global level provided many more arguments to unite countries than to divide them. The rousing support for the Declaration of Commitment, which was accepted by the General Assembly by acclamation, served to confirm this fact.

In reaching agreement on the Declaration of Commitment, delegations agreed to the inclusion of a number of important provisions. These include: genuine international agreement and commitment to specific targets for prevention; strong language on human rights and the rights of women to protect themselves from HIV infection; and a sound balance between discussion of education/prevention and treatment/care. These advances are reinforced at the commencement of each chapter and are part of the process for follow up and evaluation.

As might be expected with a document agreed to by compromise among 189 nations, the Declaration of Commitment from the Special Session has not been without its critics. While the document breaks new ground in a number of areas, it has been criticised for its lack of specificity in others, particularly in relation to the listing of vulnerable groups. Ultimately, however, to focus on these shortcomings would serve to underrate the significant advances contained in the document.

In his statement to the Special Session, Minister Wooldridge emphasised the importance of countries not losing sight of the objective of the Declaration because of particular concerns, for instance, over the identification of vulnerable groups in the Declaration. Participating in a Ministerial round table discussion, Minister Wooldridge outlined our approach in responding to the disease which is recognised internationally as one of the most successful in the world and many aspects of it are widely emulated. He also emphasised

our role in developing and financing strategies and programs to combat the pandemic in the Asia-Pacific region.

As the President of the General Assembly noted during his closing remarks, the real work has only just begun. This is no less true for Australia as we continue our efforts to sustain our domestic response to the pandemic.

The key points of Minister Wooldridge's statement to the General Assembly Special Session on HIV/AIDS are summarised below.

- Australia remains well placed to continue its contribution to arresting the spread of, and minimising the personal, social and economic impacts of the HIV/AIDS pandemic. Australia has achieved remarkable success in reducing transmission of HIV infection through sustained political consensus on Australia's HIV/AIDS policy and willingness for government to engage and work with affected and vulnerable communities.
- The Australian Government expressed disappointment that vulnerable groups - men who have sex with men, sex workers, injecting drug users, institutional and prison populations and indigenous people - were not explicitly named in the Declaration of Commitment. In Australia, the support and commitment of such groups and their active involvement and partnership has been the basis of Australia's national response to HIV/AIDS. AIDS activism has been directed towards constructive participation in Australia.
- Promoting prevention is an important part of a comprehensive integrated response which includes all aspects of infrastructure development, treatment, care and support.
- Support and encouragement for a robust and inclusive partnership between a wide range of groups has been a defining feature of Australia's response to HIV/AIDS. The full involvement of communities through, amongst others, civil society organisations including people living with HIV/AIDS, is also crucial to an international response.
- Partnership in decision making, policy development and program implementation continues to ensure that activities combatting HIV/AIDS are effective and sustainable.
- All countries must be involved in HIV/AIDS prevention and control efforts, which extend beyond their domestic situation. Australia's focus for assistance will continue to the Asia Pacific region. In July 2000, the Australian Government announced a new A$200 million Global HIV/AIDS Initiative as a major expansion of Australia's assistance for international work on HIV/AIDS. In implementing the Global Initiative, Australia will continue to support and work collaboratively with UNAIDS and other UN agencies at global and regional levels.
- At a regional level, Australia supports efforts to increase political commitment in responding to the pandemic. To assist these efforts, Australia is hosting the 6th International Congress on AIDS in Asia and the Pacific in Melbourne in October 2001. Australia is inviting Ministers from 38 countries across the Asia-Pacific region to a separate but complementary part of the Congress to consider how to address the broad range of problems caused by HIV/ AIDS, particularly its social and economic impacts.

Copies of the UN Declaration of Commitment are available from the UN Website at: http://www.unfoundation.org/.

Salmonella Zanzibar in rural South Australia

Ingrid G Tribe and Scott Cameron
Communicable Disease Control Branch, Department of Human Services, South Australia

In South Australia, human infection with *Salmonella* Zanzibar is uncommon. The last reported case of infection with this serovar was in 1995. In May 2001, 2 (1 male, 1 female, age range: 26 to 31 years) cases of *Salmonella* Zanzibar were investigated by the Communicable Disease Control Branch, Department of Human Services, South Australia. Hypothesis generating interviews sought demographic, illness, employment, travel, social activities, restaurant/take-away food consumption and animal contact information for the 7 day period prior to the onset of symptoms.

Case 1, a resident of a rural township in South Australia, reported no recent intrastate, interstate or overseas travel. The case reported purchasing food items from various local take-away and restaurant outlets in the 7 day period prior to the onset of illness. Case 2, a resident of metropolitan Adelaide, reported intermittent employment in the same rural township in the 7 day period prior to the onset of symptoms. Both cases reported eating at a local Italian restaurant between 5 and 7 May 2001. With the exception of individual serves of salmon bruschetta and antipasto, chicken-based pasta dishes were consumed by both cases. Case 2 however, was unable to recall the specific chicken based pasta dish consumed. An environmental investigation conducted 4 weeks after the exposures found food handling procedures were satisfactory. No evidence of cross-contamination could be identified. In addition, there were no reports of gastrointestinal illness in restaurant employees. However, a report of a presumptive case of food poisoning had been received by the local council. This complainant reported the consumption of a chicken-based pasta dish at the same Italian restaurant on 28 April 2001. The complainant did not seek medical attention.

Although the source for this outbreak was not established, an epidemiological investigation identified a link between infection with *Salmonella* Zanzibar and the consumption of prepared food at the restaurant. No further cases were notified. This event highlights the importance of investigatory public health action even when the numbers of case reports are low and the connection between cases is not obvious at first glance.

OzFoodNet: enhancing foodborne disease surveillance across Australia

Quarterly report January to March 2001

Martyn Kirk for the OzFoodNet Working Group

Introduction

In the latter part of 2000 the Commonwealth Department of Health and Aged Care established and funded a collaborative network — coined OzFoodNet — to enhance the existing surveillance mechanisms for foodborne disease across Australia.

The aims of OzFoodNet are to:

1. estimate the incidence of foodborne disease in Australia;
2 learn more about the causes and determinants of foodborne disease;
3. identify risky practices associated with food handling and preparation; and
4. train foodborne disease epidemiologists.

The work of OzFoodNet will improve surveillance of foodborne disease across Australia. Collaborators of OzFoodNet include State health authorities, the National Centre for Epidemiology and Population Health (NCEPH), the Public Health Laboratory Network (PHLN), Territory health departments, and national government agencies.

State health authorities have employed epidemiologists to enhance foodborne disease surveillance and conduct applied research into foodborne disease. Western Australia, South Australia, Tasmania, Queensland and Victoria are enhancing surveillance across the whole State. New South Wales is concentrating efforts on the Hunter region although comparative data for the rest of the State area were supplied where available.

Notifications in the first quarter 2001

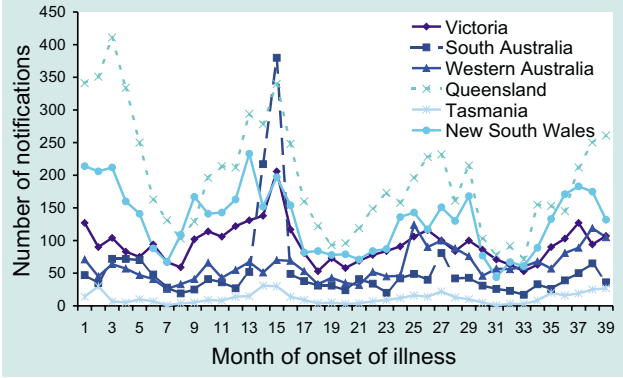
In the first quarter of 2001, *Campylobacter* was the most commonly notified enteric pathogen, with 3056 cases reported to all OzFoodNet sites, except New South Wales, where campylobacteriosis is not a notifiable disease. Despite the large number of cases, there was only one cluster investigated in OzFoodNet sites for the period.

OzFoodNet sites reported 2115 cases of salmonellosis (529 from New South Wales) and 10 outbreaks. Notifications of salmonellosis for the first quarter of 2001 were increased in the Tasmania (19.0%), and in the Hunter Public Health Unit (Hunter PHU) (15.8%), when compared with the three-year mean for the same quarter for 1998–2000. Although there was also a 37.7 per cent increase in Western Australia, this reflected a change in the notifications system there, whereby laboratory notifications were counted for the first time only from the beginning of 2000. Notifications of *Salmonella* infections from the States of South Australia (-53.4%), Queensland (-18.8%), Victoria (-12.0%), and New South Wales (-9.5%) were decreased compared to three-year mean values† (Figure).

Salmonella Typhimurium Phage Type 135 was one of the most common serovars in OzFoodNet sites (Table 1). It was represented in the top 5 serovars of all sites, except for Tasmania. Some States distinguished Typhimurium 135 into variant 135a, although reference laboratories are currently clarifying the sub-typing of this serovar. In Tasmania, the predominant serovar reported during the quarter was *S.* Mississippi (56/71 cases) — a serovar endemic to this State. The incidence of *S.* Mississippi in Tasmania was similar to previous years and there was no obvious geographical clustering. Data on *Salmonella* serovars were not available for samples from Western Australia at the time of writing this report.

OzFoodNet sites reported 22 cases of listeriosis with reported onset of symptoms in the first quarter of 2001. The highest number of cases was reported from Queensland (8 cases), followed by Victoria (6 cases), and New South Wales (5 cases). The majority of these cases were reported in older males who were immunocompromised. The median age of cases varied between 63 and 83 years. None of the cases in Queensland involved maternal-foetal infections.

Figure. Notifications of *Salmonella* infections to OzFoodNet sites, January 1998 to March 2001, by date of onset



* Totals for foodborne disease in this report differ from those reported for the same period to the National Notifiable Diseases Surveillance System. This report analysed cases by date of report and included cases with a disease onset date in 2000 and a date of report in the first quarter 2001 unless specified otherwise.

† The three-year mean value for South Australia was skewed by the large number of cases in a *Salmonella* Typhimurium 135a outbreak associated with contaminated orange juice in March 1999.

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The full membership of the OzFoodNet Working Group is listed at the end of this report.

Table 1. Top five serovar of *Salmonella* notified to OzFoodNet sites, January to March 2001, by date of onset

State		First quarter 2000	Second quarter 2000	Total 2000	% change
New South Wales	Typhimurium 135	52	28	121	85.7
	Typhimurium 135a	28	3	13	833.3
	Typhimurium 9	38	44	136	-13.6
	Typhimurium 64	31	33	76	-6.1
	Birkenhead	26	23	76	13.0
Hunter PHU	Typhimurium 135a	7	0	0	
	Typhimurium 135	5	2	10	150.0
	Typhimurium 64	3	5	13	-40.0
	Typhimurium 9	3	2	3	50.0
	Typhimurium 44	2	0	0	
Queensland	Birkenhead	62	35	97	77.1
	Saintpaul	61	44	185	38.6
	Virchow 8	48	66	175	-27.3
	Typhimurium 135	42	57	127	-26.3
	Aberdeen	27	25	58	8.0
South Australia	Typhimurium 9	19	8	28	137.5
	Typhimurium 135	19	1	3	1800.0
	Typhimurium 126	15	0	4	
	Typhimurium 64	13	4	20	225.0
	Chester	9	6	18	50.0
Tasmania	Mississippi	56	36	73	55.6
	Typhimurium 9	3	7	22	-57.1
	Bovismorbificans	1	0	1	
	Enteriditis 1	1	1	1	0.0
	Meunchen	1	0	0	
Victoria[1]	Typhimurium 9	51	58	178	-12.1
	Typhimurium 135	43	23	68	87.0
	Typhimurium 4	37	7	38	428.6
	Virchow 34	16	37	60	-56.8
	Typhimurium 170	15	9	36	66.7

[1] Victorian data reported by date of receipt at the Victorian Department of Human Services.

South Australia recorded 77.3 per cent (17/22) of cases of Shiga toxin producing *E. Coli* (STEC) from OzFoodNet sites during the first quarter of 2001. The median age of these South Australian cases was 32.5 years (range 0-87 years), and the male to female ratio was 1:1. South Australian pathology laboratories intensively screen faeces containing blood for the presence of a gene that encodes for production of shiga toxin. Other States do not conduct similar intensive screening, and detecting toxin producing *E. coli* requires special culture media or the use of nucleic acid detection methods. These factors account for the relatively higher number of reports from South Australia.

The only other OzFoodNet sites to report STEC cases were Victoria (3 cases), and Queensland (2 cases). Despite the high number of cases reported from South Australia relative to other States, the total number reported in South Australia was decreased in the first quarter of 2001 compared to previous years.

There were 3 reports of haemolytic uraemic syndrome during the first quarter of 2001. Two of these were from Queensland and one was from Victoria. *E. coli* O111 was identified in one of these cases, but no toxigenic *E. coli* were isolated from the other 2 cases.

Foodborne disease outbreaks

During the quarter, OzFoodNet sites reported 27 outbreaks that were potentially related to food. These outbreaks affected approximately 402 people and a total of 28 people were hospitalised (Table 2). Some of these outbreaks were associated with previously recognised high-risk foods or food preparations and should have been preventable. An example of this was a small family cluster of rudderfish diarrhoea that occurred in the Hunter area of New South Wales. Similar outbreaks of rudderfish diarrhoea have previously occurred in Victoria and South Australia.

During the quarter, the Queensland OzFoodNet site reported a small cluster of 3 cases of *Campylobacter* infection that was associated with a chicken kebab shop. This is the third small outbreak of *Campylobacter* associated with take-away kebab shops in the last 2 years. These infections are easily prevented providing proprietors cook

the meat thoroughly, monitor the internal temperature of the meat, and handle foods correctly.

There were 2 outbreaks of salmonellosis associated with raw or undercooked eggs served to residents of aged care facilities during the quarter. One of these outbreaks was due to *Salmonella* Heidelberg 1 (Queensland), and the other to *Salmonella* Typhimurium 135 (South Australia). There is a high risk of *Salmonella* infection associated with the consumption of raw eggs, which should not be served to elderly people.

Surveillance improvements and applied research

The work of OzFoodNet during the quarter revealed several areas where surveillance and control activities for foodborne disease need improvement. These have included differences between States in the case definition for listeriosis, and delays in communicating *Salmonella* typing information. OzFoodNet will work with stakeholders, such as State and Territory health departments, the Communicable Diseases Network Australia (CDNA) and industry groups, to effect improvements.

Table 2. Outbreaks reported by OzFoodNet sites, first quarter 2001

State	Month	Setting	Agent responsible	No. exposed	No. affected	Responsible vehicles
Vic	Jan	Function	*S.* Typhimurium 170	30	14	Beef product suspected
	Jan	Restaurant	Norwalk	12	9	Unknown
	Feb	Restaurant	Unknown	9	5	Unknown
	Feb	Restaurant	Norwalk	159	65	Possible sausages
	Feb	Restaurant	Unknown (suspected Norwalk)	56	24	Unknown
	Mar	Home	Ciguatera toxin	17	13	Coral Trout
	Mar	Hotel	Unknown	36	9	Possible cheese platter, mushroom risotto, Thai prawns or combination
	Mar	Restaurant	Unknown	18	14	Unknown
SA	Dec 00- Jan 01	Restaurant	*S.* Typhimurium 29		8	Consumption of prepared food at the restaurant
	Nov 00-Jan 20		Rotavirus in one faecal specimen		11	
	Mar		*S.* Typhimurium 126		9	Custard fruit tart
	Jan		*S.* Typhimurium 185		5	
	Mar	Aged Care	*S.* Typhimurium 135	38	17	Raw egg (served in mince & potato pie & rice pudding)
WA	Mar	Remote mine	*S.* Typhimurium untyped	~300	29	Probably bore water supply
	Feb-Mar	Detention centre	*S.* Wandsworth	~1000	50[1]	Unknown
Tas	Feb		Unknown	10	9	Unknown
Qld	Jan	Home	Ciguatera	14	14	Spanish Mackerel
	Jan	Home	Ciguatera	2	2	Spotted Mackerel
	Jan	Restaurant	C. perfringens	25	9	Reef & Beef (possible sauce)
	Jan	Camp (Health Retreat)	Unknown	141	87	Drinking water
	Feb	Restaurant	Histamine Poisoning	4	4	Mahi Mahi
	Feb	Aged Care Facility	Unknown	Unknown	19	Unknown
	Feb	Wedding	Unknown	110	6	Unknown
	Feb	Aged Care Facility	*S.* Heidelberg PT 1	Unknown	12	Suspected raw egg drink
	Mar	Aged Care Facility	*S.* Muenchen	Unknown	3	Unknown
	Mar	Caterer	*S.* Virchow PT 8	Unknown	2	Chicken
	Mar	Take-away	C. jejuni	Unknown	3	Chicken kebabs

1. All cases in this outbreak were asymptomatic.

OzFoodNet epidemiologists in each site have developed plans for studies to further our knowledge about foodborne disease in Australia. These studies include:

- a national survey of diarrhoeal prevalence;
- four case control studies into risk factors for infections due to: *Campylobacter*, *Salmonella* Enteriditis and other locally-important serovars, *Listeria*, and STEC;
- a census of pathology laboratories for faecal testing practices;
- a register for foodborne outbreaks;
- laboratory sub-typing projects on *Campylobacter*;
- a retrospective review of foodborne disease across Australia; and
- enhanced surveillance through laboratories and general practitioners.

It is expected that these studies will commence within the next three months

OzFoodNet has only recently been established, but has developed into an extensive network of foodborne disease specialists. This has the potential to benefit investigations of foodborne disease that cross State and Territory boundaries. OzFoodNet sites will provide an estimate of the incidence of foodborne disease in Australia, and will work to improve investigation and control efforts.

OzFoodNet represents a significant investment in applied research into foodborne disease. It is important for the results of this work to become incorporated into policy formulation. The results of analytical studies will provide a useful insight into the occurrences of foodborne disease in Australia.

The OzFoodNet Working Group is (in alphabetical order):

Rosie Ashbolt (Tas), Louise Carter (ACT), Meredith Caelli (Hunter PHU), Scott Crerar (ANZFA), Craig Dalton (Hunter PHU), Rod Givney (SA), Joy Gregory (Vic), Gillian Hall (NCEPH), Brigid Hardy (AFFA), Geoff Hogg (MDU), Martyn Kirk (ANZFA), Vanessa Madden (Tas), Ian McKay (DHAC), David Peacock (NT), Nittita Prasopa-Plaizier (Vic), Paul Roche (DHAC), Russell Stafford (Qld), Elenor Sullivan (WA), Nola Tomaska (NCEPH), Tony Watson (WA), Leanne Unicomb (Hunter PHU)

ASPREN definitions 2001

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 national network of general practitioners who report on a number of conditions each week. The aim of ASPREN is to provide an indicator of the burden of disease in the primary health care setting and to detect trends in consultation rates.

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

For 2001, 12 conditions are being monitored, four of which are reported in *Communicable Diseases Intelligence* (*CDI*).

These include first attendance for an episode of influenza, influenza with culture, chickenpox and shingles.

The other recordable conditions are: chlamydia genital infection – requested by patient; chlamydia genital infection – suspected by doctor; depression; domestic violence; health care plan completed – no change of management; health care plan completed – change of management; antibiotic prescription for URTI; and patient request for antibiotics for URTI.

Data for communicable diseases are published every quarter in *CDI*. For each of the reporting weeks reviewed, the number of cases is presented in tabular form together with the rate of reporting per 1,000 consultations. Brief comments on the reports are included in the surveillance highlights section if appropriate. The case definitions are as follows:

Influenza

(a) Viral culture or serological evidence of influenza virus infection; or

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

(c) six of the following:

- sudden onset (within 12 hours);
- cough;
- rigours or chills;
- fever;
- prostration and weakness;
- myalgia, widespread aches and pains;
- no significant respiratory physical signs other than redness of nasal mucous membrane and throat;
- influenza in close contacts.

Influenza with culture

Defined as above with viral cultures or serological ordered.

Chickenpox

An acute, generalised viral disease with a sudden onset of slight fever, mild constitutional symptoms and a skin eruption which is maculopapular for a few hours, vesicular for 3-4 days, and leaves a granular scab.

Shingles

Recurrence, recrudescence or reactivation of chicken pox infection. Vesicles with an erythematous base restricted to skin areas supplied by sensory nerves of a single or associated group of dorsal root ganglia. Lesions may appear in crops in irregular fashion along nerve pathways, are usually unilateral, deeper seated and more closely aggregated than those of chickenpox.

Any questions concerning interpretation may be directed to:

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Details of the current quarterly ASPREN report are available in this issue (p175).

Annual report of the National Influenza Surveillance Scheme, 2000

Paul Roche,[1] Jenean Spencer,[1] Angela Merianos,[1] Alan Hampson[2]

Abstract

Surveillance of influenza in Australia in 2000 was based on data from national and state-based sentinel general practice consultations for influenza-like illness, laboratory isolations of influenza virus and absenteeism rates from a national employer. The peak in influenza cases was in mid-September. Influenza A was the dominant strain, with the highest proportion being influenza A (H3N2), but with a significant proportion of isolates of influenza A (H1N1) (16%) for the first time since 1995. The influenza A (H3N2) isolates were predominantly related to A/Moscow/10/99 and vaccine strain A/Panama/2007/99. Influenza A (H1N1) was predominantly A/New Caledonia/20/99. The proportion of Influenza B viruses isolated also increased in keeping with a three-yearly cycle of influenza B epidemics in Australia. influenza B isolates showed a progressive drift away from the B/Beijing/184/93 strain with the majority closely related to the B/Sichuan/379/99 strain. In 2000, influenza vaccination levels reached 74 per cent in persons aged over 65 years. *Commun Dis Intell* 2001;25:107-112.

Keywords: influenza, surveillance, vaccine, general practice, strains

Introduction

Influenza is an acute, self-limiting upper respiratory tract infection. Complications however, may occur, including lower respiratory tract infection (in particular primary and secondary pneumonia, exacerbation of chronic obstructive pulmonary disease) and exacerbation of cardio-pulmonary disease.[1] Influenza-related morbidity (measured as excess hospitalisation) and mortality may result from these complications. Although influenza infection affects all age groups, the rates of serious morbidity and mortality tend to be highest among those aged 65 years and over, indigenous Australians and those with chronic medical problems. Young infants and pregnant women are also at increased risk of hospitalisation from influenza.

Outbreaks of influenza usually occur during winter months in temperate climates (peaking between December and March in the Northern Hemisphere and June and September in the Southern Hemisphere), but may occur throughout the year in tropical regions. Even though the complication rate may be low, the overall high attack rate during epidemics leads to a considerable increase in hospitalisations and mortality. In Australia in 1998, pneumonia and influenza accounted for 4,579 deaths (Australian Bureau of Statistics, 2001). Influenza pandemics occur every 10 to 30 years. During these pandemics, a quarter or more of the global population may be affected within a short period and the rates of illness and death from influenza can increase dramatically.

Influenza viruses are successful human pathogens because of their ability to vary their two external proteins, haemagglutinin (H) and neuraminidase (N). Mutations cause a gradual change in these proteins called ‘antigenic drift’, which results in annual epidemics of influenza. The greater the change in these proteins, the less likely it is that the virus will be recognised by immune cells primed by exposure to earlier infections or vaccines, and the greater the epidemic potential. At irregular intervals, there are more dramatic changes in the viral proteins, called ‘antigenic shift’, which are a result of either direct introduction of avian influenza viruses into the human population or a re-assortment of avian viruses in an intermediate host such as pigs. These ‘shifts’ result in the emergence of a new influenza virus. In the absence of immunity to these new viruses, there is rapid spread of influenza with dramatically increased rates of morbidity and mortality. The pandemic of 1918 introduced the H1N1 virus into the human population and the 1968 Hong Kong pandemic introduced the H3N2 virus. There have been no major ‘antigenic shifts’ causing pandemics of influenza since 1968. Since 1977, influenza A (H1N1 and H3N2) and influenza B viruses have been widespread globally, varying in frequency temporally and geographically.[2]

In Australia, influenza vaccines are produced after analysis of the dominant strains in the previous year’s influenza cases. Influenza vaccination is recommended to non-indigenous Australians aged 65 years and above and indigenous Australians aged 50 years and above.[3]

An effective national surveillance system is an essential component of a program for the control of influenza. Influenza surveillance aims to ensure the provision of timely information about levels of influenza activity and circulating strains, to public health departments, health care providers and the general public. The major objectives of such surveillance include:

- early detection of epidemics to enable the implementation of public health measures such as vaccination of the 'at risk' groups, control campaigns and provision of clinical services;
- characterisation of the epidemic;

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- isolation and antigenic characterisation of circulating influenza viruses to assist in the formulation of the following season's vaccine; and
- evaluation of the impact of the epidemic and associated public health measures.

This annual influenza report provides a summary of the surveillance methods and data for 2000.

Surveillance methods

Surveillance of influenza in Australia is based on 4 sets of data:

- laboratory diagnosis including virus isolation and serology by laboratories participating in the LabVISE (Laboratory Virology and Serology) Reporting Scheme);
- data on subtypes found among isolates from LabVISE laboratories, provided by the WHO Collaborating Centre for Reference and Research on Influenza;
- consultation rates for influenza-like illness diagnosed by sentinel general practitioners; and
- absenteeism data of workers from a national employer.

Laboratory-confirmed influenza became nationally notifiable from January 2001. The States and Territories will forward notifications to the Commonwealth through the National Notifiable Diseases Surveillance System (NNDSS), as their legislation and IT systems are updated.

Laboratory surveillance (LabVISE)

LabVISE is a national scheme of Australia-wide sentinel laboratories. In 2000, a total of 12 laboratories contributed to this scheme although not all provided reports each month. Laboratory reports of influenza are sent to LabVISE all year round. Although viral isolation remains the gold standard for influenza diagnosis and surveillance, most reports have relied on the detection of viral antigen and serological markers. Nucleic acid detection by the polymerase chain reaction (PCR) is now in used for diagnosis.[2]

WHO Collaborating Centre for Reference and Research on Influenza

The WHO Collaborating Centre for Reference and Research on Influenza contributes reports on the subtypes and antigenic analysis of influenza viruses isolated throughout the year in Australia. This information is used to monitor the nature of influenza strains present in Australia and the rest of the world, assess suitability of the current vaccine (by measuring the level of matching between circulating strains and the current vaccine) and determine the composition of vaccine for the following influenza season. Influenza viruses are named after the places where they were first identified. For example, A/Sydney/5/97 was first isolated in Sydney in 1997 and was influenza A isolate number 5 for that year.

Sentinel general practitioner surveillance

Sentinel general practitioner surveillance schemes detect and record clinical diagnoses of influenza-like illness. The Australian Sentinel Practice Research Network (ASPREN) collects data at a national level. In addition, data are collected through the New South Wales Sentinel General Practice Scheme, the Victorian Sentinel General Practice Scheme and the Northern Territory Tropical Influenza Surveillance Scheme.

The New South Wales and Victorian schemes report cases of influenza-like illness from the beginning of May to September each year. ASPREN and the Northern Territory schemes report throughout the year. ASPREN is the only sentinel surveillance scheme that reports on influenza-like illness from sentinel general practices located throughout Australia.

Of sentinel general practices contributing to the ASPREN scheme, most are located in capital cities and larger regional centres, mostly on the east coast of Australia. Between 7000 and 8000 consultations are recorded each week. Participation is voluntary in all sentinel general practice surveillance systems, leading to variation in the number of contributors. In 2000 the number of contributing practices varied from 52 to 77 per reporting period for ASPREN, 8 to 41 for the New South Wales scheme, 25 to 47 for the Victorian scheme and from 9 to 14 for the Northern Territory scheme.

The case definition for a clinical diagnosis of an influenza-like illness varies between the different sentinel general practice surveillance schemes (Box).

Absenteeism surveillance

Australia Post, a major nation-wide employer, provided de-identified sick leave absenteeism data during 2000 between weeks 10 and 36 (from March to September). Absenteeism was defined as an absence due to illness for at least 3 consecutive days. This definition was used to increase the specificity for absenteeism related to influenza infection. Absenteeism was reported as the rate per 100 employees and rates were calculated on a weekly basis.

Antigenic analysis of influenza virus isolates

The WHO Collaborating Centre for Reference and Research on Influenza identifies circulating strains of influenza by genetic sequence analysis of the variable region of major surface antigen, (haemagglutinin), and of the minor surface antigen, (neuraminidase) on a sample of isolates forwarded to the centre.

Hospitalisation data

To assess the impact of influenza on hospitalisation, the Australian Institute of Health and Welfare (AIHW) made available hospital separation data and average length of stay data for public and private hospitals. Data for the 1999/2000 financial year was the most recent available at the time of writing this report. Information was assessed by the ICD-10AM code that classifies influenza under 2 categories: cases of influenza where the virus is identified (J10) and cases where the virus is not identified (J11).

During the influenza season, data from laboratories and sentinel GP practices are posted on the Communicable Diseases Australia Website, which includes links to data on influenza activity in New Zealand and information on circulating influenza strains from the WHO Collaborating Centre for Reference and Research on Influenza.

Results

The influenza surveillance data presented here are limited and should be interpreted with caution. Laboratory confirmed influenza are a small proportion of all influenza cases in the year and consequently the estimation of the circulating strains is based on a small sample. Definitions of influenza-

Box. Case definitions of influenza-like illness used in Australian Sentinel Practices

The case definition for ASPREN, and for the Victorian and Northern Territory Sentinel GP schemes is:

- viral culture or serological evidence of influenza virus infection; or
- influenza epidemic, plus four criteria listed below; or
- six of the following criteria:
  - sudden onset (within 12 hours);
  - cough;
  - rigours or chills;
  - fever;
  - prostration and weakness;
  - myalgia, widespread aches and pains;
  - no significant respiratory physical signs other than redness of nasal mucous membranes and throat;
  - influenza in close contacts.

The definition of influenza used by the New South Wales sentinel GP scheme is:

- cough and;
- myalgia; and
- no abnormal respiratory physical signs other than redness of nasal mucous membranes and throat; and
- two of the following:
  - sudden onset;
  - rigours or chills of fever;
  - prostration or weakness; or
  - influenza in close contacts.

like-illness vary between sentinel practices (Box) which make comparisons difficult. In addition definitions of influenza-like illness have varied from year to year, so comparison of data across years is complex. Absenteeism data are currently based on a three-day absence. Data collected before 1996 however, were based on a single day absence. In summary, surveillance data are currently unable to measure severity of annual epidemics or to monitor yearly variations in severity.

Laboratory surveillance (LabVISE)

In 2000, a total of 1916 laboratory isolations of influenza were made in participating laboratories of the LabVISE reporting scheme. These were 1366 reports of influenza A and 550 reports of influenza B. The ratio of influenza A to B was 2.5:1.

Total influenza reports showed a low level of activity until mid-June (week 24) when there was an increase in reports to approximately 50 per week, followed by a major peak in mid-September (week 37), then a decline to baseline by late November (week 47, Figure 1). There were little temporal differences in the peaks of influenza A compared with influenza B activity throughout the year. The peak of influenza activity in 2000 was significantly later in the year than in 1999 (Figure 2).

The seasonal pattern of influenza between 1996 and 2000 is shown in Figure 3. The pattern in 2000 closely resembled that in 1997 when there was also a larger proportion of influenza B isolates (influenza A to B ratio of 1.5:1), and a later peak in disease reporting.

The breakdown of influenza cases by age and sex is shown in Figure 4. The overall male to female ratio for influenza in

Figure 1. Laboratory reports of influenza, Australia, 2000, by type and week of specimen collection

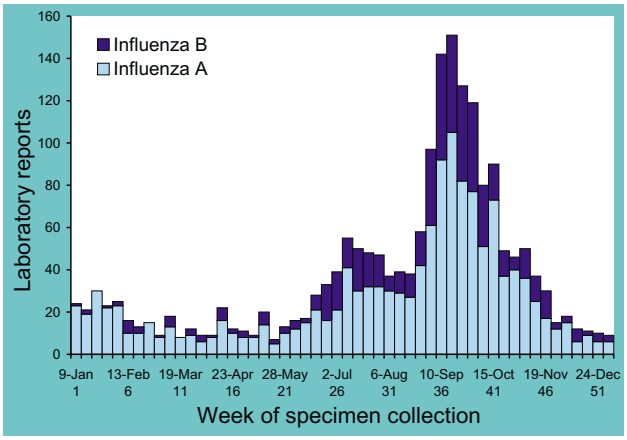


Figure 2. Laboratory reports of influenza, Australia, 1999 and 2000, by month of specimen collection

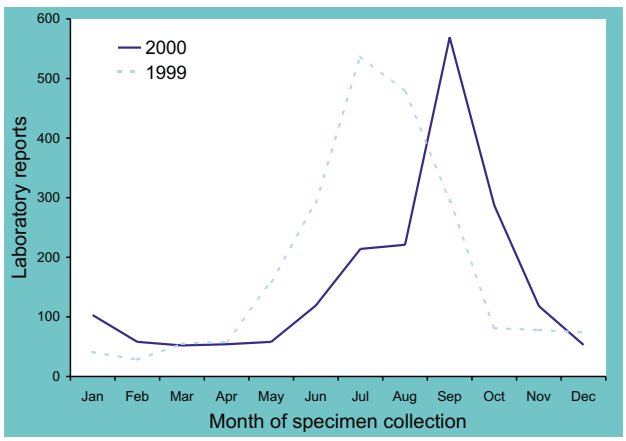


Figure 3. Laboratory reports of influenza, Australia, 1996 to 2000, by type and month of specimen collection

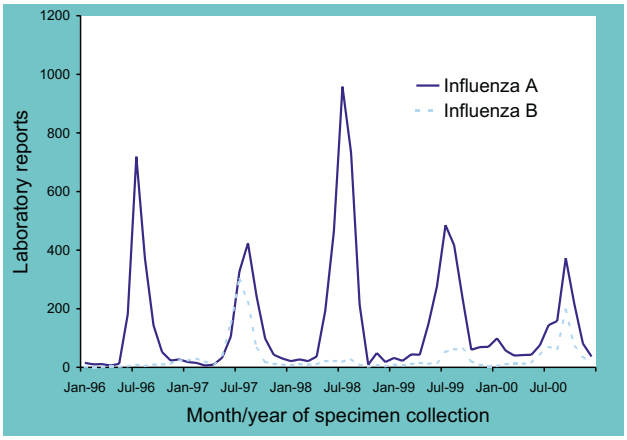
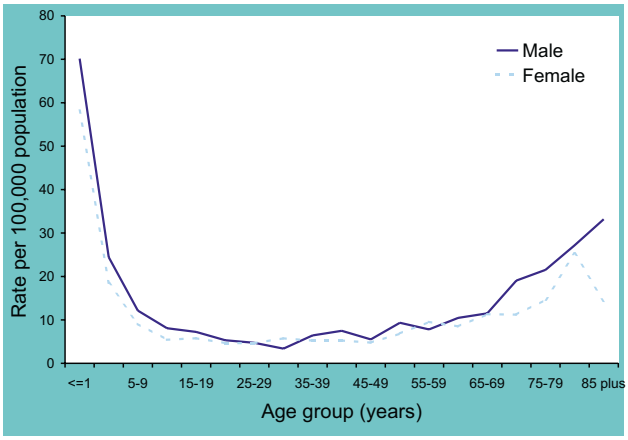


Figure 4. Rates of laboratory-confirmed influenza, Australia, 2000, by age and sex



2000 was 1.2:1. The age- and sex-specific rates were highest among infants and children aged less than 5 years, with a second peak among men aged 70 years or more and women aged 75 years or more.

A breakdown of weekly laboratory reports for influenza A by State and Territory (as defined by postcode) for New South Wales, Victoria and Western Australia is shown in Figure 5. The influenza A activity in the eastern States (New South Wales and Victoria) began to rise in week 34, one week earlier than in Western Australia. Both eastern and western States of Australia reported their peak influenza A notification in week 37.

Sentinel general practice (GP) surveillance

Sentinel surveillance data were available from the Northern Territory, New South Wales and Victoria, in addition to the nation-wide surveillance of ASPREN. The Northern Territory Tropical Influenza Surveillance scheme data showed 2 peaks of influenza activity in weeks 9 (week ending 5 March) and week 42 (week ending 22 October). The ASPREN data and that of the New South Wales and Victorian sentinel schemes (Figures 6 and 7) all showed a single peak in reporting at week 37 (week ending 17 September).

Figure 5. Laboratory reports of influenza A, New South Wales, Victoria and Western Australia, 2000, by week of specimen collection

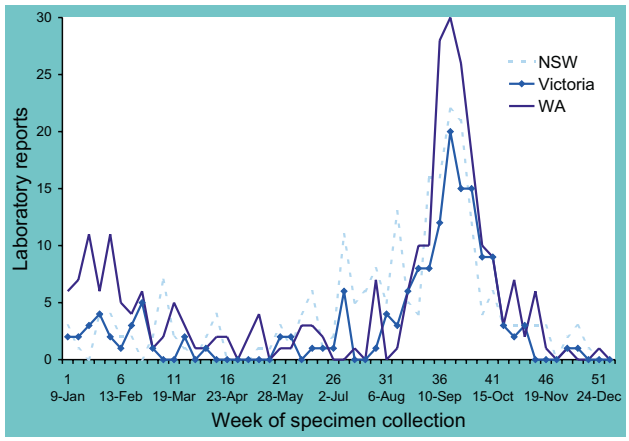


Figure 6. Consultation rates for influenza-like illness, Australia (ASPREN) and Northern Territory, 2000, by week of report

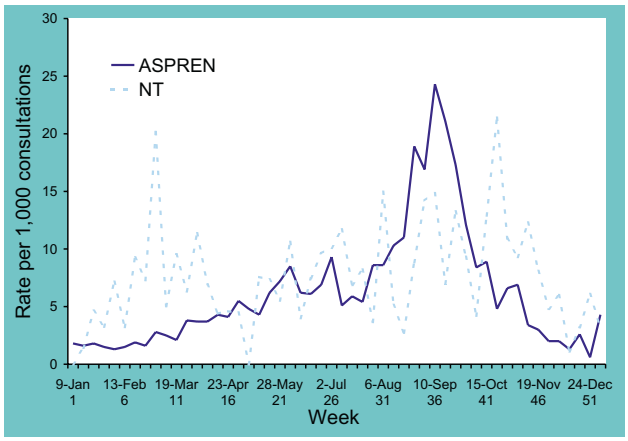
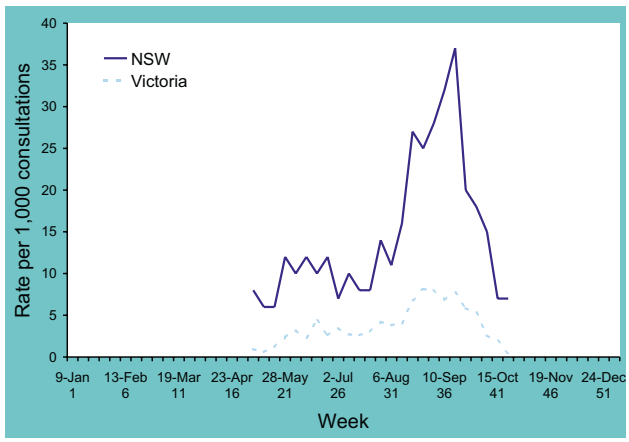


Figure 7. Consultation rates for influenza-like illness, New South Wales and Victoria, 2000, by week of report



Comparison of the ASPREN and LabVISE reports showed a similar pattern of activity, with the peak in laboratory reports one week later than that from general practitioner surveillance (Figure 8).

Figure 8. Laboratory reports of influenza and national consultation rates for influenza-like illness, Australia, 2000, by week of specimen or report

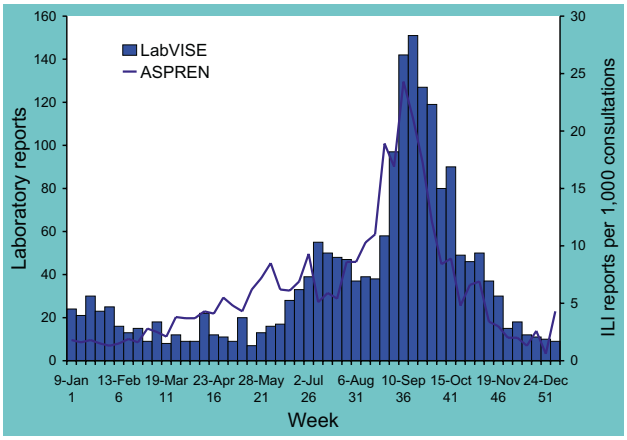
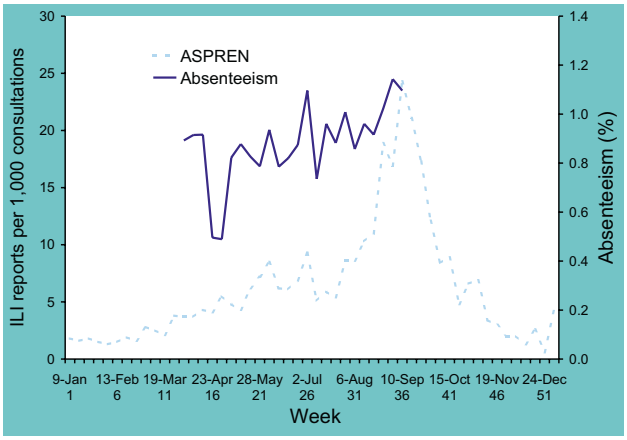


Figure 9. Rates of absenteeism and consultation rates for influenza-like illness, Australia, 2000, by week of report



Absenteeism surveillance

There was little evidence of any association between absenteeism and the peak in influenza activity in the data supplied by Australia Post. Data were only available up to week 36, which was one week before the peak in laboratory notification of influenza in 2000. Absenteeism was highest at 1.1 per cent in weeks 26, 35 and 36 (Figure 9).

WHO Collaborating Centre for Reference and Research on Influenza

The Centre received a total of 1116 influenza isolates of which 922 (83%) were viable and able to be analysed antigenically. For 97 of those isolates, genetic sequence analysis of the variable region of major surface antigen, haemagglutinin, was also undertaken, and for 22, genetic analysis of the minor surface antigen, neuraminidase, was also performed.

Of the viable isolates 518 (56%) were influenza A (H3N2) subtype, 262 (28%) were influenza B and the remaining 142 (16%) were influenza A (H1N1) subtype. The influenza A (H1N1) isolates were predominantly A/New Caledonia/20/99-like viruses (73%) with only 39 isolates characterised as A/Bayern/7/95-like. These 2 separate lineages of viruses have co-circulated for some time. Although 3 sporadic isolates of the A/New Caledonia lineage were isolated in 1999 this is the first year in which viruses of the lineage have been isolated in significant numbers in Australia. All but one of the 39 A/Bayern/7/95-like isolates came from an outbreak in South Australia.

The majority of the influenza A(H3N2) isolates (94%) were most closely related to the reference strain A/Moscow/10/99 and vaccine strain A/Panama/2007/99 and were distinguishable from the previous prototype and vaccine strain A/Sydney/5/97. Nevertheless, serological studies demonstrated that vaccines containing an A/Sydney/5/97-like strain used in the Australian 2000 winter produced similar antibody responses to the Australian 2000 A (H3N2) isolates as did vaccines containing an A/Moscow/10/99-like strain used in the 2000-2001 Northern Hemisphere winter.[4] While some antigenic heterogeneity was observed in the A (H3N2) isolates there was no evidence of significant antigenic drift beyond the A/Moscow/10/99 reference strain. Influenza B strains isolated during the 2000 season showed a progressive drift away from the B/Beijing/184/93 strain. The majority (64%) was most closely related antigenically to the new reference strain B/Sichuan/379/99.

Hospitalisation due to influenza

In 1999/2000, there were a total of 2591 admissions to Australian hospitals for influenza. Six hundred and seventy-three of these were cases in which the influenza virus was identified. Altogether influenza was responsible for 4,583 hospital patient days in 1999/2000. These data do not cover the full period of the 2000 influenza season.

Discussion

Surveillance issues

Surveillance of influenza in Australia depends on a network of different schemes including laboratory notifications, sentinel GP reports (national and state-based) and absenteeism reporting from a major national employer. In any year, changes in the reporting practices in any of these schemes will influence the estimate of influenza disease in Australia. LabVISE provides data on laboratory isolates of the influenza virus. Over the past few years, reporting through this scheme for all diseases has decreased as the number of participating laboratories has declined. In 2000, 12 laboratories reported 1916 isolates compared with 13 laboratories, which reported 3247 isolates in 1999, and 21 laboratories reporting between 1150 and 2943 isolates between 1995 and 1998. The numbers from LabVISE along with all other surveillance schemes must be interpreted as an estimate only of the total influenza incidence in Australia. LabVISE will be reviewed in 2001 and improvements in reporting through LabVISE should enhance the laboratory detection of influenza in Australia thereafter. In 2001, laboratory-confirmed cases of influenza will become notifiable in all Australian States and Territories and will be reported nationally by the NNDSS. This is expected to impact on the total numbers of influenza cases detected in Australia in the coming years.

In 2000, there was good agreement between the different surveillance schemes in the temporal trends in influenza throughout the year. Compared to 1999, influenza reports peaked relatively late in the year (mid-September). Both laboratory and sentinel GP reports showed the same time

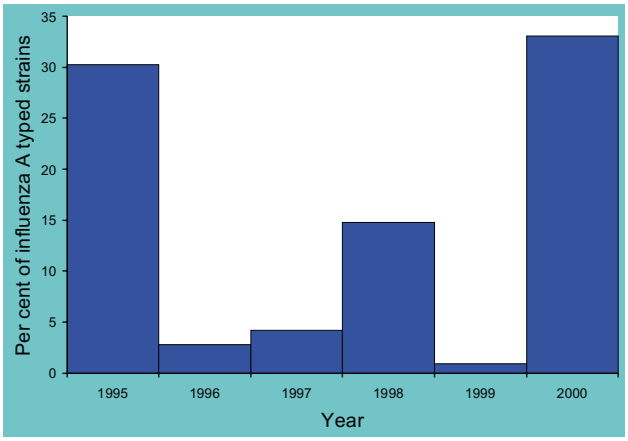
trends and there were little differences in these trends between jurisdictions.

The age-specific rates of laboratory-confirmed influenza showed a high rate in infants and in people aged 70 years or over. In these age groups there is more severe disease and a greater prevalence of co-morbidity, particularly with pneumonia. Age-specific rates should be interpreted with caution since medical attention is more likely to be sought for the very young and the very old, and influenza may be mild in healthy older children and adults.

Trends in strain frequencies

The re-appearance of H1N1 influenza strains in Australia reflects the situation seen in other parts of the world as shown in Figure 10 (FluNet data, WHO). H1N1 strains were a small minority of typed strains worldwide (1995 to 1999), but data from 2000 shows an increase to 30 per cent, in the proportion of H1N1 strains isolated.

Figure 10. Frequency of influenza A/H1N1 strains, worldwide, 1995 to 2000



The clustering of A/Bayern/7/95-like virus in South Australia illustrates the local clustering of various strains occasionally observed. This strain of influenza A has been more widely in circulation in previous years.

Influenza B isolates increased in 2000, which is consistent with a three-yearly cycle of influenza B peaks in Australia. The last peak of influenza B in Australia was in 1997 (Figure 4). Peaks of influenza A and B were within a week of each other (weeks 37 and week 36 respectively, Figure 2).

Influenza vaccine use in Australia 2000

In 2000 the Centre for Population Studies in Epidemiology, South Australian Department of Human Services, performed a review of influenza vaccine uptake on behalf of the Commonwealth Department of Health and Aged Care. Telephone interviews conducted from August to November 2000 with 10,505 Australians showed 74 per cent of Australians aged 65 years and above had been vaccinated in 2000. The level of vaccination has increased from 61 per cent in 1998 and 70 per cent in 1999. The report showed some significant differences in levels of vaccination in elderly Australians in 2000 between jurisdictions, with the Australian Capital Territory having the highest coverage (82.4%) and Queensland the lowest (69.6%). The rates of vaccination in each of the past 3 years for elderly Australians was 57.4 per cent - this varied from 47.5 per cent to 64.7 per cent in different States and Territories. Ninety-five per cent of elderly Australians received influenza vaccination free of charge in 2000.[5]

Other influenza reports in Australia, 2000

In the late-summer of 2000, there was a report of an outbreak of influenza A on a trans-Tasman cruise ship.[6] In this outbreak, 8 per cent of passengers and 4 per cent of the crew presented with influenza-like illness (a total of 108 presentations). Only 2 throat swabs were positive for influenza A, which was identified in one of the cases as an H3N2 strain. This report complements other reports of influenza outbreaks among passengers on cruise ships in the Northern Hemisphere.

An analysis of laboratory-supported influenza surveillance in Victoria, which examined the relationship between influenza-like illness and laboratory-confirmed influenza, was published in 2000.[7] The proportion of patients presenting to sentinel GP practices with laboratory-confirmed influenza- like illness varied from 49 to 54 per cent in 1998 and 1999 respectively.

World trends

The 1999-2000 influenza season in the USA was dominated by influenza A (H3N2), most commonly the influenza A/Sydney/05/97-like strains. This was the third consecutive year in which this was the dominant virus strain and is well matched to the influenza vaccine strain.[8] In Europe, the influenza A (H3N2) strain circulated widely and caused one of the 3 largest epidemics in the past 10 years in France, Great Britain and Italy.[9]

The WHO has recommended the content of the Northern Hemisphere influenza vaccine for the 2001/2002 influenza season contain an A/New Caledonia/20/99 (H1N1)-like virus, an A/Moscow/10/99 (H3N2)-like virus and a B/Sichuan/379/99-like virus.[4] This same recommended vaccine will be in use in Australia for the 2001 influenza season.

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Annual report of the Australian Meningococcal Surveillance Programme, 2000

The Australian Meningococcal Surveillance Programme

Abstract

The National Neisseria Network has undertaken meningococcal isolate surveillance by means of a collaborative laboratory based initiative since 1994. The phenotype (serogroup, serotype and serosubtype) and antibiotic susceptibility of 388 isolates of *Neisseria meningitidis* from invasive cases of meningococcal disease were determined in 2000. More than 90 per cent of the invasive isolates were either serogroup B or C. There was however, considerable diversity in the phenotypes circulating in the different States and Territories. Serogroup B strains predominated in all jurisdictions except Victoria and were isolated from sporadic cases of invasive disease. Serogroup B phenotypes were generally disparate although phenotypes B:15:P1.7 and B:4:P1.4 were widely distributed. The latter remained especially prominent in New South Wales. The number and proportion of serogroup C isolates again increased in Victoria compared with previous years. Infections with a novel phenotype that was first noted in 1999, C:2a:P1.4(7), were common in Victoria, especially in adolescents and adults, but rarely seen elsewhere in Australia. Phenotype C:2a:P1.2, was also noted in the preceding year and continued to be seen in Victoria in 2000 but was infrequently encountered in other jurisdictions. Serogroup C infections remained common in New South Wales where phenotype C:2a:P1.5 was regularly isolated. About two thirds of all isolates showed decreased susceptibility to the penicillin group of antibiotics (MIC 0.06 to 0.5 mg/L). All isolates tested were susceptible to third generation cephalosporins and to the prophylactic agents rifampicin and ciprofloxacin. Data relating to 147 laboratory-confirmed but culture-negative cases, supplemented information on culture-confirmed cases in this report. Some differences in the patterns of disease were revealed when culture-based and non-culture-based data were compared. *Commun Dis Intell* 2001;25:113-121.

Keywords: surveillance, Neisseria meningitidis*, meningococcal disease, antibiotics, penicillin*

Introduction

There is perennial interest in invasive meningococcal disease (IMD) from a public health and general community perspective. The common clinical manifestations of IMD are septicaemia and/or meningitis. Single organ disease such as arthritis occurs less frequently. Presentations may range from the mild and subclinical to the rapidly progressive and fatal. While the host response affects the ultimate outcome in individual cases, both this result and the patterns of the infection within a community may be altered by certain features of the infecting organism.[1,2]

The characteristics of the meningococci prevalent in a population and responsible for infections in individuals also influence the public health response to IMD. These features may be used to confirm or exclude the presence of an outbreak or cluster of cases suspected on clinical grounds, and to influence the public health measures used to control such an outbreak. For example, the presence of different subtypes of meningococci excludes case clustering if this is suspected epidemiologically. Currently, polysaccharide vaccines are available for some serogroups of meningococci but not for others. The imminent availability of a conjugate serogroup C vaccine will mean that decisions on its application will be affected by the pattern of IMD in Australia and the subtypes causing disease.

The Australian Meningococcal Surveillance Programme, for the examination of isolates of *Neisseria meningitidis* from cases of IMD, was commenced in 1994 through the collaboration of reference laboratories in each State and Territory. This laboratory-based activity is designed to supplement data from existing clinical notification schemes by adding information on the phenotype (the serogroup, the serotype and subserotype), on occasion the genotype, and the antibiotic susceptibility of invasive isolates to clinical data. Annual reports summarising data gathered since the inception of the Programme have been published in *Communicable Diseases Intelligence*.[3-8] The following report analyses the characteristics of meningococci isolated in the calendar year 2000. Non-culture-based laboratory testing, based on nucleic acid based amplification assays and serology, is increasingly used to confirm IMD.[9,10] This report includes data from IMD confirmed by these means.

Methods

The National Neisseria Network (NNN) is a collaborative program for the laboratory surveillance of the pathogenic Neisseria *N. meningitidis* and *N. gonorrhoeae*.[3-9] A network of reference laboratories in each State and Territory (see acknowledgments) undertakes meningococcal isolate surveillance.

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Isolate based surveillance

Each case was based upon isolation of a meningococcus from a normally sterile site. Information on the site of infection, the age and sex of the patient and the outcome (survived/died) of the infection was sought. The isolate surveillance subset of the Programme categorises cases on the basis of site of isolation of the organism. Where an isolate is grown from both blood and CSF cultures in the same patient, the case is classified as one of meningitis. It is recognised that the total number of cases was under-estimated. This particularly applies to the number of cases of meningitis, where there was no lumbar puncture or else where lumbar puncture was delayed and the culture sterile. , The above approach however, has been used since the beginning of this program and is continued for comparative purposes.

Phenotyping of invasive isolates of meningococci by serotyping and serosubtyping was based on the detection of outer membrane protein antigens using a standard set of monoclonal antibodies obtained from the National Institute for Public Health (RIVM), the Netherlands.

Antibiotic susceptibility was assessed by determining the minimal inhibitory concentration (MIC) to antibiotics used for therapeutic and prophylactic purposes. This program uses the following parameters to define the various levels of penicillin susceptibility/resistance when determined by a standardised agar plate dilution technique.[9]

- sensitive, MIC 0.03 mg/l;
- less sensitive, MIC 0.06 - 0.5 mg/l;
- relatively resistant MIC 1 mg/l.

Strains with MICs which place them in the category of 'sensitive' or 'less sensitive' would be considered to be amenable to penicillin therapy when used in currently recommended doses.

Non-culture-based laboratory-confirmed cases

Additional laboratory confirmation of suspected cases of IMD is increasingly available by means of non-culture-based methods such as nucleic acid based amplification assays (NAA) and serological techniques. NAA testing is essentially by polymerase chain reaction (PCR) techniques.[10] Data from the results of these investigations were included for the first time in the 1999 report and are again reported here. The serological results are based on results of tests performed using the methods and test criteria of the Manchester Public Health Laboratory Service (PHLS) reference laboratory, UK and assessed for Australian conditions.[11] Age, sex and outcome data for patients with non-culture-based diagnoses are recorded when available. The site of a sample of a positive PCR test is used to define the clinical syndrome. This separation is not possible for cases diagnosed serologically.

Results

Numbers of isolates from culture-confirmed cases

A total of 388 invasive isolates of meningococci were examined in 2000. There were 141 isolates from patients whose infections were acquired in New South Wales (36% of all isolates), 108 (28%) from Victoria, 50 (13%) from Western Australia, 43 (11%) from Queensland, 20 (5%) from South Australia, 14 (3%) from Tasmania, 7 (2%) from the Northern Territory and 5 (1%) from the Australian Capital Territory (Table 1).

Seasonality

Seventy-one (19%) infections were identified between 1 January and 31 March 2000, 96 (25%) between 1 April and 30 June, 125 (33%) between 1 July and 30 September and 94 (25%) between 1 October and 31 December. A winter peak of meningococcal disease is usual.

Age group

The age distribution of patients infected with invasive isolates in each State and Territory is shown in Table 2. As in previous years the national peak incidence of meningococcal disease occurred in those 4 years and under. Of the 388 total cases, 47 (12%) were aged less than one year, and 73 (19%) were in the 1-4 year age group. A secondary peak was identified in adolescents and young adults. Of the 388 cases 79 (20%) were identified in the 15-19 year age group, and a further 47 cases (12%) occurred in those aged 20-24. Overall, there were 127 (32%) cases in the 15-24 year age

Table 1. *Neisseria meningitidis* isolates, 2000, by State or Territory and serogroup

State/ Territory	Serogroup												
	B		C		A	Y		W135		NG*		Total	
	n	%	n	%		n	%	n	%	n	%	n	%
ACT	5	100.0	0	0.0	0	0	0.0	0	0.0	0	0.0	5	1.3
NSW	74	52.5	55	39.0	0	7	5.0	3	2.1	2	1.4	141	36.3
NT	6	85.7	1	14.3	0	0	0.0	0	0.0	0	0.0	7	1.8
Qld	31	72.1	10	23.3	0	0	0.0	1	2.3	1	2.3	43	11.1
SA	12	60.0	5	25.0	0	1	5.0	1	5.0	1	5.0	20	5.2
Tas	9	64.0	4	28.0	0	0	0.0	0	0.0	1	8.0	14	3.6
Vic	40	37.0	58	53.7	0	5	4.6	4	3.7	1	1.0	108	27.8
WA	40	80.0	10	20.0	0	0	0.0	0	0.0	0	0.0	50	12.9
Total	217	56.0	143	37.0	0	13	3.2	9	2.3	6	1.5	388	100

*NG = not viable for serogrouping or not serogroupable

Table 2. *Neisseria meningitidis* isolates, 2000, by State or Territory and age

	Age group (years)										
	<1	1-4	5-9	10-14	15-19	20-24	25-44	45-64	65+	NS	All
ACT	0	2	0	1	1	0	1	0	0	0	5
NSW	13	24	13	7	39	14	13	14	3	1	141
NT	2	1	0	0	0	2	1	0	0	1	7
Qld	7	8	3	4	8	7	4	1	1		43
SA	4	5	1	1	5	1	0	2	1	0	20
Tas	4	2	0	0	1	1	1	1	0	4	14
Vic	9	18	5	7	18	17	17	12	5	0	108
WA	8	13	5	2	7	5	2	4	4	0	50
Total (n)	47	73	27	22	79	47	39	34	14	6	388
%	12.1	18.8	7.0	5.7	20.3	12.1	10.0	8.8	3.6	1.6	100

NS. Age not specified

group which was unusually high compared to previous years. This peak in the distribution was particularly noticeable in Victoria and New South Wales.

Serogroup, serotype and serosubtype (phenotype) distribution

The distribution of the isolates by serogroup is shown in Table 1. Nationally, 217 serogroup B isolates represented 56 per cent of all strains, a lower proportion than in the previous 3 years. The 143 serogroup C strains (37%) represented a further increase on the number (128) and proportion (33%) detected in 1999. The number (13) and proportion (3.2%) of serogroup Y strains did not change significantly. Nine serogroup W135 meningococci were identified. No serogroup A isolates were encountered.

Some important differences in the distribution of serogroups were evident when data were disaggregated by region. Serogroup B predominated in national data (56%) and in all jurisdictions except Victoria. When examined regionally, Western Australia (80% of isolates), the Australian Capital Territory (100%), South Australia (70%), the Northern Territory (85%), Queensland (72%) and Tasmania (64%) had high proportions of serogroup B strains. In New South Wales the 74 group B strains accounted for 52 per cent of isolates. In Victoria however, serogroup B isolates represented 37 per cent of the total. Group B disease comprised unlinked and apparently sporadic cases.

Figure 1b. Distribution (%) by age of serogroup B and C infections, Victoria, 2000

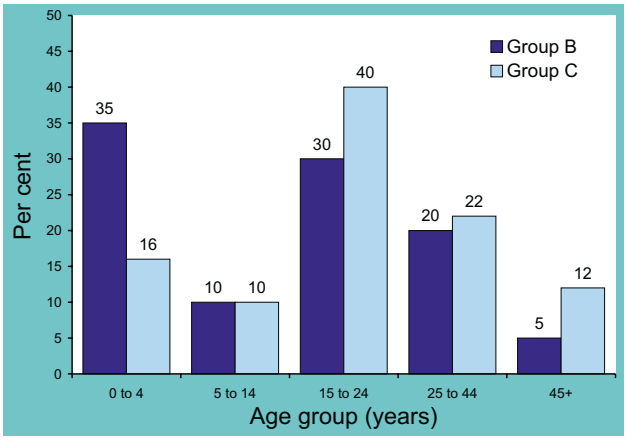


Figure 1a. Distribution (%) by age of serogroup B and C infections, centres other than Victoria and New South Wales, 2000

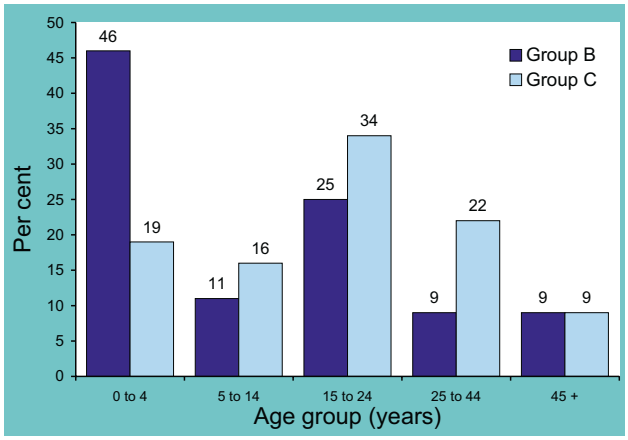
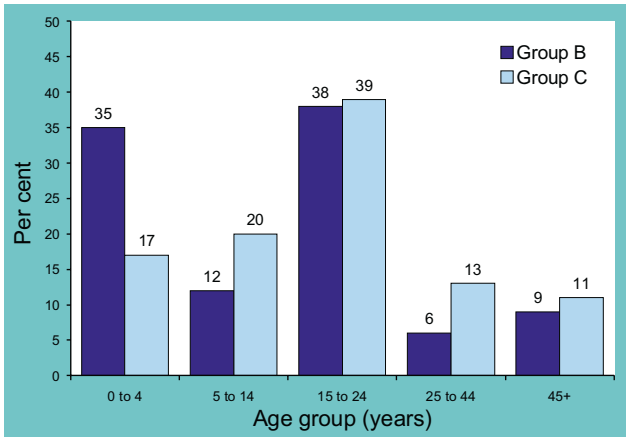


Figure 1c. Distribution (%) by age of serogroup B and C infections, New South Wales, 2000



A further increase in serogroup C infections in 2000, occurred in Victoria where 53 per cent of invasive isolates were serogroup C. The number of group C isolates increased from 42 (45% of the total) to 58 in 2000. In 1998 there were only 7 (17.5%) serogroup C strains identified in Victoria. Serogroup C isolates were also common in New South Wales in 2000 and the 55 isolates represented 39 per cent of the total, a similar proportion to that identified in 1999 (37%). One hundred and thirteen group C meningococci (79 per cent of all serogroup C strains isolated in Australia) were from cases residing in New South Wales and Victoria. The proportion of total strains identified as group C strains was lower in other States and Territories. There were 10 group C isolates (23%) in Queensland, 5 (20%) in South Australia, 10 (20%) in Western Australia and 4 (28%) in Tasmania but none in the Australian Capital Territory.

Table 3. Commonly isolated serotypes and serosubtypes and phenotypes of *N. meningitidis* of interest, 2000, by State and Territory

	Serogroup B				Serogroup C			
	Serotype	N	Serosubtype	N	Serotype	N	Serosubtype	N
ACT	4	3	1.4	1				
	15	1	1.7	1				
NSW	4	32	1.4	20	2a	46	1.5	22
			1.7	3			1.5,2	10
			1.14	4			1.2	3
			1.5	1			nst	11
			nst*	1			1.16	1
	15	9	1.7	9	2b	3	1.2	1
	14	8	1.4	2			nst	2
			nst	6	NT	5	1.5	1
	NT*	20	1.4	4			1.5,2	3
			1.15	3			1.15	1
			nst	8				
NT	15	1	1.7	1	2a	1	1.5	1
	NT	1	nst	1				
	14	3	nst	3				
Qld	15	8	1.7	4	2a	5	1.5	3
	4	1	nst	1			1.7	1
	NT	20	1.4	9	2b	1		
			nst	5	NT	3		
SA	4	2	1.4	1	2a	4	1.4	2
	1	1	nst	1			1.5	1
	14	1	nst	1			nst	1
	NT	8	15	2	NT		1.5,2	
Vic	15	8	1.7(16)	3	2a	55	1.4	24
			nst	5			1.2	10
	4	4	1.4	4			1.5,2	5
	2a	2	nst	2			1.5	2
	2b	3					nst	14
	NT	22	1.4	8	2b	2		
			1.7	2	15	1	12,13	1
			nst	6				
WA	15	10	1.7 (16)	6	2a	7	1.5	2
	4	2	nst	2			nst	3
	NT	25	1.4	7	2b	1	1.2	1
			1.15	2	NT	2	nst	2
			nst	13				

NT Not typed
nst No serosubtype

Serogroup distribution has been typically age-associated, but jurisdictional differences were evident in 2000 (Figures 1 a-c). In jurisdictions other than New South Wales and Victoria, serogroup B strains predominated in all age groups. In all jurisdictions serogroup B predominated in those aged 4 years or less. In Victoria group C strains were more frequently isolated in all other age groups. In New South Wales group C meningococci were also frequently isolated, but serogroup B was seen more often in those aged between 15 and 24 years.

There was again considerable phenotypic heterogeneity amongst invasive isolates as determined by serotyping and serosubtyping. The predominant serotypes/serosubtypes in each State and Territory are shown in Table 3. Serogroup B meningococci are more difficult to characterise by serological methods and a number could not be phenotyped. B:4:P1.4(7) strains predominated in New South Wales and were also present in Queensland, South Australia and Victoria. B:15:P1.7 strains were present in New South Wales, Queensland, Victoria, and Western Australia.

There was less heterogeneity amongst serogroup C meningococci. Isolates were usually either serotype 2a or 2b. Phenotype C:2a:P1.4(7), which appeared in Victoria in 1999, requires special comment. There were 10 such strains in Victoria in 1999 and 24 in 2000, but they were rarely encountered elsewhere in Australia. Phenotype C:2a:P1.2 was also frequently isolated in Victoria in 2000 (10 isolates) but also rarely identified in other centres. New South Wales was the only other State with a higher proportion of serogroup C strains. Phenotypes C:2a:P1.5 and C:2a: P1.5,2 accounted for 70 per cent of serogroup C strains in that State. The C:2a:P1.5 phenotype was present in most jurisdictions. Serotype 2b strains were encountered in low numbers.

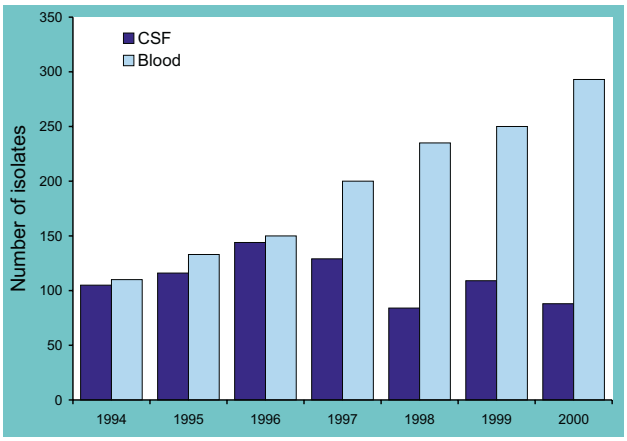
Site of isolation

There were 88 isolates from cerebral spinal fluid (CSF) either alone or with a blood culture isolate and 293 from blood cultures alone. Five isolates were identified from synovial fluid and one from skin. There has been an increase in the proportion of isolates identified from blood cultures over time (Figure 2). In 2000, the ratio of CSF isolates to blood culture isolates was 0.3:1, a substantial decrease from that recorded in 1999 (0.44:1).

Outcome data for cases with sterile site isolates

Outcome data (survived or died) were available for 278 patients (71%). Twenty-five deaths were recorded (9%) (Table 4). Outcomes were available in 70 per cent of serogroup B infections and 75 per cent of serogroup C infections. There were 9 (5.9%) deaths in serogroup B infections and 13 (12%) in serogroup C infections. Where outcomes were known, there were 4 deaths in 57 patients (7%) with meningitis. Two of these patients were infected with serogroup B, and 1 each with a serogroup C and serogroup Y strain. Twenty-one deaths were recorded in 218 bacteraemic patients (9.6%). There were 113 cases of serogroup B meningococcal bacteraemia with 7 deaths and another 90 cases were caused by serogroup C strains among whom 13 fatalities were recorded. Single fatalities were recorded with serogroup Y and W135 bacteraemias.

Figure 2. Number of meningococcal isolates from CSF and blood culture, 1994 to 2000



Antibiotic susceptibility surveillance of invasive meningococcal isolates

Penicillin

Three hundred and sixty-nine isolates of the 388 strains were tested for their susceptibility to penicillin. Using defined criteria, 118 strains (31.9%) were fully sensitive to penicillin and 251 (68.1%) less sensitive (MIC 0.06 to 0.5 mg/l). These

Table 4. Outcome of meningitic and septicaemic cases of meningococcal infection, Australia, 2000, by serogroup and culture positive cases

Disease Type	Outcome	Serogroup B	C	Y	W135	NG	Total
Meningitis	Survived	38	14	0	0	1	53
	Died	2	1	1	0	0	4
	Total	40	15	1	0	1	57
Septicaemia	Survived	106	78	6	7	0	197
	Died	7	12	1	1	0	21
	Total	113	90	7	8	0	218
All cases*	Total	153	108	8	8	1	278
	Died	9	13	2	1	0	25

* Includes 3 serogroup C strains from joint aspirates from patients who survived.

proportions differ only slightly from those recorded for recent years. The highest MIC recorded was 0.5mg/L which was identified in 3 isolates.

Other antibiotics

All isolates were susceptible to ceftriaxone (and by extrapolation to other third generation cephalosporins) and to the prophylactic antibiotics rifampicin and ciprofloxacin.

Numbers and sources of non-culture diagnoses of IMD

There were 147 diagnoses of invasive meningococcal disease in 2000 diagnosed by PCR and/or serology in the absence of positive cultures (Table 5). Both tests were positive in 7 instances where both serology and PCR testing were performed. It was more usual however, to have available samples suitable for testing by only one of the above techniques. Ninety-one cases were diagnosed by PCR testing, and a further 49 cases diagnosed by serology.

For the cases diagnosed by PCR testing, it was also possible to categorise the disease type by source of specimen (Table 5). Of the 98 cases diagnosed by PCR, 43 were from CSF or CSF and blood, 53 from blood only and 2 from blood and joint fluid. This is a different distribution from that obtained with culture-based diagnosis. Culture-based diagnosis of blood yielded 2.5 times the number of cultures derived from CSF. With PCR based diagnosis the ratio of blood to CSF positive was 1.2:1. Changes in the number and sources of non-culture-based diagnoses of invasive meningococcal disease are shown in Table 5.

Serogroup and age distribution of non-culture-based IMD

Infections diagnosed by PCR can also be serogrouped. At present this is not available for serogroups other than B or C and cannot be performed by all centres. Of the 98 cases where a PCR-based diagnosis was made, the serogroup was also determined as B or C in 64 cases (Table 6).

Table 5. Source of non-culture based diagnosis of invasive meningococcal disease, 1999 to 2000

	1999	2000
All non culture based diagnoses	92	147
PCR and serology positive	13	7
PCR positive alone	41	91
CSF PCR positive*	*36*	*35*
CSF and Blood PCR both positive*	*18*	*9*
Blood PCR positive*		*52*
Blood/Joint positive		2
Serology positive alone	38	49

* including those with positive serology

Table 6. Serogroup and age distribution of IMD diagnosed by PCR, 2000

	Age groups									
Serogroup	<1	1 - 4	5 - 9	10-14	15-19	20-24	25-44	45-64	64+	U	Total
B	5	7	0	2	7	4	5	3	0	2	35
C	2	5	6	1	8	3	4	0	0	0	29
U*	4	10	4	1	8	6	1	0	0	0	34
All	11	22	10	4	23	13	10	3	0	2	98

U* undetermined

Table 7. Age distribution of serologically diagnosed cases of IMD, 2000

<1	1-4	5-9	10-14	15- 19	20-24	25-44	45-64	>65	Total
0	4	2	7	6	9	15	6	0	49

Table 8. Outcome data for cases of IMD diagnosed by PCR, 2000, by serogroup

	CSF			Blood			
Serogroup	S	D	U†	S	D	U†	Total
B	19	2	3	8	2	1	35
C	4	0	1	17	1	4	29*
Unknown	8	0	6	18	0	2	34
Total	31	2	10	43	3	7	98

* Includes 2 positive samples from blood/joints

† Not known

For those cases diagnosed by serology alone, age distribution was different, with most diagnoses (43 of 49) in those aged 10 years or more. This reflects in part the difficulty in obtaining serum samples from young children. The categorisation of IMD by site of organism capture cannot be determined with serology. Additionally, serogroup determination is not possible.

Clinical outcome for IMD based on non-culture-based diagnosis

For IMD diagnosed by PCR based tests, the clinical outcome was known in 82 instances. There were 3 deaths where PCR testing of blood alone was positive (2 of serogroup B and one serogroup C). Of a further 43 patients with a PCR CSF sample, 31 survived (19 group B, 4 group C, 9 undetermined serogroup) and 2 died (both serogroup B). Two cases who were diagnosed with serogroup C by PCR testing of blood or joint fluid, survived. Forty-seven of 49 cases diagnosed serologically survived and the outcome was unknown in the remaining cases.

Discussion

The total of 388 isolates examined by NNN laboratories in the Australian Meningococcal Surveillance Programme in 2000 was the highest since the inception of the Programme in 1994. The numbers of isolates examined between 1997 and 1999 represent small aggregate changes only. When data are disaggregated by jurisdiction however, differences become more apparent. The number of isolates available in Victoria increased from 41 in 1998 to 94 in 1999 and further to 108 in 2000. In contrast, the number of isolates from Queensland decreased from 81 to 66 to 43 over the same period. Isolate numbers in New South Wales and Western Australia increased slightly in 2000 but varied little from 1999 totals in other centres.

The number of isolates available for examination will always be less than the number of clinically notified cases because clinical surveillance case definitions include culture negative cases. The increasing capacity for laboratory confirmation of clinically suspected IMD by non-culture-based diagnosis however, has narrowed this differential. In 2000, 147 clinical cases were confirmed only by non-culture based laboratory examinations, an increase from the 86 diagnoses made by this means in 1999. These procedures include NAA assays using PCR and/or serological examination. Data on these cases were included separately in this report. Some of the PCR techniques in use can provide additional data on the serogroup of the isolate. It is likely that the use of these techniques will increase and that with further refinements in their application, additional subtyping data will also be available. Serological diagnosis of less florid cases has also increased. NNN laboratories may be contacted for advice regarding these tests.

The ratio of cases of meningitis to bacteraemia in culture-confirmed cases declined further in 2000, continuing a trend first noted in 1997. This trend was the subject of comment in the preceding report.[8] It was also noted in that report that there was a distinct difference in the source of PCR-based diagnostic material, with more diagnoses from CSF compared to blood. There is a possibility of bias in the PCR diagnostic data for 1999 as PCR was initially performed on CSF samples only. In addition, the sensitivity of PCR techniques in blood samples is less than for CSF. In the 2000 data, there was an increase in PCR based diagnoses from blood and a corresponding 'correction' in the proportions of diagnoses from CSF and blood by this technique.

The predominant disease pattern throughout the country continued to be sporadic infection with serogroup B meningococci. The proportion of serogroup C cases in aggregated data again increased in 2000. Analysis of serogroup distribution by State or Territory however, reveals considerable differences. About 80 per cent of serogroup C strains are found in the 2 larger States and Victoria is the only jurisdiction where the majority of strains are serogroup C. Cases of serogroup C increased from 7 in 1998 to 42 in 1999 and in 2000 the total reached 58. Serogroup C infections have been prominent in New South Wales for a number of years, although serogroup B strains have always been the majority. Serogroup C cases were also sporadic in 2000 and no serogroup A meningococci were isolated. The proportion of serogroup Y and W135 strains remained unchanged.

Children aged 4 years or less are traditionally the age group most frequently infected and a secondary incidence peak in young adults and adolescents is also usual. This pattern changed in 2000 with those in the 15-24 age range having more infections than those aged 4 years or less in aggregated data. Again this pattern varied by region with Victoria having the highest proportion of young adult cases. In New South Wales the case numbers in the two age groups were similar but elsewhere the usual infant case predominance prevailed. Serogroup B infections were the most frequently seen in the infant age group. Serogroup C disease occurred more often in the young adult age group and was responsible for the peak in adult cases in Victoria. In contrast, the high secondary peak in young adults in New South Wales involved more serogroup B cases.

Phenotyping data obtained on the basis of serotyping and serosubtyping emphasise the considerable differences that exist in meningococcal subtypes causing IMD in different jurisdictions. The heterogeneity of serogroup B isolates present in Australia was once more evident in 2000. Of interest amongst the group B strains were phenotypes B:4:P1.4(7) and B:15:P1.7 associated with hyperendemic disease in New Zealand and Europe respectively. B:4:P1.4(7) strains were prominent in New South Wales with this phenotype representing about 15 per cent of all isolates. Phenotype B:15:P:1.7 was widely distributed.

Of particular interest in 1999 was the emergence in Victoria of a phenotype C:2a:P1.4(7) and this phenotype persisted in 2000, accounting for about 22 per cent of all isolates in that State. C:2a:P1.5 was present in most jurisdictions and was the most frequently isolated phenotype in New South Wales. In Victoria, C:2a:P1.2 was relatively common (about 10 per cent of isolates) but infrequently encountered elsewhere. These variations illustrate the temporal and geographic variation in meningococcal subtypes that occurs in Australia and the volatility in predominant phenotypes that may occur. Meningococci have a well-recognised capacity for recombination through horizontal gene transfer and this may be expressed in phenotypic heterogeneity.

The overall mortality recorded in 278 assessable culture-positive cases remained at about 9 per cent and a higher mortality rate was again observed with serogroup C cases. Although serogroup C strains have been associated with increased mortality overseas, other factors, such as

age, and time from onset to presentation and treatment, may also explain this difference. No data were available on this however.

No strains resistant to penicillin were detected in 2000. The highest MIC recorded was 0.5 mg/L in 3 isolates and the proportion of 'less susceptible' strains remained essentially unchanged. All isolates were susceptible to the prophylactic agents rifampicin and ciprofloxacin and to the third generation cephalosporins.

The NNN is a continuing, long-term collaborative study that has examined a total of about 2200 strains from all States and Territories since 1994. It has assisted in clarifying and expanding information on invasive meningococcal isolates in Australia to augment data collected separately by clinically-based surveillance systems. The nature and high public recognition of meningococcal disease together with the proposed release of new vaccine types, suggests that the efforts of this Programme should continue. For further details please contact the relevant NNN member (see acknowledgments for contact numbers).

Acknowledgments

Isolates were received in the reference centres from many laboratories throughout Australia. The considerable time and effort involved in forwarding these strains is recognised and these efforts are greatly appreciated. These data could not have been provided without this assistance and the help of clinical colleagues and public health personnel.

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An outbreak of serogroup C meningococcal disease associated with a secondary school

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Abstract

An outbreak of 3 cases of invasive meningococcal disease occurred in a secondary school on 2 campuses in Victoria. Despite having only one isolate (a C.2a:nst strain), meningococcal DNA was identified by polymerase chain reaction (PCR) in early culture-negative blood specimens of the other 2 cases. Both were subsequently shown by PCR to be capsule serogroup C by PCR. An committee was formed to manage the response to the outbreak. Chemoprophylaxis was offered to family and children who had been in close contact with the cases. As one strain had been confirmed as being of a vaccine-preventable group, vaccination was offered to the whole school community as well as the families of cases. The direct costs of the outbreak to public health, which would have been identical whatever the causative serogroup, was $8,178. Vaccine charges accounted for most of the additional $56,941 cost of vaccinating the target group of 1600 students, staff, and families. No further cases have been associated with this outbreak. *Commun Dis Intell* 2001;25:121-125.

Keywords: surveillance, Neisseria meningitidis*, meningococcal disease, antibiotics, penicillin, vaccination*

Introduction

At the end of August 1999, amongst the usual and expected seasonal rise in meningococcal infections, 3 children with meningococcal septicaemia, who attended the same secondary school, were notified to the Victorian Department of Human Services, Communicable Diseases Section within 48 hours of each other. Two presented to a local hospital and one consulted a general practitioner with a 13-19 hour history of symptoms. Blood for culture was collected from all 3 cases, one before and two after the administration of antibiotics. All 3 children were transferred to the same major paediatric facility by paediatric emergency transfer (PETS).

Public health management of meningococcal disease

There are 3 strategies for the control of meningococcal disease: chemoprophylaxis; vaccination; and the dissemination of information.

Chemoprophylaxis (antibiotics which efficiently remove meningococci from the nasopharynx) is given to prevent further transmission between carriers (including cases who are treated with penicillin alone) and susceptible individuals.[1-4] Public health case management begins with a review of the recent activities of cases in order to identify two groups of people who should be offered chemoprophylaxis:

- the group of people which includes the carrier who transmitted the organism to the case, and who may pose a risk to other susceptible individuals; and

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- potential co-primary and secondary cases, who acquired meningococcal infection at the same time or shortly after the case. These people need special advice and monitoring, as prophylaxis may not prevent secondary cases.[5]

Vaccination for the protection of defined populations can be undertaken if characterisation of the organism proves it to be of a vaccine-preventable strain.[1,6] Almost half of microbiologically confirmed cases in Victoria in 1999, were shown to be due to serogroup C strains, for which polysaccharide vaccines are available. In developed countries, serogroup C strains are responsible for about two-thirds of outbreaks. It is helpful therefore to be able to characterise invasive strains to identify outbreaks. Appropriate specimens for culture for strain identification include blood, CSF, joint aspirates, throat swabs, and picked spots or punch biopsies of affected skin.[7,8]

Viable meningococci may be retrieved for up to 3 hours after instituting antimicrobial therapy,[9] and CSF and picked spots or punch biopsy specimens can provide a culture-positive specimen for somewhat longer after commencement of antibiotics.[10] In small children, a positive throat swab is uncommon,[11] whilst teenagers have a meningococcal nasopharyngeal colonisation rate of 10-30 per cent.[12-14] A positive throat swab in an otherwise clinical case therefore, provides useful microbiological guidance for public health purposes.[15]

Cases related in time may be from different or the same serogroup, type, subtype and molecular type; an outbreak involves cases of an identical strain. We report here the epidemiological and microbiological features and public health management of this small outbreak, including a public health interventions cost analysis.

Public health investigation

Epidemiology

We collected a routine case history from the primary contacts of each case, noting in particular, details of recent extra-curricular school activities.

Communication

Public health management of the outbreak included the design of the vaccination program, planning vaccine delivery, and the development of effective multiple communication strategies including the school, local newspaper, television and radio media.

Microbiology

Blood for culture was collected from all 3 cases prior to PETS transfer, but only those from Case 3 accompanied the case to hospital.

With the exception of a throat swab from Case 3 late on the day of admission, no specimens were collected for microscopy and culture by the paediatric facility. Thus we were reliant upon results from the blood specimens collected before admission, and on specimens collected for other purposes retrieved from the hospital laboratory, for decisions relating to public health management of these apparently related cases.

At the time of this outbreak, staff at the Microbiological Diagnostic Unit (MDU), State Neisseria Reference Laboratory, were investigating the possibility of using molecular techniques including polymerase chain reaction (PCR) and nucleotide sequencing methods to assist in the identification of strains of meningococci. Specimens from all 3 cases were therefore also subjected to molecular investigation.

Vaccination

The school meningococcal vaccination program was designed after ascertaining that sufficient polysaccharide vaccine was available to conduct the campaign.

Results

Epidemiology

The secondary school attended by the 3 cases has approximately 1500 enrolled pupils and 150 staff, divided into 2 campuses. Discussion with the parents of the cases identified several important considerations.

- The first 2 cases were close friends, but had spent much of their incubation times apart.
- The third case was unknown to the other two, and attended a separate campus.
- The 3 pupils had recently been involved in different school extension camp activities.
- One-hundred and forty students and 16 staff from one campus went to Canberra. Students attending this camp reported a high rate of coughs and colds.
- Eight students and a senior teacher from one campus went on a sports camp to Darwin involving in a great deal of physical activity, arriving home 'exhausted'.
- Fifty-six students and 12 staff from both campuses went on a one-day beach retreat day and picnic.
- Although the school camps to Canberra and Darwin involved only one campus, the retreat had involved students from both campuses.

The attack rate in the school community, with 3 cases in 1600 individuals, was high at 187/100,000. However, if the first 2 cases were considered to be co-primary and considered as a single case, the subsequent occurrence of a temporarily-linked but socially unlinked case from the same community alerted public health staff to the possibility of an outbreak with an attack rate of 114 per 100,000 population. A summary of the main temporal epidemiological features of the outbreak is presented in the Figure.

Microbiology

A single positive culture was retrieved from the blood specimen from Case 1. By conventional laboratory analysis, the group was confirmed as serogroup C, and by monoclonal antibody assay typing and subtyping, 2a:nst (see discussion). PCR quickly confirmed the presence of meningococcal DNA in early blood specimens of all 3 cases.

The subsequent use of another more specific PCR assay confirmed the 2 culture-negative specimens as serogroup C. Further molecular analysis revealed these organisms all had a specific and identical mutation in the *porA* sequence, strongly suggesting that the same strain of *Neisseria meningitidis* was the cause of all 3 cases.

Public health management of the outbreak

Prophylaxis to case contacts

In accordance with the National Health and Medical Research Council (NHMRC) guidelines, intimate family

Figure. Temporal epidemiological feature of the outbreak, chemoprophylaxis and vaccination campaign, Victoria, 1999

R Rifampicin administered to family contacts and school camp/year group contacts.

C Confirmation that blood culture from case 2 had yielded strain of serogroup C.

↓ Immunisation campaign and mop-up.

of the cases were also considered to be close contacts in need of chemoprophylaxis. In practice, this involved the whole of a year group on one campus, and a number of children from a different year group from both campuses.

The vaccination campaign

As one case had been confirmed as being serogroup C, and the isolate had been recovered from one of the pair of friends, the likelihood of the second of these two being caused by a different strain was considered remote. It was therefore decided to offer vaccination to the group of people at high risk of secondary disease, including all students enrolled at the school, school staff, and close family contacts.

The organisation of equipment, staff, infrastructure support and vaccine supplies was completed in a single day. The following day — 36 hours after the confirmation of a serogroup C strain in one of the cases and demonstration of the presence of capsule serogroup C meningococci in the blood of all three by PCR — a mass meningococcal vaccination program was undertaken targeting all students enrolled at the school, school staff, and all close family contacts.

Our school-based communication strategy for parents resulted in a very high rate of signed parental consent to the vaccination campaign. Of the target of 1600 staff and students of the school, 1530 were vaccinated on the campaign day. Vaccination sessions for the school were held on the school campuses, during school hours, over 4 hours, reaching over 95 per cent of the school population. The family members and the remaining students were vaccinated 2 days later. There were no major sequelae following vaccination.

Communication

Public anxiety surrounding the outbreak was very high. Responding to public queries and the preparation of press packs occupied many hours of staff time.

The cost of the outbreak

The costs associated with this outbreak are summarised in the Table. Early public health response to the outbreak was estimated to have cost $8,178, largely accounted for by staff time spent in response to public enquiries. These costs would have been incurred, whether the outbreak had been due to a serogroup B strain, for which no vaccine is currently available, or serogroup C. The cost of chemoprophylaxis, at $1.51 per adult course, was less that 5 per cent of the total cost of the public health management of the outbreak.

The cost of the vaccination campaign was $56,941, most of which was accounted for by vaccine, at a bulk rate of $27.50 per dose. The staff costs for delivering the vaccination campaign were very similar to those for the management of the outbreak.

The costs to agencies outside the Communicable Diseases Section have not been included in this analysis, because we have no means of estimating these. There undoubtedly will have been an equivalent cost to local health and education services, as many calls to public health were from other professionals ringing for advice to pass on to others, for example the safety or otherwise of children using communal school buses.

Discussion

The epidemiology of meningococcal disease of serogroups B and C disease is different. In developed countries, whilst serogroup B is the cause of most endemic disease,

Discussion

The epidemiology of meningococcal disease of serogroups B and C disease is different. In developed countries, whilst serogroup B is the cause of most endemic disease, serogroup C is more often responsible for outbreaks. To illustrate, a recent analysis of school-based outbreaks of meningococcal disease reviewed the principles of school outbreak management in 22 outbreaks. Serogroup C was responsible for 14 outbreaks compared with serogroup B, 7, and serogroup Y, a single outbreak.[16]

Several serogroup C outbreaks have been reported in groups of teenagers since 1992, mainly C.2a:P1.2 and related strains. The first, in Ottawa, Canada, was loosely defined precipitating a fairly ineffectual prophylaxis program, followed by a vaccination program which halted the outbreak.[17] The following year a Danish report documented 20 teenage cases over 7 months in 3 outbreaks. In this group meningococcal carriage was studied in detail demonstrating that carriage patterns were unrelated to local attack rates.[14] The most recent report comes from England and documented a series of 7 cases in a population of 7100 during 2 months. A vaccination campaign reached 83 per cent of the identified risk groups. During the next year 3 further cases occurred in local teenagers, one a vaccinated child.[18]

An outbreak of the same phenotype based on a student population in Sydney over a 7-week period consisted of a pair of co-primary cases, a related secondary case, and a fourth case who was considered, by pulsed field gel electrophoresis (PFGE), to be unrelated.[19] Vaccination was offered after the diagnosis but before PFGE results were available. The outbreak provoked much disquiet and the vaccination campaign caused considerable expense. The authors report that they would not have offered vaccination if the PFGE results, which suggested that the cases were unrelated, had been available earlier as the first 3 cases did not fulfill the definition of an outbreak provided in the NHMRC guidelines.

There are shortcomings with both chemoprophylaxis and polysaccharide vaccines as preventive public health tools. Chemoprophylaxis may only delay rather than prevent the onset of secondary cases.[20] Polysaccharide vaccine provides protection for up to 5 years,[21] however, it does not eliminate nasopharyngeal carriage[22] and cases have occurred shortly after a C-strain mass vaccination campaign.[18,23,24]

Table. The cost of the outbreak and vaccination campaign

	Units	Average unit staff cost per day, Aus$	Total Aus$ cost
Outbreak costs			
Medical officers	13 days	> $345	4770
EHO and nursing staff	15 days	$169	2538
Administration and media staff	2 days	>$120	419
Total staff costs			7727
Telephone, fax, consumables			100
Rifampicin to contacts: 153 Year 9 & staff; 64 Year 10 & staff; 15 family and friends	232 courses of 16 capsules = 3712 capsules	$9.45 for 100 caps	351
Management and administration sub-total			8178
Vaccination campaign costs			
Medical officers	7.0	as above	2654
EHO and nursing staff	27.5	as above	4654
Administration and media staff	2.5	as above	333
Total staff costs			7641
Vaccine			
School	1600 doses		
Family members	15 doses		
Wastage	~ 105 doses	$27.50 per dose	47,300
Vaccination-associated consumables			500
Travel			1500
Sub-total of vaccination campaign costs			56,941
Total cost of the outbreak and vaccination campaign			65,119

NB. The costs of accommodating the vaccination sessions, printing and distributing parental information and consent forms, and the management of the students was borne by the school involved.

* *PorA* VR (variable region) Type 5-1, 10-4 with a G T base substitution at position 76 of the coding sequencing, which would make the encoded *porin* protein non-functional. See database at: http:// www.mlst.zoo.ox.uk/porA-vr/porA

The secondary attack rate in the close contacts of cases however, has been variously reported as 4.34/1,000 in untreated contacts,[21] and 0.5 per cent (5/1,000) in microbiologically confirmed cases in all close contacts.[25] On this basis, in the combined school and family population we would have expected to experience at least one and up to 6 more cases in this outbreak, if we had not undertaken the $57,000 vaccination campaign.

The early interpretation of these 3 cases as an outbreak was helpful in guiding our decision to vaccinate. The importance of incorporating molecular microbiological information in public health decision making is that we were sure that only one serogroup C strain was active in the community at that time. All 3 cases were subsequently shown to be have an unusual *porin* type* and were probably identical. Whilst gratifying, this does not alter the rationale for these decisions. In the future, molecular typing methods will provide powerful enhancement to public health management of meningococcal disease.

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Editorial: Meningococcal disease

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Meningococcal disease causes at least 500,000 cases and 50,000 deaths worldwide each year. Epidemics of meningococcal meningitis occur in many parts of the world. Serogroup A is associated with explosive epidemics with attack rates up to 500 per 100,000. An epidemic of serogroup A in the African 'meningitis belt', which began in 1996, has resulted in at least 300,000 cases to date and many thousands of deaths.[1] Serogroup B is the major cause of sporadic meningococcal disease in industrialised countries. Serogroup C, which also occurs in industrialised countries, causes both sporadic and epidemic disease. Serogroup C outbreaks typically result in attack rates between those of serogroups A and B.[2] Serogroups Y and W135 are uncommon in Australia.

In New Zealand, an epidemic of serogroup B meningococcal disease is in its eleventh year (Martin, Communicable Disease Control Conference, April 2001, abstract 2). In 2000 there were 480 cases of meningococcal disease in New Zealand giving a notification rate of 13.3 per 100,000 compared with 3.1 per 100,000 in Australia in the same year. The epidemic in New Zealand is particularly concentrated in the North Island among Maori and Pacific Islander communities. Overcrowded housing has been identified as a strong risk factor for meningococcal disease in this epidemic.[3] Phenotype B:4:P1.4(7) associated with hyper-epidemic disease in New Zealand was also present in New South Wales in 2000 (see the Australian Meningococcal Surveillance Programme Annual Report 2000, this issue pp 113-121)

Meningococcal disease in Australia has been in decline since a pandemic of serogroup A disease during World War II, when notification of 'meningitis' was 33.1 per 100,000.[4] Notifications fell to <0.5 per 100,000 in 1987 but increased due to an outbreak of serogroup A disease among indigenous populations in Central Australia[5] and a rise in notifications of group B and C disease. The notification rate for meningococcal disease to the National Notifiable Diseases Surveillance System (NNDSS) has been slowly increasing over the past 10 years from 1.6 per 100,000 in 1991 to 3.1 per 100,000 in 2000 (Figure 1). In common with other industrialised countries, the increase of meningococcal disease in Australia has been primarily due to the expansion of virulent phenotypes of serogroups B and C[2] causing small outbreaks.[6,7]

The rates of meningococcal disease in 2000 varied from 5.1 per 100,000 in the Northern Territory to 1.6 per 100,000 in the Australian Capital Territory. The disease shows a typical late winter peak each year between July and September (Figure 2). The disease typically affects children aged 0 to 4 years but also occurs in young adults aged 15 to 19 years (Figure 3). In 2000, the number of cases among adolescents and young adults aged 15 to 24 was almost equivalent to the number of cases among infants and children aged less than 4 years. There were 41 deaths attributable specifically to invasive meningococcal disease in 1999, giving an overall case-fatality rate of 7.2 per cent. This may be an underestimate however, as some of the deaths due to unspecified meningitis and septicaemia may have been caused by the meningococcus (ABS, 2001).

Figure 1. Notification rate for meningococcal disease, Australia, 1991 to 2000

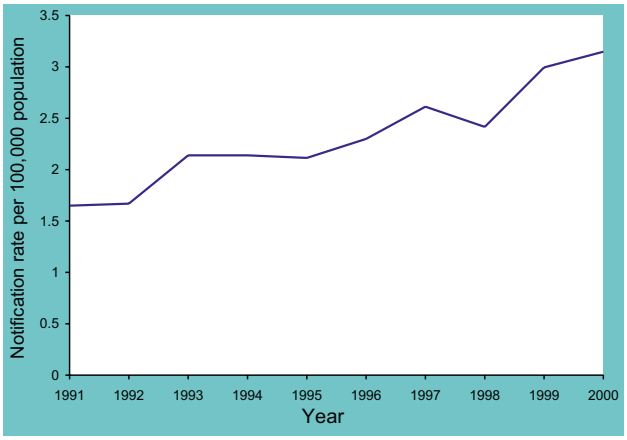


Figure 2. Notifications of meningococcal disease, Australia, 1991 to 2000, by month of onset

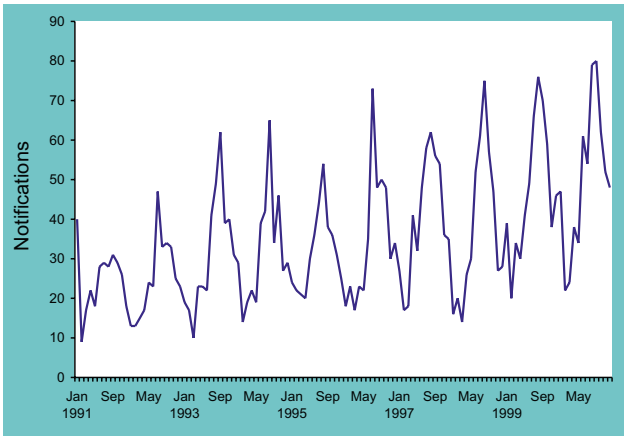
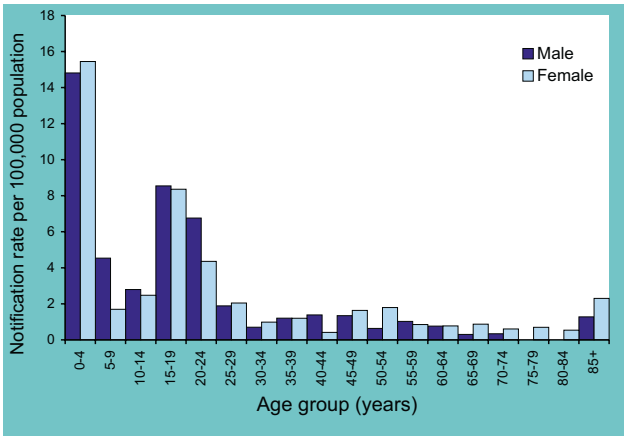


Figure 3. Notification rate of meningococcal disease, Australia, 2000, by age and sex



There is some variation among the case definitions of meningococcal disease in use across Australia (Table 1). The Meningococcal Working Group of the Communicable Diseases Network Australia (CDNA) is developing a standard case definition for invasive meningococcal disease that will be used in all jurisdictions. This will help provide a more accurate measure of the incidence of meningococcal disease in Australia.

Since 1994, the National Neisseria Network (NNN) has provided important information on the serogroups of *Neisseria meningitidis* circulating in Australia and on their antibiotic susceptibility. A variable proportion of culture confirmed and of all notified meningococcal cases in Australia each year are all referred to the NNN (Tables 2 and 3). In 2000, 89 per cent of notified cases were included in the NNN reports (388 culture positive and 147 culture negative samples). The culture positive sample tested by the NNN represents isolates from all jurisdictions with between 56 per

Table 1. Definitions of meningococcal disease in use in Australia

Jurisdiction/ Organisation	Definition of meningococcal disease
NHMRC (National Health & Medical Research Council) (in use in the ACT, SA, Tasmania, Victoria)	Isolation of *Neisseria meningitidis* from a normally sterile site; OR Detection of meningococcal antigen in joints blood or CSF OR Detection of gram negative diplococci in blood or CSF.
New South Wales	**Suspected case:** Any person with signs or symptoms of meningococcal disease. **Presumptive case:** A suspected case with: contact with an infectious confirmed case 2 to 10 days before onset; OR A clinical diagnosis of meningococcal disease; OR A positive antigen test on CSF. **Confirmed case:** Culture of *N. meningitidis* from normally sterile site; OR Culture of *N. meningitidis* from the conjunctiva; OR Detection of gram-negative diplococci in a normally sterile site; OR A positive PCR test on CSF or blood in a case with clinically compatible illness; OR Positive serology indicating *N. meningitidis* infection, either as a single positive IgM antibody titre, or as a 4-fold rise in antibody titre between acute and convalescent specimens where the convalescent serum is taken more than 2 weeks after onset of illness.
Northern Territory	**Confirmed case:** Clinically compatible illness AND Isolation of *N. meningitidis* from a normally sterile site; OR Detection of a gram-negative intracellular diplococci in blood CSF or skin; OR isolation of *N. meningitidis* from skin in the absence of a positive blood culture. **Probable case:** Clinical pupura fulminans in the absence of positive blood cultures; OR Detection of meningococcal antigen in CSF.
Queensland	As per NHMRC with the addition of detection of *N. meningitidis* nucleic acid in joints, blood, CSF, tissue or urine.
Western Australia PHLN (Public Health Laboratory Network)	Isolation of *N. meningitidis* from blood CSF or other normally sterile site; OR Detection of gram-negative diplococci in blood CSF or other normally sterile site or in smears from skin lesions in a patient with meningitis, septicaemia or other clinically compatible illness. **Definitive criteria:** Isolation of *N. meningitidis* from a normally sterile sit; OR Detection of *N. meningitidis* by NAT; OR Single high titre IgM and/or IgG titres to outer membrane antigens of *N. meningitidis*. **Suggestive criteria:** Detection of gram-negative diplococci in gram-stained material from a normally sterile site; OR Positive polysaccharide antigen test in CSF with other laboratory parameters consistent with meningitis.

Table 2. Number of cases of invasive meningococcal disease (IMD) notified to NNDSS and number of culture confirmed cases tested by the NNN, 2000, by State and Territory

	ACT	NSW	NT	Qld	SA	Tas	Vic	WA	Total
IMD reported to NNDSS (n)	5	253	9	51	32	15	162	72	599
Culture confirmed IMD tested by the NNN	5	141	7	43	20	14	108	50	388
Culture confirmed cases as a proportion of all cases on the NNDSS	100	56	78	84	63	93	67	69	65

Table 3. Proportion of cases of meningococcal disease notified to NNDSS tested by the NNN, 2000, by age group

	< 1 year	1-4 years	5 to 9 years	10-14 years	15 to 19 years	20 to 24 years	25 to 44 years	45 to 64 years	65+	Total
No. reported to NNDSS	70	121	41	36	114	76	72	50	15	599
No. referred to NNN*	58	99	39	33	108	69	64	43	14	535[†]
% of all cases reported by NNDSS and NNN	82.9	81.8	95.1	91.7	94.7	90.8	88.9	86.0	93.3	89.0

* Cases diagnosed by culture, serology or PCR

cent and 100 per cent of notified cases in the States and Territories being culture positive (Table 2). There was also a representative sample of laboratory-confirmed cases from each age group in the NNN report (Table 3). Increasing use of non-culture methods for diagnosis of meningococcal disease was noted. In this report 147 cases were diagnosed by PCR, serology or both. Some of these diagnostic methods, such as serology were more likely to be used in older patients. Sixty-four cases on the NNDSS were either notified on clinical or epidemiological grounds or laboratory specimens had not been referred to the NNN.

The NNN report for 2000 highlights two important features of meningococcal disease in Australia.[8] There was a significant difference in the distribution of different *N. meningitidis* phenotypes geographically and among different age groups. Serogroup C isolates, especially phenotype C:2a:P1.4,[7] are common in Victoria, particularly among adolescents and young adults. This phenotype is infrequently found in other centres; the reasons for this geographical difference are unclear. *N. meningitidis* serogroup B phenotypes associated with hyperendemic disease in New Zealand predominate in New South Wales and are found widely distributed elsewhere in Australia.

Meningococcal disease in Australia, while moderately resistant to penicillins, is susceptible to third generation cephalosporins and to rifampicin and ciprofloxacin used in prophylaxis. Drug resistance in *N. meningitidis* does not yet compromise the treatment and control of meningococcal disease in Australia. Continued surveillance of meningococcal antibiotic susceptibility however, is essential to disease control.

The impending release of the CDNA '*Guidelines for the early clinical and public health management of meningococcal disease in Australia* is principally aimed at assisting primary care providers with the emergency management of cases of suspected invasive meningococcal disease and to assist public health practitioners to prevent further transmission after an index case has been reported. Combined with information sheets sent to all general practices in Australia, it is hoped that the diagnosis and treatment of the individual patient and management of outbreaks of meningococcal disease will improve. The report will shortly be available on the Communicable Diseases Australia Website at: http://www.health.gov.au/pubhlth/cdi/cdihtml.htm.

Ultimately the most effective public health strategy for controlling meningococcal disease may be routine vaccination of at-risk populations.[9] A study recently published[10] examined the cost effectiveness of meningococcal vaccination using the polysaccharide vaccine in Australia. Skull and colleagues concluded that in a population with incidence rates in excess of 14 per 100,000, vaccination of persons in the 15-19 year age group would be cost effective assuming that there would be a 5 year duration of protection.

A recent report on the new meningococcal serogroup C conjugate vaccine in England[11] showed an efficacy of 97 per cent in adolescents and a 92 per cent efficacy in infants 9 months after a single dose. The efficacy of the vaccine in infants was far superior to that obtained with the meningococcal polysaccharide vaccine. A more recent study has shown a 25 per cent increase in serogroup B disease across all age groups in the United Kingdom since the vaccination campaign (Kaczmarski, 2001 abstract). This observation supports a hypothesis that serogroup replacement may be an important factor in the epidemiology of meningococcal disease after the introduction of new vaccines. It therefore remains to be seen what the value of meningococcal vaccines will be in the future control of meningococcal disease.

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Measles immunity among young adults in Victoria

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Abstract

Measles outbreaks in Victoria in 1999 and 2001 have suggested that a substantial proportion of young Victorian adults may be susceptible to measles infection. We performed a serosurvey of 300 18-30-year-old healthy blood donors and 312 sera retrieved after diagnostic testing for a non-rash illness in patients of the same age group, with the aim of estimating the proportion of young adults in Victoria immune to measles. We also aimed to define more precisely the birth cohorts at risk of measles infection, with cohorts reflecting the measles immunisation policies of previous years. There was no significant difference in measles immunity between the 300 blood donors (79.0%, 95% confidence interval 73.9-83.5) and the 312 patients whose sera had been stored (84.0%, 95% CI 79.4-87.9, p=0.11). There was, however, a significant difference in immunity by birth cohort. In the combined results from both samples, the proportion of people born between 1968 and 1974 who were immune to measles was 88.4 per cent (95% CI 84.1-91.6) while the proportion of those born between 1975 and 1981 was 74.1 per cent (95% CI 68.7-79.1). This study confirms that a substantial proportion of young Victorian adults are susceptible to measles, but also demonstrates that those born between 1975 and 1981 are more likely to be non-immune than those born before 1975. A review of published Australian data supports this conclusion and confirms the need for a measles control program aimed at young adults. *Commun Dis Intell* 2001;25:129-132.

Keywords: measles, immunity, cohort analysis, immunisation, young adults

Introduction

In some countries where universal measles vaccination has been introduced over an extended period of time, young adults are emerging as the group most at risk of measles infection.[1,2] The first measles outbreak in Australia involving predominantly young adults occurred in Victoria, with spread to South Australia, between February and May 1999. Approximately 85 per cent of the 75 notified cases confirmed with measles in this outbreak were born between 1968 and 1981 (aged between 18 and 30 years).[3] These young adults were most likely to be susceptible to infection because of the timing of changes to measles vaccination practices in Australia. Measles vaccine was first licensed in Australia in 1968, recommended for children aged 15 months in 1971, and included for 12-month-old infants in the first national childhood immunisation schedule in 1975.[4] Prior to the introduction of vaccine, most people acquired immunity through infection with wild measles virus in childhood. Despite suggestions of an initially poor uptake,[5] the availability of measles vaccine in Australia from 1968 lead to a reduction in circulating wild measles virus. This was reflected in decreased measles and measles encephalitis admissions to Fairfield Hospital.[6]

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The reduction in circulating wild measles virus and low vaccine coverage has left a cohort of young Australian adults with varying susceptibility to infection. In response to this, the Federal Minister for Health has announced funding to improve measles vaccination coverage in all Australians aged 18 to 30 years.[7] To more clearly define the specific birth cohorts at risk of infection; to estimate this risk; and to promote improved efficiency of this vaccination program, we studied measles immunity in 2 groups of young adults resident in Victoria.

Methods

We anticipated that susceptibility to measles among young adults might be as high as 20 per cent. To estimate this proportion with 5 per cent precision at the 95 per cent confidence level (CI), a sample size of 246 was needed. People aged 18 to 30 years are scattered throughout the community and would have been difficult to sample randomly. Instead we studied 300 healthy blood donors from Melbourne (median age 21 years, range 18-30; 44% male) and 312 patients (median age 24 years, range 18-30; 49% male) whose serum had been stored at the Victorian Infectious Diseases Reference Laboratory (VIDRL) following diagnostic testing for a non-rash illness. Age was defined at the date blood was collected. Ethics approval for this study was obtained from the Australian Red Cross Blood Service, Victoria, and the Research and Ethics Committee of the Royal Melbourne Hospital Research Foundation.

Measles specific immunoglobulin G (IgG) was determined at VIDRL using a standard commercial enzyme immunoassay (Dade Behring Enzygnost, Marburg, Germany) in accordance with the manufacturer's instructions. Sera initially defined as equivocal were re-tested. People were considered measles immune if their measles IgG was reported as positive, corresponding to a measles specific IgG concentration of approximately 320 mIU/mL.

Sera from healthy blood donors were collected in March 1999 and sera collected and stored at VIDRL between January and September 1999 were retrieved for testing.

Immunity was analysed by birth cohorts, based on the immunisation practice at the time. People born between 1968 and 1970 inclusive, the period when the measles vaccine was first licensed in Australia, and prior to the recommendation of measles vaccine for children aged 15 months, were analysed as one cohort. There was no change in measles vaccine policy in the next 4 years and people born between 1971 and 1974 inclusive were analysed as a second cohort. In 1975 measles vaccine was included in the childhood immunisation schedule. People born in 1975 or later were therefore analysed as a third cohort. Exact 95 per cent confidence intervals (95% CI) for proportions were calculated using the binomial distribution. Tests for association were performed using Fisher's exact test or the chi-squared distribution.

Results

Estimates of measles immunity for the samples of healthy blood donors and stored serum samples are shown by birth cohort and sex in the Table. The overall measles immunity amongst the 300 blood donors was 79.0 per cent (95% CI 73.9-83.5) and amongst the 312 patients whose sera had been stored was 84.0 per cent (95% CI 79.4-87.9). There was no significant difference between the 2 samples in the

Table. Proportion of young Victorian adults immune to measles by birth cohort, sex, and institutional source of sample

	Australian Red Cross Blood Service, Victoria[2]								
	Male			Female			All		
Birth cohort[1]	N	% imm[4]	95% CI[5]	N	% imm[4]	95% CI[5]	N	% imm[4]	95% CI[5]
1968-1970	28	85.7	68.3-96.0	29	89.6	72.6-97.8	57	87.7	76.3-94.9
1971-1974	42	88.1	74.3-96.0	56	85.7	78.3-92.7	98	86.7	78.3-92.7
1975-1981	64	67.2	54.3-78.4	81	72.8	62.2-77.6	145	70.3	62.2-77.6

Table. (continued) Proportion of young Victorian adults immune to measles by birth cohort, sex, and institutional source of sample

	Victorian Infectious Diseases Reference Laboratory[3]								
	Male			Female			All		
Birth cohort[1]	N	% imm[4]	95% CI[5]	N	% imm[4]	95% CI[5]	N	% imm[4]	95% CI[5]
1968-1970	34	91.2	76.3-98.1	35	88.6	73.2-96.8	69	89.9	80.2-95.8
1971-1974	48	85.4	72.2-93.9	46	93.5	82.1-98.6	94	89.4	81.3-94.8
1975-1981	73	71.2	59.4-81.2	76	84.2	74.0-91.6	149	77.9	70.3-84.2

1. For description of birth cohorts, see text.
2. Healthy blood donors aged 18-30 years in 1999.
3. Sera stored post diagnostic testing for a non-rash illness from patients aged 18-30 years in 1999.
4. % imm= Percentage of subjects immune to measles.
5. 95% CI= Exact 95 per cent binomial confidence interval.

estimation of the proportion of young adults immune to measles (p=0.11). Within each sample, males were more susceptible than females but the differences did not reach statistical significance.

There was no significant difference in immunity by exact year of birth within each of the defined birth cohorts. There was however, a significant difference in immunity by birth cohort, with both samples demonstrating significantly lower immunity amongst people born in or after 1975 compared with the 2 earlier birth cohorts.

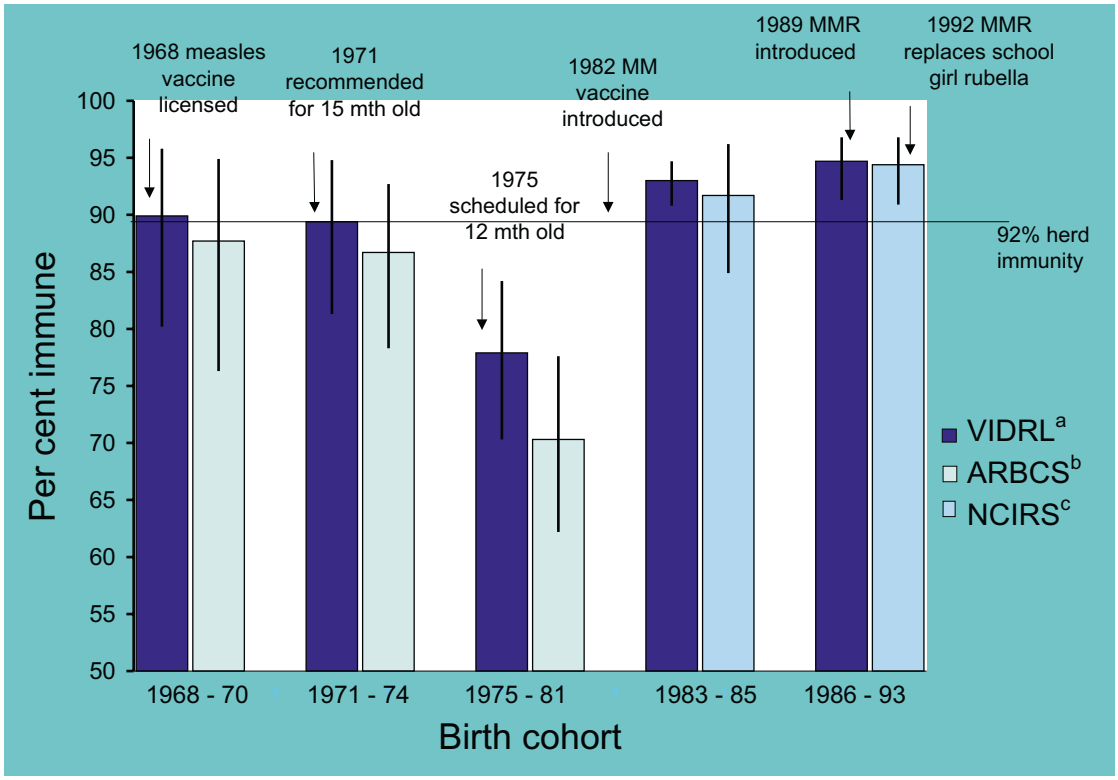
In the sample of healthy blood donors, 87.1 per cent of young adults born before 1975 were immune to measles compared with 70.3 per cent born in or after 1975 (p=0.0004). In the sample from VIDRL, 89.6 per cent of young adults born before 1975 were immune to measles compared with 77.8 per cent born in or after 1975 (p=0.005). There was no significant difference in the proportion of people immune in either sample when comparing those born in 1968-70 with those born in 1971-74. Neither was there a significant difference from the 2 samples when comparing the proportion of people immune born in or after 1975. In combining the results from both samples, we estimated that the proportion of people born between 1968 and 1974 who were immune to measles was 88.4 per cent (95% CI, 84.1-91.6) while the proportion of those born between 1975 and 1981 was 74.1 per cent (95% CI, `68.7-79.1).

Discussion

The results of 2 serosurveys of convenience samples (healthy blood donors and stored sera from diagnostic testing for a non-rash illness) support the concept of a young adult cohort susceptible to measles infection in Victoria. We have shown that younger people, born in or after 1975, are relatively less likely to be immune to measles compared with those born before 1975. This is consistent with continuing circulation of wild virus (and subsequent natural immunity) until 1975, when the first national immunisation schedule, which included measles vaccine for 12 month olds, was implemented. Partial population immunity after 1975 can be attributed to a decrease in circulating wild measles virus in conjunction with incomplete vaccine coverage.

The 2 convenience samples may not represent the general community, but it is difficult to assess the direction and magnitude of any biases. While it is acknowledged that blood donors are healthier than the general community, it is not clear what implications this has for measles immunity in a sample of blood donors. Neither is it clear whether people who have had serum drawn for a diagnostic test of an illness not involving a rash will be more or less likely to be immune to measles than the general population. Generalisation of

Figure. Proportion of Victorians immune to measles (95% CI) by birth cohort and changes in vaccination policy by year



a. VIDRL Victorian Infectious Diseases Reference Laboratory

1968-1981 birth cohorts: 312 sera collected in 1999 and stored post diagnostic testing for a non-rash illness from patients aged 18-30 years;

1983-1993 birth cohorts: 1118 sera collected in 1999 by 3-stage random cluster survey of Victorian school students.

b ARCBS Australian Red Cross Blood Service – Victoria.

1968-1981 birth cohorts: sera from 300 healthy blood donors aged 18-30 years collected in 1999.

c NCIRS National Centre for Immunisation Research and Surveillance

1983-1993 birth cohorts: Data from 376 sera collected in 1999 and submitted to NCIRS from Victorian laboratories as part of the National Evaluation of the Measles Control Campaign

statistical inferences from the convenience samples should be made cautiously, as neither sample faithfully represents the population of young adults in Victoria. Estimates of immunity by birth cohort from the 2 samples however, were consistent and there are no published random samples of measles immunity amongst young Australian adults.

We estimated that approximately 12 per cent of young Victorian adults born between 1968 and 1974 were susceptible to measles, increasing to one in four of those born between 1975 and 1981. Herd immunity for measles is estimated to occur at a population immunity in the range of 92-95 per cent.[8] This level of immunity has been demonstrated amongst Australian primary school aged children following the 1998 Measles Control Campaign[9] but has not been demonstrated for young adults in Australia. This is illustrated in the Figure using data from the current study and a study evaluating the Measles Control Campaign in Victoria.[10] The figure also includes comparative Victorian data from the national evaluation of the Measles Control Campaign.[9] Immunity in these birth cohorts is comparable, since collection of all sera from Victoria was done in 1999, the year in which the outbreak of measles among young adults occurred.

There are 2 other reports of measles immunity amongst young people living in Victoria. Of 83 residents at the Melbourne Juvenile Justice Centre, aged 14 to 17 years (included in the 1975-81 birth cohorts), 20 per cent were susceptible to measles.[11] In a sample of 540 Australian born university health care students tested in 1997 and 1998, 96 per cent of whom were aged less than 23 years, 9 per cent were found to be susceptible to measles.[12] Neither of these samples is probably representative of the general population, and awareness of the importance of vaccine preventable diseases in the student sample may explain the lower estimate of measles susceptibility. The estimate of susceptibility among the residents of the Juvenile Justice Centre is consistent with the current study.

Available data from other Australian States also support the findings of the current study, but suggest some geographic variation in at-risk birth cohorts. A recent measles outbreak in South Australia consisted of 7 cases with a median case age of 32 years.[13] Unpublished data from a serosurvey conducted in 1997 referred to in this report, indicate that only 3 per cent of persons in South Australia born before 1975 were susceptible to measles. Data from the stored sera of 1300 patients in New South Wales show that in 1997, 14 per cent of persons aged 14 to 19 years (born 1979-83) were susceptible to measles compared with an estimated 5 per cent population susceptibility amongst people aged 20 to 25 years.[14] A recent Queensland serosurvey involving 3367 people aged 16 to 25 years (born 1972-83) whose sera had been collected in 1999, showed that approximately 16 per cent were susceptible to measles.[15]

The susceptibility of young adults to measles infection, in Victoria at least, will continue to be a problem as highlighted by the 1999 outbreak, and confirmed by this study. Indeed, a second outbreak of measles, affecting mainly young adults, has occurred in Victoria in the early months of 2001.[16] Given the success of the Measles Control Campaign and the routine 2-dose measles-mumps-rubella vaccination schedule, improving the control of measles in the general community will increasingly rely on the development of vaccination programs for young adults.[3] In addition to opportunistic vaccination of those from the at-risk birth cohorts, it may be prudent to ensure specific groups of young adults have documented evidence of 2 doses of a measles containing vaccine. These include health care workers, tertiary students, travellers, and armed forces recruits. Young adults born between 1975 and 1981 are more at risk, but probably more accessible, than the older cohort. We should make every effort to vaccinate this group before they too become harder to reach.

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Measles immunity in young Australian adults

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Abstract

Previous state-based serosurveys and recent outbreaks have indicated that young adults may be at risk of measles. To provide a national picture of immunity in adults, we tested 2126 sera from 19-49 year olds that had been opportunistically collected from laboratories across Australia, between July 1996 and November 1998. Sera were stratified into age groups based on expected levels of immunity. Sample numbers were proportional to the population size in each State and Territory. Immunity was determined using an anti-measles IgG enzyme immunoassay (EIA) according to the manufacturer's instructions. Results were compared with those on sera from 2 groups of 1-18 year olds; one group collected before the Measles Control Campaign (conducted in the second half of 1998) and the other group collected after the Campaign. Immunity was highest (98.3%) in subjects aged at least 30 years (born before 1968) reflecting greater exposure to the measles virus in these older subjects. Immunity was lowest in those aged 1-6 years (born in 1994-8; 83.6%) and 18-22 years (born in 1974-80; 88.9%). The relatively low level of immunity in 18-22 year olds is probably due to lower vaccination coverage in this group compared with younger cohorts (aged 6-17 years). These results indicate the ongoing need to improve vaccine uptake in infants and suggest that a vaccination campaign targeting young adults would be beneficial. *Commun Dis Intell* 2001;25:133-136.

Keywords: measles, immunisation, measles control campaign, young adults

Introduction

Recent outbreaks have indicated that young adults aged 18-30 years may be at risk of measles infection.[1-4] It is thought that they may have low levels of measles immunity as they are too old to have been part of the 2-dose measles–mumps-rubella (MMR) vaccination program (introduced in 1994) but have grown up in a period when exposure to wild measles virus was declining. Serological evidence to test this hypothesis is available from some jurisdictions;[5-7] however, no national data are available. To obtain a national picture of adult immunity to measles in Australia we tested sera from 19-49-year-olds that were collected as part of the evaluation of the Measles Control Campaign (MCC), conducted in the second half of 1998.

Methods

Serum samples

All major public and private diagnostic laboratories throughout Australia were invited to contribute sera that had been submitted for diagnostic testing and would otherwise have been discarded; 45 of these 52 laboratories agreed to participate. Subjects who were known to be immuno-compromised, multiply transfused, or infected with human immunodeficiency virus, or to have possible recent measles infection were excluded. Only one sample from any subject was tested. The sera available from 19-49 year olds had collection dates between June 1996 and November 1998, but were classified as a pre-Campaign sample as most (99.7%) were collected prior to the MCC.

Antibody assays

De-identified sera were tested using the Enzygnost (Behring Diagnostics, Marburg, Germany) anti-measles IgG enzyme immunoassays (EIA), at the Institute of Clinical Pathology and Medical Research (ICPMR), Sydney, Australia. Methods and interpretation of results were according to the manufacturer's instructions. Equivocal results were re-tested. Those that remained equivocal were classified as non-immune, as past experience indicated that these sera were likely to have levels of immunity lower than those associated with protection from infection.[8]

Sample size estimation

The sera were stratified into age groups with similar expected levels of immunity based on past serosurveys and each cohort's likely exposure to measles and vaccination history (Table 1). For ages 19-30 years, we wanted to be able to detect a 5 per cent difference between the current and any future serosurvey using a level of significance of 5 per cent and a power of 80 per cent.[9] For the 30-49 year age range, a precise estimate of immunity for this serosurvey for each 5-year age group was required (an absolute precision of ±3 per cent of the true value with 95% confidence).[10] The required sample size was distributed equally among years of age and sex within each age group and proportionally by the population sizes in each jurisdiction.

Statistical analysis

We determined the percentage of positive, negative and equivocal results for each age group. Ninety-five per cent

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confidence intervals (CI) were calculated for the percentage immune (positive) in each age group. The chi-square test was used to compare proportions and p values less than 0.05 were considered statistically significant. Statistical analyses were performed using Epi Info version 6.04b[11] and Confidence Interval Analysis (CIA).[12]

To provide a complete picture, we included the results of the national serosurveys previously conducted to evaluate the MCC. The sera used in these studies were from 1-18 year olds and were collected either in the 2 years before the MCC (2936 pre-Campaign sera), or the 5 months after the Campaign (2918 post-Campaign sera). A detailed report of these data can be found elsewhere.[8,13] For this analysis, we incorporated the results of the pre-Campaign sample of 18 year-olds with those for the 19-22 year age group. Immunity for 18 year-olds was unaffected by the Campaign, unlike immunity in the 12-17 year age group, and was similar to immunity in the 19-22 year age group.

For each age group we calculated the corresponding ranges for year of birth. For the pre-Campaign sample, it must be noted that the range of years in which subjects could have been born is wider than the corresponding age group as these sera were collected over a 3-year period rather than at one point in time.

Table 1. Required number of sera to be tested and actual number tested, by age group

Age group (ages in 1996-8)	Expected proportion immune	Sample size required	Sample size tested
19-22 yrs	0.87	600	596
23-25 yrs	0.90	429	435
26-27 yrs	0.94	207	130
28-29 yrs	0.94	207	147
30-34 yrs	0.95	203	205
35-39 yrs	0.95	203	200
40-44 yrs	0.95	203	203
45-49 yrs	0.95	203	210

Ethics approval

The study was approved by appropriate institutional ethics committees and the state-wide Health Confidentiality and Ethics Committee of the New South Wales Health Department.

Results

Tests were performed on sera from 2126 individuals aged 19-49 years (Table 1). For each age group, except 19-22 and 23-25 years, proportions by State or Territory of residence were comparable with those of the 1997 Australian population (Australian Bureau of Statistics). For the ages 19-25 years, there were insufficient sera from Victoria and Western Australia so we over-sampled sera from New South Wales. (Note: the proportion of positive sera for 19-25 year olds was similar in Victoria, Western Australia and New South Wales — results not shown). In addition, there were fewer sera available from the 26-27 and 28-29 year age groups than required (Table 1). As the seroprevalence was similar for these 2 groups they were combined to achieve a higher level of precision for the seroprevalence estimate.

Measles immunity

In the pre-Campaign sample of sera from 1-49 year olds, measles immunity generally increased with age to be above 95 per cent in age groups over 29 years (Figure 1, Table 2). Following the MCC, immunity increased significantly in those age groups targeted by the Campaign, namely preschool (2-5 years), primary school children (6-11 years) and high school students (12-17 years). The level of immunity did not differ significantly between males and females either before (p=0.09) or after (p=0.9) the Campaign

When we examined the most recent data available (i.e. sera collected post-Campaign for 1-17 year olds and sera

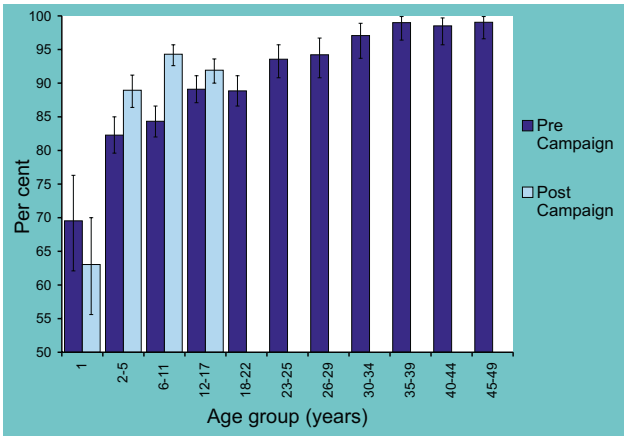
Table 2. Percentage of sera positive for measles IgG antibody before and after the Australian Measles Control Campaign, by age group

	Pre-campaign			Post-campaign			
Age group (years)	No. tested	% Seropositive (95% CI)†		No. tested	% Seropositive (95% CI)†		p value*
1	174	69.5	(62.1-76.3)	184	63.0	(55.6-70.0)	0.2
2-5	756	82.3	(79.6-85.0)	715	89.0	(86.4-91.2)	<0.001
6-11	958	84.3	(82.0-86.6)	965	94.3	(92.6-95.7)	<0.001
12-17	899	89.1	(87.1-91.1)	904	91.9	(90.0-93.6)	0.04
18-22	745	88.9	(86.6-91.1)				
23-25	435	93.6	(90.8-95.7)				
26-29	277	94.2	(90.8-96.7)				
30-34	205	97.1	(93.7-98.9)				
35-39	200	99.0	(96.4-99.9)				
40-44	203	98.5	(95.7-99.7)				
45-49	210	99.0	(96.6-99.9)				

* p value for comparison of the percentage of seropositive results pre and post-Campaign

†CI Confidence Interval

Figure 1. Percentage of sera positive for measles IgG antibody, before and after the Australian Measles Control Campaign, by age group and including 95 per cent confidence intervals



collected pre-Campaign for 18-49 year olds) the key findings were: (a) the proportion of immune subjects was high (98.3%; 95%CI: 97.2-99.1) in those 30 years of age or older (born before 1968); and (b) subjects aged 18-22 years (born in 1974-80) had a significantly lower level of immunity than an older cohort aged 23-25 years (born in 1971-5, p=0.008); and younger cohorts aged 12-17 years (born in 1982-7, p=0.04) and 6-11 years (born in 1988-93, p<0.001, Figure 2).

Equivocal results

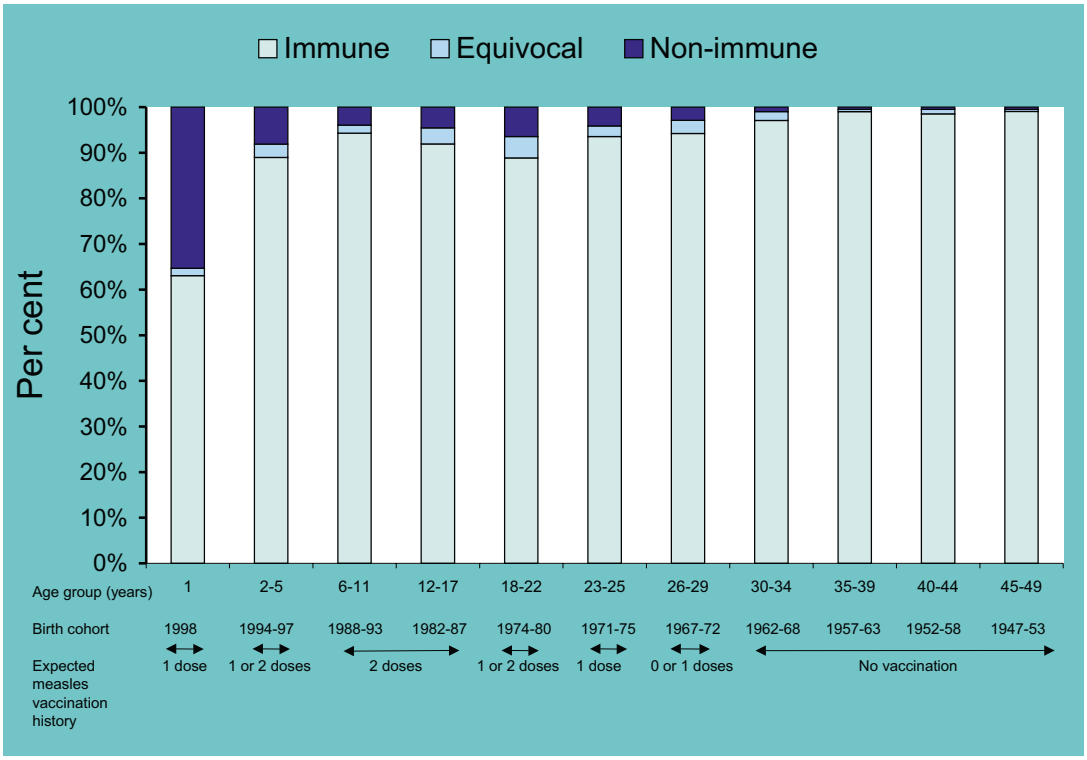
The proportion of equivocal results varied by age, but not by gender or period of collection. In the pre-Campaign sample there was an increasing proportion of equivocal results up to the age of 19 years, then a progressive decrease for older ages to below 1 per cent in the 5-year age groups over 34 years. A similar trend for 1-18 year olds was seen in the post-Campaign sample. Using the most recent data available for each age cohort, both the 12-17 and 18-22 year age groups had significantly higher levels of equivocal results than the younger and older age groups respectively (p=0.02, for comparison of 6-11 and 12-17 year age groups; p=0.04, for comparison of 18-22 and 23-25 year age groups), (Figures 2 & 3).

Discussion

The pattern of immunity found here is due to a complex mixture of interacting factors. However, the major determinants of each cohort's immunity levels are their vaccination history and past exposure to measles virus. Older aged cohorts have obviously lived longer and therefore had more time to come in contact with the measles virus, but they are also more likely to be immune due to the higher incidence of disease in the past. The high levels of immunity in those older than 28 years of age in 1996 or 30 years in 1998 are to be expected as the incidence of measles was high[14,15] prior to the approval of the measles vaccine in 1968. Before this, epidemics occurred every 2-3 years and eventually 95 per cent of the population was infected.[16]

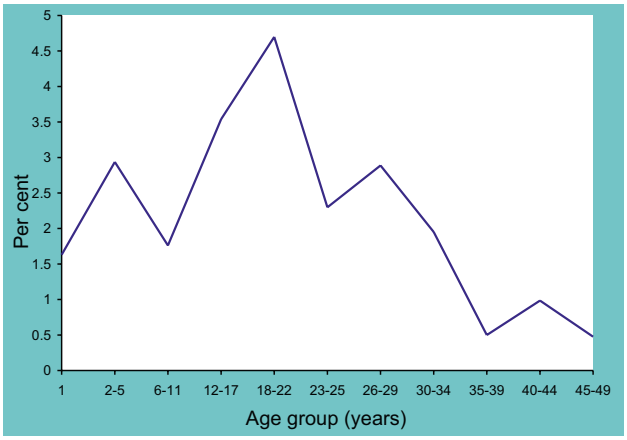
Cohorts born since measles vaccine became available have differing levels of immunity due to variations in their risk of infection and vaccination coverage. With each new birth cohort there was a reduction in risk of infection (due to a decrease in the circulation of measles virus) and an increase in vaccination coverage with one dose of measles vaccine at one year of age.[17-19]

Figure 2. Most recent* national data available for immunity to measles, Australia, by age group/year of birth and expected vaccination history



* Sera from 1-17 year olds collected in January-May, 1999 (after the Measles Control Campaign). Sera from 18-49 year olds collected in June 1996-November 1998.

Figure 3. Equivocal measles IgG results using the most recent* national data available, Australia, by age group



* Sera from 1-17 year olds collected in January to May, 1999 (after the Measles Control Campaign). Sera from 18-49 year olds collected in June 1996 to November 1998.

Those aged 18-22 years (born in 1974-80) had the lowest level of immunity of any adult age group and the highest proportion of equivocal results. This cohort has lived in a period when the incidence of measles was substantially lower than for older cohorts,* but the uptake of the first dose of measles vaccine at one year of age was still below 50 per cent.[16] In addition, most of this cohort would not have been eligible for the adolescent MMR dose given to 10-16 year olds between 1994 and 1999, unlike younger cohorts (aged 6-17 years, born in 1982-1993) who would have been eligible for 2 doses of MMR vaccine either as part of the MCC or the routine schedule for adolescents.[20] The high proportion of equivocal results compared with other ages may be due to reduced opportunities to boost immunity naturally via contact with the measles virus (compared with older ages) and a longer time since vaccination in infancy (compared with younger cohorts).

Our results appear to reflect historical changes in immunisation policies and disease incidence. Little is known however, about whether opportunistically collected sera are representative of the true level of immunity in the Australian population. Our convenience sample of sera was obtained from most major laboratories around Australia. Any selection biases are likely to be limited because these laboratories offer a wide range of diagnostic services, therefore reasons for which the sera were submitted are unlikely to differ between laboratories or over time.[8,21]

Conclusion

Based on the most recent national serosurvey data available, there are 2 cohorts with levels of immunity below 90 per cent — those aged under 6 years in 1999 (born in 1994-1999) and those aged 18-22 years in 1996-98 (born in 1974-1980). Only persons aged 30 years and over in 1996-98 (ie born before measles vaccine was available) had immunity levels above 95 per cent.

These results indicate the ongoing need to improve vaccine uptake in infants and suggest that a vaccination campaign targeting young adults would be beneficial.

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* Although there were no national notification data for measles between 1948 and the mid 1980s, data about measles admissions and cases of encephalitis in Victoria[15] and national notifications since 1991[17] suggest that the incidence of measles declined during this period.

Measles: how many hospitalised cases are we missing?

Glenda Lawrence,[1,2] Stephen Lambert,[3] Heath Kelly,[4] Ross Andrews[1]

Abstract

We aimed to determine whether the Victorian measles surveillance system had missed hospitalised cases of measles during an inter-epidemic period. We searched the Victorian Inpatient Minimum Dataset (VIMD) for the period 1 January 1997 to 30 June 1998 to identify patients with ICD-9 discharge codes for measles (055). The data were compared with that held in the Victorian measles surveillance dataset. The hospital case notes of patients identified in the VIMD but not in the measles surveillance dataset were reviewed systematically to determine whether the patients met case definitions for laboratory-confirmed or clinically compatible measles. Sixteen admissions (15 patients) were identified with a measles ICD-9 code. Eight patients were not identified in the measles surveillance dataset. Of these, one was a laboratory confirmed case of measles and two met a clinical case definition but all should have been notified to the Department of Human Services as suspected cases. While the small number of missed notifications is encouraging in terms of overall measles surveillance, it highlights important deficiencies in the awareness of hospital staff of their role in the control of measles, particularly as Australia moves towards the elimination of measles. *Commun Dis Intell* 2001;25:137-140.

Key words: hospitals, surveillance, eradication, ICD-9, measles

Introduction

Australia has moved to the elimination phase of measles eradication to arrest indigenous transmission of the virus.[1,2] Surveillance and laboratory confirmation of measles are increasingly important as incidence declines.[3]

In Victoria, measles is notifiable by both clinicians and laboratories within 24 hours of a presumptive diagnosis. In 1997, the Department of Human Services (DHS) and the Victorian Infectious Diseases Reference Laboratory (VIDRL) implemented a system of enhanced surveillance.[4,5] This has ensured that each measles notification is dealt with in a uniform manner and has greatly improved the proportion of cases who have laboratory tests performed. It does not however, provide any information about cases of measles that are not notified.

One method of assessing the ability of measles surveillance to detect all cases in the community is to review other surveillance datasets that collect information about measles cases. The Victorian Inpatient Minimum Dataset (VIMD) contains ICD-9 discharge codes for all hospital separations in Victoria. We used the VIMD to identify ICD-9 discharge codes that indicated measles as a contributory cause of the hospital admission. The major aim of the study was to assess whether the surveillance system had missed hospitalised cases of measles during an interepidemic period.

Methods

Case definitions

Case definitions for measles were those used in Victoria in the enhanced measles surveillance program.[2,4]

A laboratory-confirmed case was defined as a person who met one of the following criteria: a positive test for measles-specific IgM, or a four-fold rise in measles antibody titre in paired acute and convalescent sera, or isolation of measles virus from a clinical specimen, or a positive measles-specific PCR test of a clinical specimen.

A clinically compatible case was defined as a person with a morbilliform rash, cough and fever present at the time of rash onset who was not laboratory confirmed, because either no specimen was collected or blood was collected too early after the appearance of the rash (less than 72 hours).[6] Additional signs and symptoms consistent with a diagnosis of measles may also have been present including coryza, conjunctivitis and Koplik spots on the oral mucosa.

Data sources and analyses

The VIMD for the period 1 January 1997 to 30 June 1998 was searched to identify patients with an ICD-9 code for measles (055) as the principal or other level diagnosis. Details from the VIMD were cross-matched with the Victorian enhanced measles surveillance dataset to determine whether hospitalised cases had been notified. The surveillance database contained details of all notified cases of suspected and confirmed measles. There was no unique identifier present in both databases. The fields used for cross-matching were: age or date of birth, geographical

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proximity of notification address and hospital address, and relationship between the onset and notification dates recorded in the measles surveillance dataset and the hospital admission and separation dates recorded in the VIMD.

Ethics approval to review the patients' hospital records was obtained from the Department of Human Services' Ethics Committee. With each hospital's approval, we reviewed the records systematically and collected information about the clinical features of the illness, laboratory testing for measles and whether measles was mentioned as a diagnosis.

Results

Sixteen hospital admissions with ICD-9 codes for measles were identified in the VIMD between 1 January 1997 and 30 June 1998 (Figure).

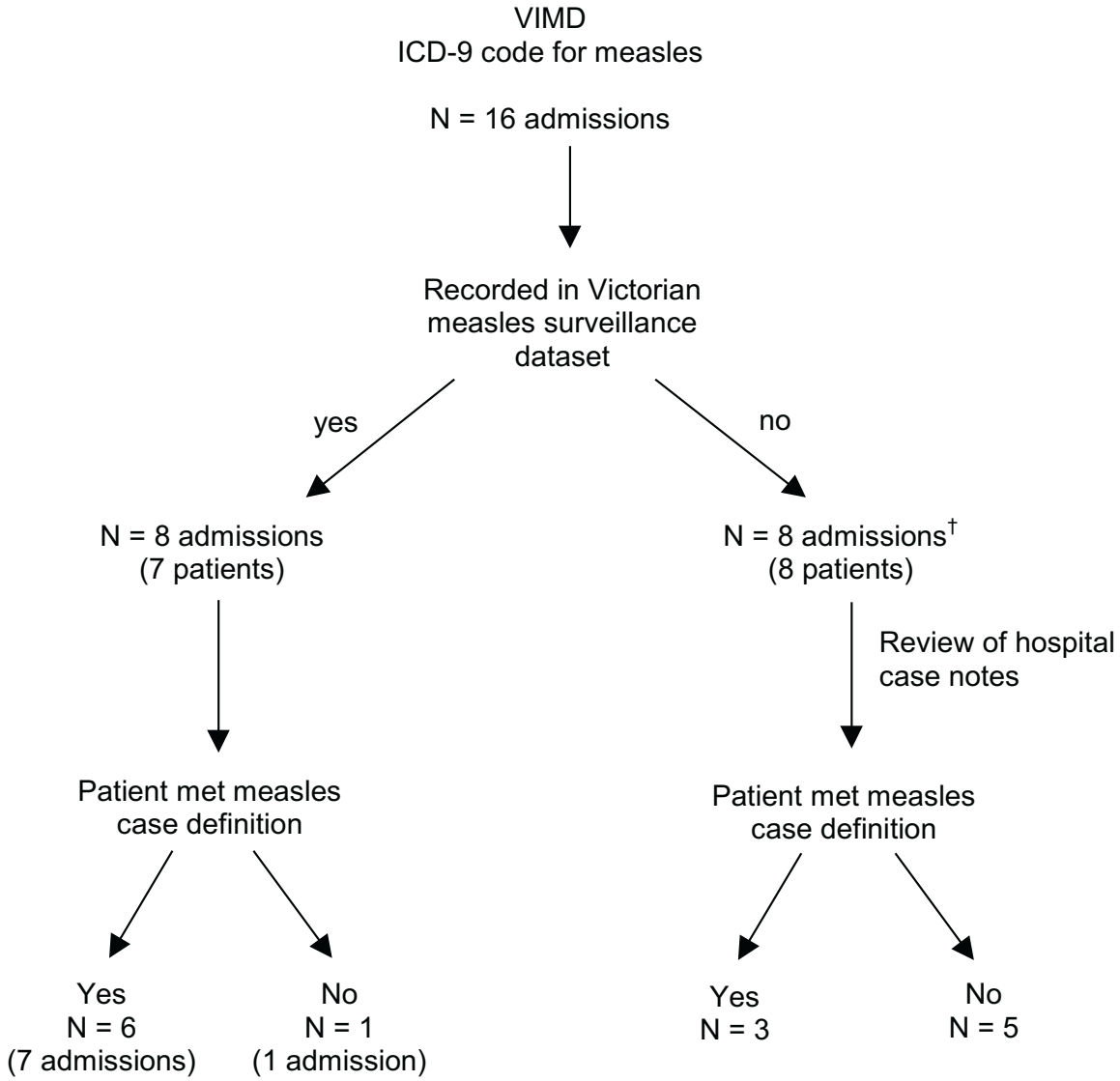
Patients reported in Victorian measles surveillance dataset

Notification records were identified in the measles surveillance dataset for 8 of the admissions, which corresponded to 7 patients (one person appeared twice in the VIMD with 2 different UR numbers). Six of these 7 patients were recorded as laboratory confirmed measles cases in the measles surveillance dataset. The seventh patient was recorded as 'laboratory rejected' because measles serology, performed at least 72 hours after the appearance of the rash, was IgM negative.

Patients not reported in Victorian measles surveillance dataset

The case notes were reviewed for each of the 8 patients who were identified in the VIMD but not in the measles surveillance dataset. The data are summarised in the Table. All had a history of fever and rash recorded in their hospital

Figure. Flow diagram of hospital admissions identified in the VIMD with an ICD-9 code for measles for the period 1 January 1997 to 30 June 1998. *



* Admissions are grouped by whether the patient was recorded in the Victorian measles surveillance dataset, and whether they met a case definition for either laboratory-confirmed or clinically-compatible measles.

† Further described in the Table.

admission notes. The ages of the patients ranged from 8 months to 24 years.

Patients 1, 2 and 3, all aged 8 months, were the only 3 of the 8 patients who met case definitions of laboratory confirmed or clinically compatible measles (Table). All 3 should have been notified as presumptive cases of measles under the Infectious Diseases Regulations of the Victoria Health Act. Patient 1 had IgM positive measles serology. There was no record of laboratory testing for measles for Patient 2, although at the time of discharge, the paediatric registrar noted that measles was the probable diagnosis. Patient 3 had a provisional diagnosis of measles recorded in the hospital notes and measles serology was performed. However the specimen, taken less than 48 hours after the appearance of rash, was negative for measles IgM and there was no evidence that repeat serology was performed.

Patient 4 was admitted to a hospital emergency department 'mildly febrile' and with a mild rash on her trunk. The medical officer recorded '?Impr: Measles' in the patient's case notes as one of several diagnoses considered at the initial medical examination. The patient was discharged after 4 hours. There was no evidence that the child met a clinical or laboratory definition of measles. The ICD-9 coding for measles appeared to have arisen from the initial notation used by the medical officer.

Patients 5, 6 and 7 were all young boys who were hospitalised with cellulitis and infected wounds following accidents (swimming, burns and skate boarding). All were given intravenous antibiotics and wound swabs from Patients 6 and 7 grew *Staphylococcus aureus*. All were febrile and had rashes that appear likely to have been related to either their infection or antibiotic treatments. None met case definitions for measles. It appears that nursing and medical notations of 'morbilliform rash' and 'measles-like rash' led to ICD-9 codes for measles being recorded for each of these patients.

Patient 8 was hospitalised with a rash and developed a fever 2 days after admission. The only mention of measles in the hospital records was in the discharge summary.

Summary

In summary, between 1 January 1997 and 30 June 1998, the Victorian measles surveillance system detected 7 hospital admissions (6 patients) who met laboratory confirmed or clinically compatible measles, but missed another three. During the same period, 21 laboratory confirmed and 17 clinically compatible cases were detected through surveillance. A further 251 suspected cases of measles were notified that, when investigated, did not meet laboratory or clinical case definitions.

Discussion

For the 18-month period analysed, the Victorian measles surveillance system detected 6 of 9 of hospitalised cases of measles identified from the VIMD. During this same period, measles transmission appears to have been interrupted, and an endemic strain was not circulating.[5,7] Five hospital admissions coded as measles in the VIMD are highly likely not to have been measles but appear to have been coded incorrectly through misinterpretation of the medical or nursing case notes or lack of more specific information in the notes. It is possible that hospitalised cases of measles were

Table. Summary of clinical and laboratory results for 8 patients with ICD-9 codes for measles in the VIMD, 1 January 1997 to 30 June 1998

Patient No.	Age	Max temp C	Rash	Rash duration (days)	Fever before rash	Cough	Coryza	Conjunctivitis	MMR vaccine	Serology result	Comment
1	8 m	39.2	+	NR	NR	-	+	+	NR	IgM pos	Laboratory confirmed
2	8 m	40.3	+	>3	+	+	+	NR	NR	NR	Clinically compatible
3	8 m	39.0	+	>3	+	+	+	+	NR	IgM neg*	Clinically compatible†
4	5 y	mild	+	NR	NR	NR	NR	NR	NR	NR	Not measles
5	5 y	37.7	+	NR	NR	+	+	NR	+	NR	Not measles
6	8 y	39.0	+	NR	-	+	NR	NR	NR	NR	Not measles
7	12 y	39.9	+	NR	NR	NR	NR	NR	NR	NR	Not measles
8	24 y	38.4	+	>3	-	NR	NR	NR	NR	NR	Not measles

\+ Present
\- Absent
NR Not recorded
* Serology performed less than 72 hours after the appearance of the rash
† Hospital records noted the appearance of 'white spots' on the oral mucosa, possibly Koplik spots

not detected in our study due to incorrect diagnosis or ICD-9 coding.

Reasons that hospital personnel did not notify the 3 cases identified in the study may include a lack of awareness among hospital medical staff of their obligation under the infectious diseases regulations and their role in the control of this highly infectious disease.[8] This has important implications in terms of measles control and surveillance as Australia moves towards elimination of measles and highlights the potential for measles transmission in health settings. Hospital inpatients in paediatric units pose an important risk group, since they may be unimmunised due to their age and/or be immunocompromised.

In the 1999 measles outbreak in Victoria, 37 per cent of cases were hospitalised.[9] At least 4 health workers became infected through patient contact, and 2 others were probably infected through indirect contact in health settings. Recently in Queensland, lack of awareness by hospital staff of measles control and prevention measures, resulted in an extended investigation to trace people who were present in an emergency department waiting room at the same time as several laboratory confirmed measles cases.[10] Our study provides further evidence of the need for education of hospital and other health professionals about the control of measles transmission in hospital and medical settings in Australia, including the importance of notifying suspected cases to public health authorities.

In conclusion, the results of the study are encouraging in terms of overall measles surveillance in Victoria but highlight some important issues in the era of elimination. These include the need to raise awareness among medical personnel of their role in the control of measles in the population, the importance of *appropriate* timing and methods of laboratory testing to confirm the diagnosis, and the lack of reliability of both clinical diagnosis and discharge coding in identifying cases of measles in health care settings.

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Editorial: Measles elimination in Australia

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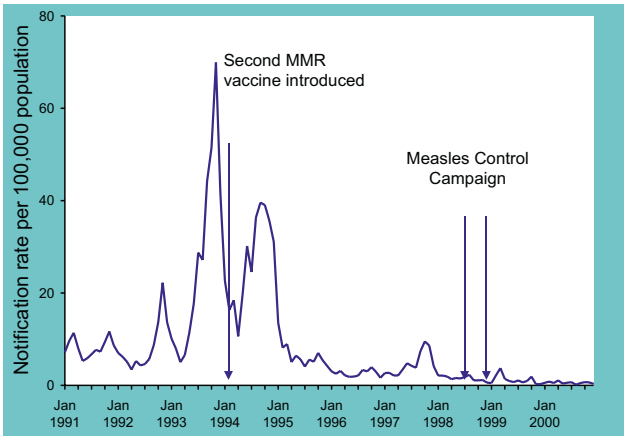
Measles is the most important cause of vaccine-preventable death in the world. In 1998, there were an estimated 30 million measles cases and 880,000 measles-associated deaths worldwide with 85 per cent of deaths occurring in Africa and South East Asia.[1] In recent years a dramatic reduction in measles incidence and the elimination of endemic measles transmission has been achieved in a number of countries using a variety of vaccination strategies.[2]

In Australia, measles reports to the National Notifiable Diseases Surveillance System (NNDSS) are at low levels as the result of important vaccination initiatives over the past few years. The Measles Control Campaign (August to November 1998) vaccinated 1.7 million children with a second dose of the measles-mumps-rubella (MMR) vaccine (Figure 1). As a result, immunity to measles among children increased from 84 per cent to 94 per cent which was estimated to have averted 17,500 cases of measles.[3]

Figure 1. Notification rate of measles, Australia, 1991 to 2000, by date of notification



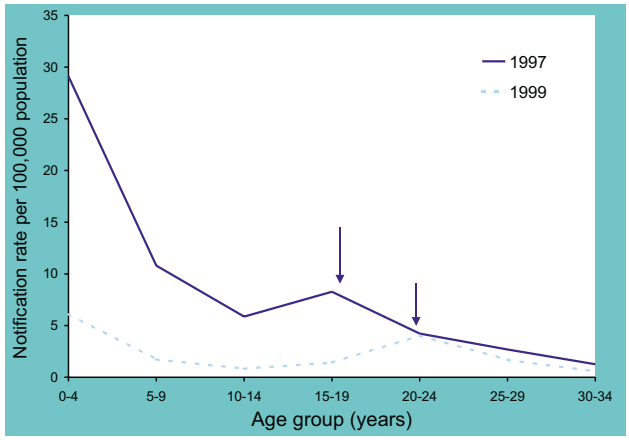
There is a 'missed middle' of young adults born between 1975 and 1981 however, who have neither been vaccinated nor been exposed to wild measles virus. In the past few years Australia has recorded measles outbreaks among young adults, often associated with an index case who has travelled to countries with high rates of measles.[4,5] This is supported by the shift seen in the peak age-specific notification rates for measles in Australia, from infants in the early 1990s to young adults at the end of the decade (Figure 2).

Measles susceptibility among young adults is discussed in 2 articles in this issue of the *Communicable Diseases Intelligence*. If interruption of endemic measles transmission is to be achieved in Australia, 92-95 per cent of the whole community must be immune to measles.[6] The question that Kelly et al and Gidding et al address is — has this level of immunity been achieved in young adults?[7,8] In Victoria, measurements of anti-measles antibodies in blood samples taken in 1999 show that fewer young adults born between 1975 and 1981 have antibodies to measles (74%) than either younger or older age groups. Nationally, 88.9 per cent of 18 to 22 year-olds are immune, compared with 98.3 per cent of those more than 30 years of age. Both studies seem to indicate that increasing the vaccination coverage of this age cohort must be a high priority for effective measles control in Australia. The recent decision to promote measles vaccination among 18-30 year-olds in Australia and to increase vaccine coverage by offering free MMR vaccine through private and public health services will be an important step towards improving coverage rates in this age group.

Hospitals should be aware of their responsibility to report cases of measles to health authorities. Lawrence and colleagues compared Victorian hospital discharge data and Victorian Enhanced Measles Surveillance data over an 18 month period in 1997-1998 and found 8 hospitalised cases of rash illness, identified as measles in the discharge summaries, not reported to the State surveillance authorities.[9] This finding highlights the fact that surveillance data are always an estimate of the total disease incidence and are usually an under-estimate. Increased professional and public awareness that measles is a disease of significant public health importance will need to be maintained to eliminate measles from Australia.

Evidence is accumulating that endemic transmission of measles in Australia is being interrupted. All measles cases in Western Australia in 1999-2000 (Dowse, Communicable Diseases Control Conference, April 2001, abstract 58) were imported from overseas or epidemiologically linked to imported cases. Using measles virus genotyping, Lambert and colleagues have shown that endemic measles virus strains are no longer circulating in Victoria. Instead, sporadic introduction of imported strains is responsible for limited focal spread (Lambert, Communicable Diseases Control Conference, April 2001, Abstract 60). If one accepts that measles elimination should be defined as a situation in which endemic transmission has stopped, sustained transmission cannot occur (because the proportion of

Figure 2. Age-specific notifications rates for measles, Australia, 1997 to 1999



susceptible people is sufficiently low), and secondary spread from importations will end naturally without intervention,[10] then Australia may have already achieved measles elimination.

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Corrections for *CDI*

The Australian Encephalitis Sentinel Chicken Surveillance report published in the April 2001 issue of *Communicable Diseases Intelligence* (*CDI*) has been reprinted in this issue due to a misalignment of the table headings (see p158).

Victorian *Legionella* commentary correction

In the previous issue of *CDI* it was reported that an outbreak of legionellosis in Victoria contributed to 38 of the 65 cases reported to the National Notifiable Diseases Surveillance System with an onset within the reporting period. While there were 38 cases of legionellosis with an onset date within this time period, the cases were not linked to a single outbreak.

There were 36 confirmed cases and one possible case of legionellosis in Victoria notified to the Department of Human Services between January and March 2001, of whom 30 (83%) were male. One case was in an overseas visitor who acquired his infection in Melbourne, 3 live and work in non-metropolitan areas, whilst 32 (89%) live and work in metropolitan Melbourne. Of the 36 cases, one case was confirmed as *Legionella longbeachae*, 32 (89%) as *Legionella pneumophila* 1 (Lp1), and the remaining 3 as unspecified *Legionella pneumophilae*. Diagnosis was confirmed by culture in 5 (14%) cases, by seroconversion in 5 (14%) cases, and by urinary antigen to Lp1 in 31 (86%) cases. Confirmation by multiple methods was made in 5 cases; one seroconversion to Lp1 and four Lp1 culture-positive cases were first identified by urinary antigen. The CBD outbreak involved 5 people, 2 of whom died, who worked or visited the same area of the city during their incubation times.

Non-TB mycobacteria

In Communicable Diseases Surveillance Highlights section of the April 2001 *CDI*, non-TB mycobacteria infection was reported as a new nationally notifiable disease. This was reported in error. This condition was previously nationally notifiable however, the National TB Advisory Committee of the Communicable Diseases Network Australia recommended its removal from the national list from 1 January 2001. It remains under surveillance in some jurisdictions.

New South Wales National Notifiable Diseases Surveillance System tables correction

The previous issue of *CDI* included no reports of cryptosporidiosis, shigellosis, influenza and invasive pneumococcal disease for New South Wales for the period of 1 January to 31 March 2001. This was incorrect. During this period there were:

52 notified cases of cryptosporidium;

32 cases of shigellosis;

7 cases of laboratory confirmed influenza; and

27 cases of invasive pneumococcal disease.

The error was due to a technical problem during data delivery. Japanese encephalitis, Kunjin virus infection and Murray Valley encephalitis are notifiable in New South Wales, however, there were no notifications for these diseases during the reporting period.

Report of the Australian Rotavirus Surveillance Program, 2000/2001

Paul Masendycz, Nada Bogdanovic-Sakran, Carl Kirkwood, Ruth Bishop, Graeme Barnes
National Rotavirus Reference Centre

Abstract

The National Rotavirus Reference Centre together with 15 collaborating laboratories Australia-wide conducted rotavirus surveillance from June 1999. The serotypes of rotaviruses that are responsible for the hospitalisation of children with acute diarrhoea were determined for the period June 2000 to May 2001. We examined 1108 rotavirus specimens using a combination of monoclonal antibody immunoassay, reverse transcription-PCR, and Northern hybridisation. Serotype G1 strains were the most prevalent overall (49.5%), and found in all centres. Serotype G9 rotaviruses, which were first identified in 1997, were second in importance (18.1%). Serotype G2 viruses were next (12.5%), followed by the re-emergence of serotype G4 viruses (9.7%). The findings of this study have implications for vaccine development strategies where protection against serotypes additional to G1-G4 may be required. *Commun Dis Intell* 2001;25:143-146.

Keywords: rotavirus, surveillance, serotype, vaccine, gastroenteritis

Introduction

Rotaviruses are the single most important cause of severe gastroenteritis in young children worldwide. The virus is responsible for the hospital admission of up to 10,000 children per annum Australia-wide.[1] The national surveillance program was designed to monitor the serotype variation of rotaviruses prior to and after anticipated rotavirus vaccine release in Australia. It supplements data from existing notification schemes which simply record the presence of the virus. Surveillance relies upon the co-operation and participation of sentinel laboratories from all States and the Northern Territory. This report covers the period June 2000 to May 2001.

Methods

Collaborating laboratories from each State and the Northern Territory undertook rotavirus detection by enzyme immunoassay (EIA) or latex agglutination. Rotavirus positive specimens were collected, stored frozen and forwarded to the Royal Children's Hospital (RCH) in 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 4 major group A human rotavirus serotypes (G1, G2, G3 and G4). Specimens with an absorbance value greater than 0.2 were considered positive for that serotype. Strains unable to be assigned a serotype were genotyped by reverse transcriptase/polymerase chain reaction (RT/PCR) using serotype specific oligonucleotide primers.[2] Northern hybridisation analysis utilising G type specific DNA probes under stringent hybridisation conditions was also employed to confirm serotype specificities.[3] Polyacrylamide gel electrophoresis confirmed the sharing of electropherotypes between collaborating centres.

Results

Number of isolates

A total of 1391 specimens were received from 15 collaborating centres. Specimens containing insufficient specimen for testing or specimens that were not confirmed to be positive for rotavirus, were omitted from the serotyping data. A total of 1108 specimens were analysed for the June 2000 to May 2001 sampling period.

Rotavirus season

The peak months of rotavirus activity for all centres are shown in Figure 1. This combines the winter epidemics experienced in southern and eastern Australia with the irregular outbreaks in the Northern Territory.

Age distribution

The age distribution of rotavirus positive patients showed that the peak incidence occurred in children aged between 6 months and 24 months (Figure 2). The male:female ratio was 1.19:1. This result is similar to the findings of the 1999/2000 collection period.[4]

Serotype distribution

Rotavirus serotypes circulating in Australia from June 2000 to May 2001 are shown in Figure 3. Overall, serotype 1 was the most common serotype nationally, being responsible for 49.5 per cent of specimens, and was the predominant type in 8 of the 16 centres studied (Brisbane, Townsville, Adelaide, Perth, Northern Western Australia, Narrabri, Melbourne and Horsham). Serotype G9 was the second most common and was responsible for 18.1 per cent of infections. Type G9 viruses were present in Melbourne, Sydney, Brisbane, Perth, Horsham, Adelaide, northern Western Australia, Darwin and Alice Springs. Type G9 viruses were found to be the most important infecting rotavirus serotype in Alice Springs during a May 2001 rotavirus outbreak.

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Figure 1. Reports of rotavirus, Australia, June 2000 to May 2001

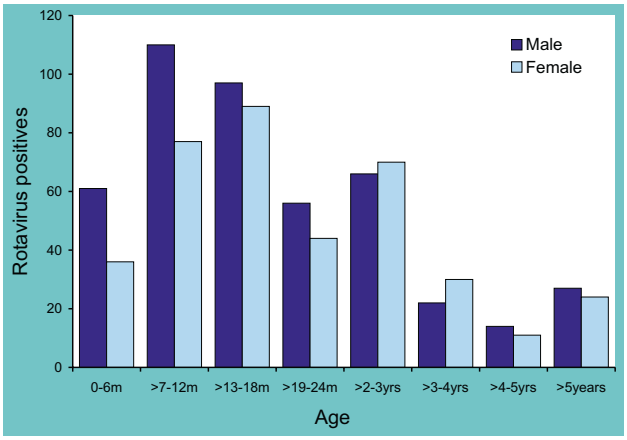


Figure 2. Reports of rotavirus, Australia, 1 June 2000 to 31 May 2001, by age

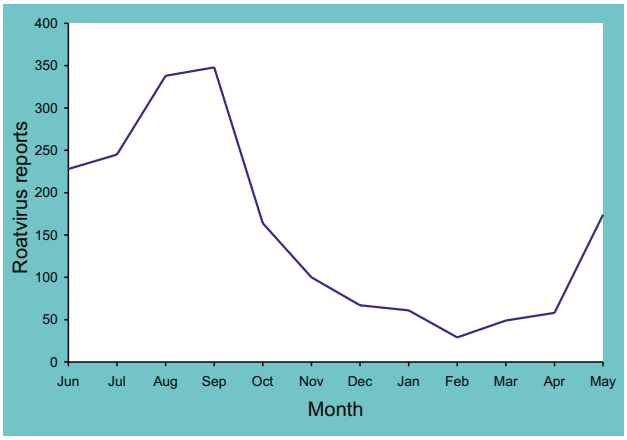
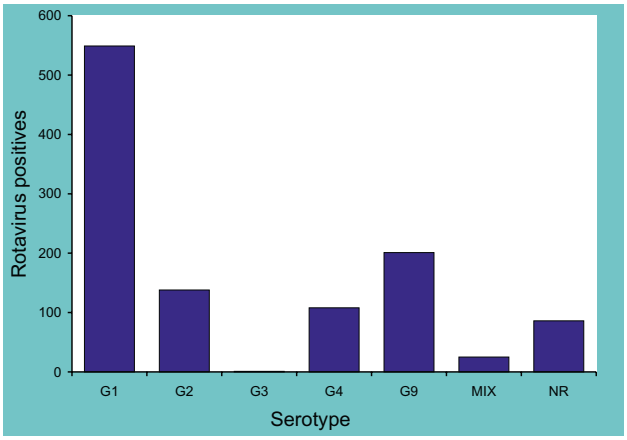


Figure 3. Reports of rotavirus, Australia, 1 June 2000 to 31 May 2001, by serotypes



Over 150 children were admitted to the Alice Springs Hospital. The epidemic was unusual in that there were 2 infecting serotypes circulating during the outbreak (G1 and G9), with G9 the most prevalent serotype (66% during May 2001). The G9 epidemic appears to have spread to Darwin at the time of writing this report (23 July 2001).

Serotype G2 was the next most common serotype, identified in all centres except Townsville and Alice Springs. Interestingly, this was the most common serotype in western Sydney (46%). Serotype G4 was identified in 8 of the 15 centres (Sydney, Narrabri, Darwin, Gove, Perth, Adelaide and Melbourne) and was the predominant serotype in 2 centres, south east Sydney (62%) and Gove (88%). Significantly, serotype G4 strains were more prevalent in 2000/2001(9.7%) than the previous 1999/2000 season (1.9%) (Table).[4]

One serotype G3 virus was detected during the sampling period. Mixed infections (2.8%) were elevated when compared to the same sampling period for 1999/2000 (less than 1%).[4] A serotype could not be assigned to 7.8 per cent of specimens. This result was lower than for the corresponding time period in 1999/2000[4] due to increased use of molecular detection techniques.

These data illustrate some important differences between the distribution of serotypes in different parts of Australia, and map the occurrence of a 'new' serotype G9 which recently emerged worldwide.

Discussion

National rotavirus surveillance from 1 June 2000 to 31 May 2001 was highlighted by 2 outbreaks reported during the sampling period, a serotype G4 outbreak in Gove in the Northern Territory's 'Top End' in September 2000, and a G9 outbreak in Alice Springs in May 2001.

Analysis of rotaviral RNA by polyacrylamide gel electrophoresis of Darwin specimens collected in August 2000 and Gove specimens collected in September 2000, showed that the strains all shared the same electropherotype. The electropherotype appeared initially in Darwin in non-hospitalised children, and appeared to move east from Darwin to the isolated communities around Gove in Arnhem Land, where children were admitted to hospital with acute gastroenteritis. A similar electropherotype was circulating in Sydney earlier in 2000, suggesting the virus had moved from Sydney to Darwin.

Alice Springs experienced a large rotavirus outbreak in May 2001. Serotype G9 was detected in children from wide-spread remote locations as far as the Western Australian and South Australian borders. The outbreak put significant pressure on existing hospital facilities, and highlights the need for a rotavirus vaccine effective against serotype G9 as well as serotypes G1 to G4. The strain appears to have its origins in Perth where it was circulating from March to May 2001. Both the Alice Springs and Perth strains shared the same RNA electropherotype. The subsequent northward spread of serotype G9 strains to Tennant Creek, Katherine and Darwin, suggests the newly emerging strain is capable of causing widespread morbidity.

G1 viruses dominated in most centres and were present in all centres studied, This is consistent with previous studies undertaken in Australia (1993-1996), [5] the United Kingdom (UK) (1996) [6] and the United States of America (USA) (1996-1997). [7]

Serotype G2 specimens were identified by RT/PCR and Northern hybridisation because our serotype G2 MAbs no longer recognise the viruses. Gene sequence analysis of the MAb binding site of the G2 virus, showed the virus possessed an amino acid change at the same position as selected antigenic variants, that were unable to bind to the neutralising MAb. The change suggests G2 viruses

Table. Reports of rotavirus serotypes, Australia, June 2000 to May 2001, by centre

Centre	Serotype							Total
	G1	G2	G3	G4	G9	Mixed	NR*	
SE Sydney	10	6		33	2		1	52
W Sydney	31	65		30	5	8	2	141
Narrabri	10	2		1				13
Brisbane	33	5			3	2	9	52
Townsville	12							12
Darwin	15	1			6		10	32
Darwin WesternPath	1	1		9	1			12
Alice Springs	65				96		6	167
Gove	1	1		22		1		25
Perth	160	7		2	38	4	11	222
WA PathCentre	67	6		1	3		16	93
Adelaide	81	5		1	1		2	90
Hobart	2	12					3	17
Horsham		2			8			10
Melbourne	59	25	1	9	38	10	21	163
Total	549	138	1	108	201	25	86	1108

* Non reacting to G1, G2, G3 and G4 monoclonal antibodies

† Alice Springs/Darwin rotavirus epidemic still continuing at time of printing

circulating in 2000/2001 are different serologically from G2 viruses from 1983.

The antigenic similarities between G4 and G9 viruses were again apparent. G9 specimens reacted with both G4 and G9 serotyping MAbs. The use of RT/PCR and Northern hybridisation confirmed serotype and clarified any serological cross-reactivities. Serotype G9 viruses were first described in Australia in 1999.[8] G9 viruses which were previously reported in India (1993-1994),[9] Bangladesh (1987-1997),[10] Malawi (1997-1998),[11] the USA (1996-1997),[7] and the UK (1996),[6] appear to persist as a major infecting serotype in Australia. Its circulation and significance should be closely monitored because of the potential impact it may have on vaccine development strategies.

The first evidence of serotype diversity within the one geographical location was noted during the 2000/2001 sampling period. Rotavirus specimens received from southeast Sydney were predominantly G4 viruses, whereas those from western Sydney appeared to be mainly G2 viruses. The reason for this difference is unclear. Continued surveillance of Sydney rotavirus serotypes should provide greater insight into rotavirus serotype diversity within a single geographical location.

The increased reliance on RT/PCR as a diagnostic tool in the study, led to a decrease in the number of non-typable specimens and a corresponding increase in the number of specimens with mixed infections. Mixed infections have been reported up to a frequency of 18.8 per cent in Ireland (1997-1999),[12] where standard rotavirus serotypes were mixed with strains that had not been identified in that country before. Mixed infections have the potential to generate reassortants resulting in new or emerging strains that may be the causative agents in major rotavirus outbreaks of the future. Judicious use of RT/PCR as an effective surveillance tool is justified.

Ongoing surveillance of rotavirus serotypes is warranted due to the impact of new and emerging rotavirus serotypes. This is particularly pertinent when considering the deleterious impact G9 rotaviruses have had on the communities in and around Alice Springs in 2001. Variation in the predominant strains in different centres is important when considering the potential impact of vaccination, and to inform the design of second and third generation vaccines.

Asia-Pacific collaboration

The National Rotavirus Reference Centre (NRRC) hosted Dr Janice Lo, a senior medical and health officer of the Department of Health for the Hong Kong Government. Janice spent 3 months with the NRRC examining the Australian Rotavirus Surveillance System. During her stay she undertook a laboratory based rotavirus surveillance course utilising a sample cohort from Hong Kong. Janice plans to submit her results to the Hong Kong Medical Journal and intends to initiate a rotavirus surveillance study in Hong Kong using the skills she acquired in Australia.

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Rotavirus positives were collected from numerous centres throughout Australia. The significant time and effort involved in the collection, storage, packaging, compiling data and forwarding of specimens was much appreciated. Without the contribution of the following key people the study would not have been possible.

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CDI Instructions for authors

Communicable Diseases Intelligence (*CDI*) is a quarterly joint publication of the Surveillance Section, Communicable Diseases and Environmental Health Branch, Commonwealth Department of Health and Aged Care and the Communicable Diseases Network Australia. Its aim is to provide timely information about communicable diseases in Australia to those with responsibility for their control. *CDI* has a particular emphasis on public health issues.

CDI invites contributions dealing with any aspect of communicable disease incidence, risk factors, surveillance or control in Australia. Submissions can be in the form of original articles, short reports, surveillance summaries, reviews or correspondence.

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The Surveillance Section, Communicable Diseases and Environmental Health Branch, has recently established a List Server to inform subscribers of the availability of new material published on the Communicable Diseases Australia Website. These include new issues of *Communicable Diseases Intelligence* (*CDI*), surveillance report updates and new data regarding Australia's nationally notifiable diseases.

The April issue of *CDI* called for expressions of interest for subscribers to this new service. The Surveillance Section arranged for these respondents to be added to the subscription list. Now that the service is established interested parties may subscribe, or unsubscribe themselves or colleagues, at any time.

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Epidemiology of malaria in Victoria 1999-2000: East Timor emerges as a new source of disease

Susan A Skull,[1,2] *Graham Tallis*[2]

Introduction

Malaria remains a global problem on a huge scale, with approximately 300-500 million cases and up to 3 million deaths annually.[1] In Australia, malaria is uncommon, with approximately 600-900 cases per year notified between 1991 and 1997. Victoria contributes approximately 80-130 notifications per year, making it the third most common State or Territory for malaria cases in Australia after Queensland and New South Wales.[2] Trends across this time period have appeared relatively stable.[2] Cases are imported and most often occur in young male travellers.[2] Deaths are infrequent.[3] Only 2 locally acquired cases of malaria have occurred in Australia since 1962; both in Queensland.[4,5] There have been no previous reports of malaria epidemiology for Victoria in the medical literature and only limited data from elsewhere in Australia. We therefore undertook to describe in detail the profile of malaria cases for Victoria during 1999 and 2000.

Methods

All cases of malaria notified to the Communicable Diseases Section, Department of Human Services, Victoria were reviewed for the period 1999 to 2000. Data were extracted from the Notifiable Infectious Diseases Surveillance (NIDS) database and where available, additional information was obtained from (non-systematically collected) hand-written records. Data routinely collected for NIDS includes notification date, malaria type, survival, country of birth, location of illness onset, primary occupation and reason for travel. Patient records were not reviewed however, data from detailed case report forms were also available for a number of cases admitted to one tertiary Melbourne hospital. Data were entered into Epi Info version 6.0 software.[6]

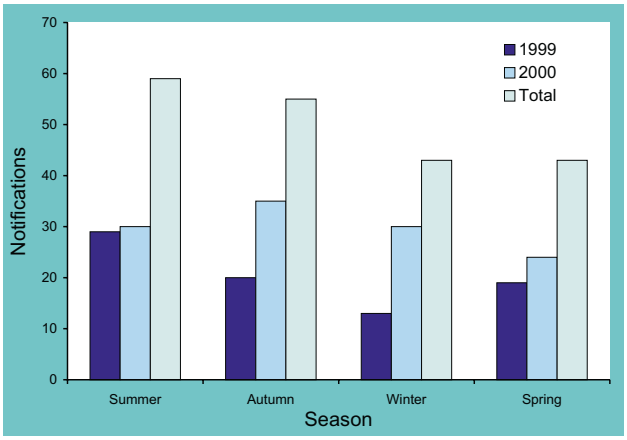
Results

There were 200 notifications for malaria in 1999 to 2000 (81 in 1999, 119 in 2000), representing an average of 2 notifications per week or 0.76 per cent and 0.5 per cent of total Victorian disease notifications respectively. Three individuals had 2 notifications during this period, of which 2 were relapses and one was a reinfection. The mean age of cases was 34 years (range 0-79 years, median 32 years). Seventy-five per cent were male. There were no deaths.

For those cases with a recorded country of birth, 57 per cent (76/133) were Australian born. The next most common countries of birth were Papua New Guinea (PNG) (7%), India (4%) and Sudan (4%). Reasons stipulated for travel outside Australia included holidays (31/98, 32%), employment or education (30/98, 31%), business (7/98, 7%) and visits to relatives (6/98, 6%). The most common reason given for entering Australia was education (72/118, 61%) followed by returning immigrants (11/118, 9%) and returning expatriates (8/118, 7%).

A seasonal trend of notification was observed (chi squared for trend p=0.03), with the greatest number of notifications occurring in the summer months (30%), followed by autumn (28%), spring and winter (22% each) (Figure).

Figure. Notifications of malaria, Victoria, 1999 to 2000, by season



Plasmodium vivax was responsible for 138/198 (70%) of all malaria notifications. Falciparum malaria contributed a further 20 per cent (40/198), *P. ovale* 13/198 (7%) and mixed infections occurred in 5/198 (2%) cases. Two notifications did not record species identification. The majority of *P. vivax* malaria originated in PNG, Indonesia or East Timor, while *P. falciparum* originated from PNG or Africa, and *P. ovale* from Africa. Confirmation of malaria species by a reference laboratory occurred in 139/200 (69%) cases.

Onset of disease in Australia, was most often (81/96, 84%) after return from malaria endemic countries. Malaria was most often acquired in PNG (72/200, 36%) followed by East Timor (26/200, 13%) and India (11/200, 5%) (Table). All but 2 cases from East Timor occurred in 2000. By region,

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Melanesia was the most common area for acquisition (80/200, 40%) due to the high rates from PNG, South East Asia (58/200, 29%) and Africa (40/200, 20%) (Table).

Employment status was known for 122/200 (61%) cases, of whom 108 (88%) were in active employment or were students. Of interest, 13/122 (11%) were health professionals. In 1999 only 4 cases were employed as either military personnel or aide workers (all in PNG). In 2000, however, this increased to 25, of whom 20 were personnel based in East Timor. This group represented 17 per cent (20/119) of all notifications in 2000 and an important new source of notifications for Victoria.

Forty-one cases answered an additional questionnaire at a tertiary hospital. Of these, 21/40 (54%) admitted seeking travel advice prior to departure to malaria endemic countries, including PNG (18/40, 44%), East Timor (3/40, 7%), Indonesia (3/40, 7%), Ghana (2/40, 5%) and Uganda (2/40, 5%). Almost half (19/41, 46%) had previously experienced malaria. Ten (52%) of these were frequent visitors to PNG. Use of insect repellent as protection against mosquito bites was reported by 13/41 (32%) cases and screens were used at night by 11/41 (27%). Antimalarial drug prophylaxis was taken by 18/40 (44%) of whom one (6%) admitted experiencing side effects. The vast majority took doxycycline (13/18). Other drugs taken were chloroquine (3) and mefloquine (2). One person took Maloprim™ in addition to chloroquine. There were insufficient data to comment on the nature of advice provided to travellers or its suitability for the regions visited, or the compliance in those who took drug treatment.

Discussion

Malaria is a rare and imported disease in Victoria. The number of notifications appears to have increased during 1999 and 2000. Disease occurs predominantly among young men, with notifications occurring most often in the warmer months of the year. *Plasmodium vivax* continues to be the most common form of malaria seen, originating most frequently in PNG, Indonesia or East Timor.

Although data are somewhat limited, there is a clear lack of uptake of advice and prophylactic treatment among cases. Only half the notified cases had sought pre-travel advice or attempted to take treatment. A similar proportion described previous episodes of malaria. In addition, as no malaria prophylactic drug treatment can guarantee protection against infection, the lack of precautions taken against mosquito bites through use of insect repellent or screens at night is also a concern.

Important subgroups affected by malaria are those who travel to and from PNG without prophylactic treatment, health professionals, and more recently, military personnel and aide workers based in East Timor. With the recent increased Australian presence in East Timor, health providers should be aware of the need to provide advice on malaria prophylaxis to those intending to visit malaria endemic countries. There remains an ongoing need to encourage travellers to all malaria-endemic areas to seek professional advice about preventive treatment against malaria, to know how to take this treatment effectively, and to use protective measures against mosquitoes in addition to drug treatment.

Table. Notifications of malaria, Victoria, 1999 to 2000, by probable country and region of infection

Region/country	1999	2000	Total
Africa			
Africa	5	2	7
Congo	0	1	1
Eritrea	0	1	1
Ethiopia	1	0	1
Gambia	0	1	1
Ghana	1	4	5
Kenya	2	1	3
Madagascar	0	1	1
Malawi	1	1	2
Nigeria	1	2	3
South Africa	0	1	1
Sudan	2	6	8
Tanzania	2	0	2
Uganda	2	1	3
Zambia	0	1	1
Total	17	23	40
South East Asia			
Indonesia	4	6	10
Bali	1	0	1
Bintan Island (Indonesia)	1	2	3
Borneo	1	0	1
East Timor	2	24	26
Flores (Indonesia)	1	0	1
Irian Jaya (Indonesia)	2	2	4
Lombok	1	3	4
Malaysia	0	1	1
SE Asia	1	1	2
Sumatra	1	0	1
Thailand	0	1	1
Vietnam	0	3	3
Total	15	43	58
Southern Asia			
Afghanistan	3	1	4
Bangladesh	0	1	1
India	4	7	11
Pakistan	2	1	3
Sri Lanka	0	2	2
Total	9	12	21
Melanesia			
Bougainville	1	0	1
Papua New Guinea	36	36	72
Solomon Islands	1	3	4
Vanuatu	1	2	3
Total	39	41	80
Central America			
Honduras	1	0	1
Total	81	119	200

From a public health point of view, it would also be useful for notifiable Diseases databases to include information on the nature of advice provided to travellers and its suitability for the regions visited. This would enable feedback for patients (many of whom are repeat travellers), as well as doctors who may not be aware of the constantly changing profile of malaria resistance in endemic countries.

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Locally-acquired *Plasmodium falciparum* malaria on Darnley Island in the Torres Strait

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Australia was declared malaria-free by the World Health Organization in 1981. Locally acquired malaria is now uncommon in north Queensland. During the 1990s, 4 cases of locally-acquired *Plasmodium vivax* malaria were reported from north Queensland, consisting of single cases in Cape York[1] and Cairns[2] and 2 on Badu Island in the Torres Strait.[3] The Torres Strait however, has a real risk of malaria transmission due to its proximity to Papua New Guinea (PNG) where malaria is endemic. We report on a locally-acquired case of *P. falciparum* malaria on Darnley Island in the Torres Strait.

The index case

The Tropical Public Health Unit in Cairns was contacted on 23 March 2001 regarding a 29-year-old man from the Torres Strait (Darnley Island, Figure) who was smear positive for *P. falciparum* malaria. The man had presented to the clinic on 19 March 2001 with headache and muscle pain, which had been present since 17 March 2001. No diagnosis was made but he re-presented for review on 22 March 2001. On that occasion he had a temperature of 40.4°C, joint pain, cough, headache and loose bowel motions. Blood films for malaria collected that day were positive with 51,937 *Plasmodium falciparum* per uL. Ring forms were present but no gametocytes (the sexual forms of the parasite) were seen. He was commenced on quinine sulphate the same day and subsequently made a good recovery.

The index case had not left the island during 2001 apart from fishing trips within a short distance offshore. More particularly, he had not visited PNG. Interviews with the patient and several acquaintances confirmed this. Therefore, he was presumed to have acquired *P. falciparum* infection on the island. Prior to symptom onset his routine in the evenings was to sit either on the beach or inside his partially-screened house. The only exceptions were on the evening of 8 March when he was at a church funeral and feast until about 22:00, and on 2 March when he was at the community canteen from about 17:30 until 02:00 the next morning.

Potential sources of *Plasmodium falciparum* malaria on Darnley Island

In 2001 there were 2 cases of *P. falciparum* malaria on Darnley Island, with onset dates prior to the index case (Table). Several PNG nationals had visited Darnley Island and could have introduced *P. falciparum*. A group of 5 PNG nationals from Kadawa village (Figure) had been on Darnley Island for 2 to 3 weeks up until 24 March 2001. A sixth member of this group, a male in his early 30s from Parama (Figure), left the island on about 11 March after developing headache and joint pain. He did not seek medical care.

A second group (father, mother, and 2 sons) from Daru Island stayed on Darnley Island from 2 to 15 March 2001. The father became unwell and was reviewed at the clinic on 13 and 14 March when he was treated empirically with quinine (Fansidar® was recommended but not taken). Malaria screening was not performed.

Two additional family groups encompassing 13 people from Kadawa village, attended a church gathering on Darnley Island in mid-December 2000 and stayed until 1 March 2001.

Entomological investigations

In addition to mosquito trapping, emergency mosquito control measures were implemented on Darnley Island to minimise the risk of further transmission of *P. falciparum*. The primary vector of malaria in Australia, *Anopheles farauti sensu lato (s.l.)*, is widespread in the Torres Strait but is generally found in low numbers on Darnley Island.[4] Darnley Island is hilly, with few swamps. *Anopheles* breeding near the community is limited to 4 small tidal creeks, a mangrove swamp and small puddles. No *Anopheles* larvae were

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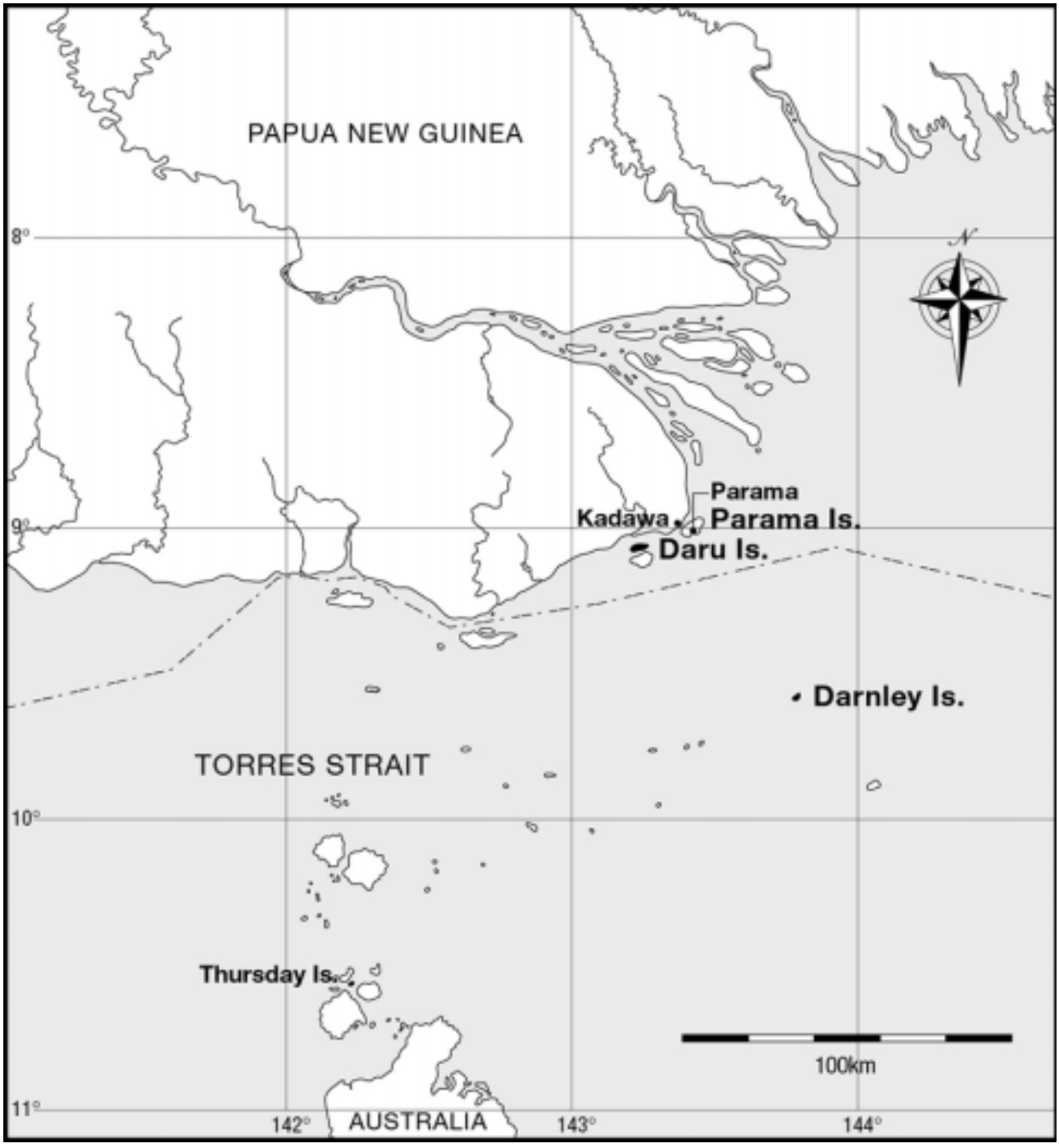
Table. Cases of *Plasmodium falciparum* malaria with onset before the index case in 2001

Characteristic	Case 1	Case 2
Sex and age	M, 11 yr 4 mo	M, 2yr 1 mo
Onset or presentation date	3 Jan 2001	9 Feb 2001
Date of first smear	4 Jan 2001	11 Feb 2001
Parasite count	40,287/uL	5,150/uL
Gametocytes	No	No
Comments	PNG national who had been on the island a few weeks.	Local, but spent April 2000 to January 2001 on Daru Island, PNG. Transferred to Thursday Island Hospital 9 Feb 2001

Figure. Map of the Torres Strait and Papua New Guinea



collected during larval surveys of the area. Adult mosquitoes were collected with CDC light traps baited with 1 kg of dry ice and octenol and set on 27 March near the index case's house and wooded areas within 1 km of the house. All traps were placed upwind of insecticidal fogging operations. Only 4 female *Anopheles* mosquitoes, consisting of 3 *An. farauti s.l.* and an *An. annulipes* were collected in the traps.

The Darnley community was fogged for mosquitoes after a public meeting to advise residents. A Dyna-Fog model 2605 Superhawk™ thermal fogger was used to apply bioresmethrin (Resilin™) at 200 mL/ha on the evenings of 26 and 27 March 2001. Most of the community was fogged from the main road using a 4WD vehicle.

Public health responses

A member of the Tropical Public Health Unit reviewed any patients who presented to the island clinic with fever between 24 and 26 March 2001. Malaria screening of a 49-year-old man and a 5-month-old girl with fevers was negative. The malaria case was discussed with island councillors, and posters warning of the risk of malaria and recommending the use of preventative measures were displayed around the villages. There have been no subsequent cases of *Plasmodium falciparum* malaria on the island at the time of writing.

Discussion

The shortest incubation period for *P. falciparum* malaria is 7 days, and 95 per cent of cases develop symptoms within one month.[5] The index case was, therefore probably infected between 10 February and 10 March 2001. The duration of sporogony (the exogenous sexual phase in the *Anopheles* vector) is 9-10 days for *P. falciparum* at 28°C.[6] Therefore, the mosquito that infected the index case would have taken an infective blood-meal between about 1 February and 1 March, or perhaps even a little earlier.

It is possible that individuals from the first 2 PNG groups mentioned above could have introduced *P. falciparum* malaria onto Darnley Island as 2 individuals sought medical attention on 11 and 13 March 2001. Depending on the onset of their symptoms and their respective incubation periods, they may have been infectious to *Anopheles* mosquitoes at the appropriate time. No members of the other 2 family groups became unwell while on the island. Depending on the human host's previous exposure and level of immunity, however, *P. falciparum* parasitaemia can produce minimal or no symptoms.[5] Thus, one of these people could have been the source of infection. Neither of the known *P. falciparum* malaria cases occurring on the island prior to the index case was likely to have been the original source of his infection because neither case was gametocytaemic. Both were treated promptly, and one was immediately evacuated from the island. It is possible that an unrecognised case of *P. falciparum* malaria occurred in one of the PNG nationals on the island. Another source of local transmission could have been an infected mosquito that arrived on a boat from PNG. Malaria-infected *Anopheles* transported on aeroplanes ('airport malaria')[2] and boats[3] have transmitted malaria.

Entomological investigations suggest that the risk of malaria transmission on Darnley Island is small. No larvae were found in the limited breeding sites sampled. Few female *An. farauti s.l.* were collected, although numbers could have been reduced by insecticidal fogging. Nonetheless, the presence of a locally-acquired malaria case indicates that malaria transmission is possible in the Torres Strait despite a small population of vectors, although the likelihood of a large outbreak is very small.

Acknowledgements

Mr Walter Lui Snr., immigration officer, is thanked for providing information on arrival and departure dates of PNG nationals on the island. All members of the council, and especially Mr Gutcheon, are thanked for assisting with assessment of the situation and facilitating mosquito control. We thank Harry Seriat and Dave Sellers for helping with mosquito trapping and fogging operations. Thanks also to Di James for drawing the map of the Torres Strait.

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Letter to the Editor

Murray Valley encephalitis

I was very interested in the paper titled *Murray Valley encephalitis virus surveillance and control initiatives in Australia*, which was published in the April 2001 edition of *Communicable Diseases Intelligence*. I would like to comment on some of the matters discussed.

The 'Forbes model' was briefly mentioned on pages 36 and 37. Since this is still frequently used to aid predictions for the occurrence of Murray Valley encephalitis (MVE) in south-eastern Australia, I would like to make some observations about this hypothesis.

Unfortunately, Forbes' paper, published in 1978, is in some places difficult to read (particularly the figures) and in others it is somewhat confusing and contradictory. I know the circumstances in which this paper was written; for the 20 years prior to 1978 I had been a close friend of Forbes and his colleague at Fairfield Hospital, Melbourne. For several years after the 1974 epidemic of MVE, Forbes tried to further refine the known association between the occurrence of MVE in the Murray Valley and excess rainfall in eastern Australia. He made a retrospective study of rainfall preceding the only three 'large' epidemics that occurred in the vicinity of the Murray River, viz in 1918, 1951 and 1974. He did not include 1956 when three cases of MVE occurred on the Murray River, because he considered this was 'a minor outbreak' compared to the others; besides the rainfall pattern prior to 1956 did not conform with those preceding the other outbreaks. Since the data he used to develop his hypothesis were only derived from three outbreaks of MVE, his conclusions must be considered to be very tenuous. If there is a firm correlation between rainfall patterns and the occurrence of MVE cases in the Murray valley, it will only be determined by observations spread over many decades to come.

I understand that Forbes' hypothesis has been used to try to predict MVE outbreaks in northern New South Wales. This is an inappropriate application of his theory, because he was only trying to use rainfall patterns which could predict epidemics of MVE 'in the region of', or 'in the vicinity of', or 'along' the Murray River. Moreover, there has been a tendency for some people to 'guess' the amount of rainfall in the watersheds of eastern Australia by simple perusal of quarterly Bureau of Meteorology maps, which depict the distribution of rainfall in decile ranges. Assessment of rainfall in this manner, does not conform to Forbes' methodology. The 7th decile range is not the same of the 7th decile value which was used by Forbes. Furthermore, he specified numbered rainfall districts from which rainfall figures were aggregated to provide total rainfall in each quarter for the four particular watersheds he selected. The rainfall in these catchments was then compared to the aggregate of the 7th decile or 70th percentile values for each of the catchments. If rainfall is not calculated in this way, Forbes' hypothesis cannot be applied legitimately.

Forbes concluded that for MVE to occur in the vicinity of the Murray River, it must be preceded by certain rainfall patterns. For all of the four main watersheds he selected, the 'pre-epidemic pluvial pattern consists of rainfall in excess of decile 7 standard for one of both quarters of the previous summer followed by similar excess rainfall in the final quarter of the year immediately preceding the epidemic'. The phrase 'one or both quarters of the previous summer' means one or both of the quarters that either precede or follow January 1 of the previous summer. Details of how rainfall figures must be computed to comply with Forbes' methodology, have recently been summarised (E Wishart, Victorian Institute of Animal Science, in the press).

On page 37 of the *CDI* paper, it is stated that the Forbes' model incorrectly predicted MVE virus activity in south-eastern Australia during the 1999/2000 and 2000/2001 seasons. However, when rainfall figures are strictly computed according to Forbes' method, his hypothesis did not predict MVE in the vicinity of the Murray River for the 1999/2000 summer but it was predictive of an epidemic in the summer of 2000/2001 (E Wishart, personal communication). Although this demonstrates that Forbes' hypothesis is unreliable, it may be still useful as an aid, since there are few, and no reliable, other predictive methods. A statement made in the same paragraph of the *CDI* paper regarding Nicholls' model, is also incorrect. Nicholls' hypothesis did not predict MVE activity in the 1999/2000 season but it was predictive for MVE in the 2000/2001 summer (E Wishart, personal communication).

My final comments are about the discussion on page 34 of the *CDI* paper, concerning the nomenclature of the Australian flavivirus encephalitities, where it is recommended to use the terms 'MVE encephalitis and KUN encephalitis'. When spelt out, 'MVE encephalitis' becomes Murray Valley encephalitis encephalitis. Surely this must have resulted from an oversight by the proof reader. The possible outcomes of MVE virus infection in humans, can be summarised as follows:

'The Murray Valley encephalitis virus (MVE virus) can commonly infect humans without producing apparent disease (subclinical infection), or it may cause a comparatively mild disease (mild infection). In a small percentage of all people infected, this mild infection may progress and result in a more serious disease of the central nervous system, which is called Murray Valley encephalitis (MVE)'.

Dr Noel Mck. Bennett
Chairman of the Victorian Arbovirus Task Force

Response to Letter to Editor:

The observations of Dr Bennett of Forbes' hypothesis are correct. The statements in the *Communicable Diseases Intelligence* article *Murray Valley encephalitis virus surveillance and control initiatives in Australia* that the Forbes and Nichols models predicted MVE in 1999/2000 are incorrect.

The comments by Dr Bennett in response to the article highlight an important aspect of predictive modelling of outbreaks encephalitic MVE infection, namely that neither model is definitive. Any model should only be used as a guide to public health planning, and neither should be used to provide a "yes/no" response to whether an outbreak will occur in a particular season. The take home messages are that the ecology of the virus is very complex, and human

outbreaks of disease are difficult to predict. Our article did not intend to promote one model over the other, but attempted to highlight the limitations of both models, which prohibit use of either as the only predictor of human disease.

Both theories are based on a very small data set, and were constructed on data when knowledge of mosquitoes and mosquito borne disease in Australia was still in its infancy. The ecology of MVE has changed since the outbreaks used by Forbes to develop his model. Since 1974, there have been dramatic changes in land and water use, as well as changes to mosquito control and public health education programs. As Bennett states, the correlation between predictive factors and outbreaks will only be determined over the coming decades. Any model will need to be progressively evaluated and refined as each outbreak occurs.

The abbreviation MVE refers to the virus, so the full form would be 'Murray Valley encephalitis virus encephalitis'. While it would be less cumbersome if the virus had a single name (as does Kunjin virus), it does distinguish the virus from the clinical outcome of infection with the virus. The terms asymptomatic MVE infection, non-encephalitic MVE infection and encephalitic MVE infection should be used.

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Correction

Professor Bart Currie was unintentionally omitted as an author on the paper entitled *Murray Valley encephalitis virus surveillance and control initiatives in Australia* published in the April issue of *Communicable Diseases Intelligence*. We apologise to Professor Currie for this oversight. The full author list is as above.

A case of Kunjin virus encephalitis in a traveller returning from the Northern Territory

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Case History

On 6 May 2001, a 67-year-old Australian born, Caucasian male presented to the Emergency Department of the Austin and Repatriation Medical Centre (A&RMC) with a 3 day history of fever, lethargy and confusion. This occurred one week after returning from a trip to the Northern Territory.

His previous medical problems included ischaemic heart disease, a repaired abdominal aortic aneurysm, hypertension, hyperlipidaemia and congestive cardiac failure. He smoked 20 cigarettes per day and had a history of heavy alcohol consumption. He had no history of diabetes.

His medications were aspirin, frusemide, lisinopril, simvastatin, and a nitroglycerol patch. Fifty years ago, he had an adverse reaction to penicillin with angioedema and an urticarial rash.

Four weeks before admission he went on a fishing trip in the Northern Territory. He travelled by road, through outback regions of Victoria, New South Wales, Queensland, the Northern Territory and South Australia, spending time in Daly River, Coolum, Darwin, Dunmarra, Avon Downs, Innaminka and Mataranka. He was away for 3 weeks and camped in tents or outside in a swag throughout the trip. He recalls numerous times where he was exposed to mosquitoes with large numbers of bites at Dunmarra. During the time away, he remained well as did his 5 travelling companions. There was no contact with any farm or non-domesticated animals.

Four days after his return to Melbourne, he developed 'flu-like symptoms. He had fever and rigours plus a mild headache. He became increasingly lethargic and was intermittently confused. He had no other features of meningism, no respiratory symptoms and no rash. Over the following 24 hours, his symptoms progressed and he was brought in to the Emergency Department of the A&RMC.

On arrival, he was febrile at 39^{O}C and his conscious state fluctuated from being fully alert and orientated to being difficult to rouse. Initial laboratory studies revealed a haemoglobin of 145 g/L (range 130-180), a white cell count of 10.8 x 10^9/L (4.0-11.0) with neutrophils making up 8.10 x 10^9/L (2.0-7.5) and lymphocytes 1.51 x 10^9/L (1.0-4.0), and a platelet count of 168 x 10^9/L (150-400). Erythrocyte sedimentation rate was 11 mm/hr (<20). There were occasional reactive lymphocytes seen on the blood film. His sodium was 128 mmol/L (135-145), his creatinine 0.120 mmol/L (0.030-0.110), a random blood glucose was 10.2 mmol/L (3.3-8.0) and the C-reactive protein (CRP) level was 1.5 mg/L (1.6-8.7). The remainder of his biochemical screen was unremarkable as were his arterial blood gases. These results were consistent with an acute infective process of a

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Table 1. Results of immunofluorescence (IF) serological investigations

Test	7 May 2001	11 May 2001	18 May 2001	19 June 2001
Flavivirus Total IF	<10	40	320	320
Flavivirus IgM IF	Negative	Indeterminate	Indeterminate	Negative
KUN Total IF	<10	10	40	40
KUN IgM IF	Negative	Negative	Negative	Negative
MVE Total IF	<10	<10	<10	<10
MVE IgM IF	Negative	Negative	Negative	Negative
JE Total IF	<10	<10	20	20
JE IgM IF	Negative	Negative	Negative	Negative

viral type. The low sodium would fit with inappropriate secretion of anti-diuretic hormone, which is known to occur in infections of the central nervous system. His renal impairment was most likely secondary to volume depletion and the elevated glucose has revealed a new diagnosis of diabetes mellitus.

Computerised tomography of his brain was performed with and without contrast and was normal. His cerebrospinal fluid (CSF) had 196 polymorphs/ l, 36 lymphocytes/ l and 5 erythrocytes/ l. Protein was 0.55 g/L (<0.45) and glucose 6.3 mmol/L (2.2-5.5). Gram stain showed no bacteria. These results fit with an acute infection of the central nervous system, such as an encephalitis or a meningitis. Three sets of blood cultures were taken.

The picture was felt to be consistent with a viral encephalitis although bacterial meningitis including *Listeria*, staphylococcal sepsis or melioidosis were considered possible. Intravenous acyclovir was commenced, as were intravenous co-trimoxazole and vancomycin, because of the penicillin allergy. Serum was sent for arboviral serology and CSF was sent for polymerase chain reaction (PCR) testing for herpes simplex virus (HSV), enteroviruses and Murray Valley encephalitis virus (MVE). Magnetic resonance imaging of his brain with gadolinium contrast and FLAIR sequence showed some age related ischaemic change but no features suggesting HSV encephalitis.

Over the following days, his conscious continued to fluctuate. A multiplex PCR for HSV, cytomegalovirus and Varicella-zoster virus was negative so the acyclovir was ceased. Enteroviral and MVE PCRs were also negative. There was no growth from any cultures of CSF or blood so the vancomycin was discontinued after 5 days. After a week his condition had improved and he stopped having episodes of confusion. He received the co-trimoxazole for a total of 10 days. He was discharged on day 17 and at follow-up has made a complete recovery.

Investigations subsequently revealed the likely diagnosis of Kunjin (KUN) encephalitis. Results of serological testing are presented in Table 1. Clear four-fold rises were seen over several bleeds in flavivirus group-specific antibody and in KUN specific antibody. A smaller rise was seen in Japanese encephalitis (JE) antibody, while MVE antibody was not detected. Serological cross-reactions between flaviviruses are common. These serological results were supported by the referral of sera to the Queensland Health Pathology and Scientific Services, who also obtained results consistent with KUN virus infection using haemagglutination inhibition assay (not shown). Serology for melioidosis, rickettsia, leptospirosis, Q fever, Ross River, Barmah Forest and Dengue fever were also negative.

Comment

KUN is a member of the flavivirus family along with MVE, JE and dengue virus. Its name is taken from one of the Aboriginal clans living near the Mitchell River in north Queensland, from where the virus was first isolated. It can occur throughout much of Australia, being most common in the Northern Territory and northern Western Australia. However, it is more prevalent than MVE in temperate southeastern areas.[1] It has been recently found to be a variant of West Nile virus, which occurs in Africa, Asia and Europe, and which recently caused an outbreak of encephalitis in New York.[2]

Approximately 15 cases of KUN encephalitis have been recorded around Australia.[1] KUN causes a clinically similar illness to MVE but is generally less severe. It can also cause a non-encephalitic illness with fever, malaise and possibly joint involvement. Its natural host appears to be wading birds but other birds and mammalian vertebrates have shown serological evidence of infection. The major vector is the freshwater mosquito *Culex annulirostris* but other mosquitoes can become involved seasonally. There is no specific treatment or vaccine available.

Flavivirus activity in Australia is monitored through mosquito monitoring programs, sentinel chicken surveillance and human disease surveillance. Detection of KUN in humans and sentinel chicken flocks in the southern States of Australia is rare. In the first half of 2001, sentinel chicken flocks seroconverted to KUN virus in northern Victoria, western New South Wales, the central and 'Top End' regions of the Northern Territory and northern Western Australia.

Diagnosis is generally made serologically with paired sera. The time between onset of symptoms and seroconversion is generally between 2 and 5 weeks.[3] Differentiating the various flaviviruses is not always possible but neutralisation or epitope blocking enzyme immunoassay can be used to separate MVE from KUN. It may also be possible to isolate the virus from an acute serum sample.[3] As diagnosis is not always straight forward, clinicians in southern States need to remember the possibility of flaviviruses in cases of encephalitis, particularly in those with an appropriate travel history. This case also serves as a reminder of the importance of mosquito born Diseases that particularly occur during the summer and autumn months and are most likely to be acquired in the northern areas of Australia.

Acknowledgement

Thanks to Dr Greg Smith, Queensland Health Pathology and Scientific Services, for additional serological testing.

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Australian encephalitis: Sentinel Chicken Surveillance Programme

This report was published in the April 2001 issue of *Communicable Diseases Intelligence* but is reprinted in this issue as the table column headings were misleading. (See the Communicable Diseases Surveillance - Additional reports section for the latest report for May/June.)

Sentinel chicken flocks are used to monitor flavivirus activity in Australia. The main viruses of concern are Murray Valley encephalitis (MVE) and Kunjin which cause the potentially fatal disease encephalitis, in humans. Currently 30 flocks are maintained in the north of Western Australia, 9 in the Northern Territory, 12 in New South Wales and 10 in Victoria. The flocks in Western Australia and the Northern Territory are tested year round but those in New South Wales and Victoria are tested only from November to March, during the main risk season.

Results are coordinated by the Arbovirus Laboratory in Perth and reported bimonthly. For more information and details of the location of sentinel chicken sites see Commun Dis Intell *2000;24:8-9.*

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January/April 2001

Sentinel chicken serology was carried out for 29 of the 30 flocks in Western Australia in January and February 2001. There were 5 seroconversions to flaviviruses in January, 3 from the Kimberley and 2 from Ophthalmia Dam (near Newman) in the Pilbara. In February the number of seroconversions increased, particularly in the Kimberley region. There were 29 seroconversions in the Kimberley and one from the town of Newman in the Pilbara. The number of chickens positive for flavivirus antibodies by ELISA at each site and the identity of the infecting virus(es) are shown in Table 1. As a result of these findings the Health Department of Western Australia issued a second health warning to residents living in these areas warning of the increased risk of infection with MVE virus. The Kimberley Public Health

Table 1. Flavivirus seroconversions in Western Australian sentinel chicken flocks, January to February 2001

	January 2001			February 2001		
Location	MVE	KUN	FLAVI	MVE	KUN	MVE/KUN
Kimberley						
Wyndham				3		4
Kununurra	1	1		4*	2	1*
Halls Creek				4		1
Fitzroy Crossing				5	1	2
Derby	1			1		
Broome				1		
Pilbara						
Newman (town)				1		
Ophthalmia Dam		1	1			

MVE antibodies to Murray Valley encephalitis virus detected by ELISA
KUN antibodies to Kunjin virus detected by ELISA
FLAVI antibodies to a flavivirus only detected by ELISA
* some results not yet confirmed

Unit has issued similar warnings to Kimberley Aboriginal communities.

Sentinel chicken serology was carried out for 28 of the 30 flocks in Western Australia in March and April 2001. There were 17 seroconversions to flaviviruses in March from the Kimberley and 33 from the Pilbara. The number of chickens positive for flavivirus antibodies by ELISA at each site and the identity of the infecting virus(es) are shown in Table 2. As a result of these findings the Health Department of Western Australia issued a third health warning to residents living in these areas warning of the increased risk of infection with MVE virus. The Kimberley, Pilbara and Gascoyne Public Health Units have issued similar warnings to Aboriginal communities. In April activity decreased significantly and there were 3 seroconversions in the Kimberley (2 MVE, 1 FLAVI) and 4 seroconversions to MVE from Ophthalmia and the Harding dams in the Pilbara. There have been no cases of MVE reported from Western Australia.

The Western Australian sentinel chicken program is funded by the Health Department of Western Australia.

Serum samples from all of the 8 Northern Territory sentinel chicken flocks were tested at the University of Western Australia in January and February 2001. There was one new seroconversion to Kunjin virus in January from Katherine. In February there was a total of 14 new seroconversions to flaviviruses. The number of chickens positive for flavivirus antibodies by ELISA at each site and the identity of the infecting virus(es) are shown in Table 3. In addition there

Table 2. Flavivirus seroconversions in Western Australian sentinel chicken flocks, March to April 2001

	March 2001				April 2001	
Location	MVE	KUN	MVE/KUN	FLAVI	MVE	FLAVI
Kimberley						
Kalumburu						1#
Wyndham					1#	
Halls Creek	3				1	
Fitzroy Crossing	1		1			
Derby			1	1		
Broome	8		1			
Lombadina	1					
Pilbara						
Port/South Hedland	6		1			
Karratha	2					
Harding Dam	5				1#	
Marble Bar	1					
Pannawonica	2					
Tom Price	8					
Ophthalmia Dam	2	1			2	
Newman town	1		1			
Exmouth	3					

MVE antibodies to Murray Valley encephalitis virus detected by ELISA
KUN antibodies to Kunjin virus detected by ELISA
FLAVI antibodies to a flavivirus only detected by ELISA
some results not yet confirmed

Table 3. Flavivirus seroconversions in the Northern Territory sentinel chicken flocks, January to February 2001

	January 2001	February 2001			
Location	KUN	MVE	KUN	MVE/KUN	FLAVI
Howard Springs				1*	
Katherine	1		1*		1*
Tennant Creek		2		2	
Alice Springs		3*	1*	2*	1*

MVE antibodies to Murray Valley encephalitis virus detected by ELISA
KUN antibodies to Kunjin virus detected by ELISA
FLAVI antibodies to a flavivirus only detected by ELISA
* some results not yet confirmed

were 2 suspected cases (subsequently confirmed) of encephalitis caused by MVE virus from Alice Springs. Health warnings have been issued by the Territory Health Department.

Serum samples from all of the 8 Northern Territory sentinel chicken flocks were tested at the University of Western Australia in March and April 2001. There were 11 new seroconversions to flaviviruses in March and 5 In April. The number of chickens positive for flavivirus antibodies by ELISA at each site and the identity of the infecting virus(es) are shown in Table 4. A new case of MVE, with a date of onset in March 2001, was reported and the patient's travel history suggests the infection was acquired in the Northern Territory.

For the first time since 1974 there have been seroconversions to MVE and Kunjin viruses in the New South Wales sentinel chicken flocks. Flavivirus activity was detected in 7 of the 12 flocks with 8 seroconversions occurring in January 2001 and 5 in February 2001. The number of chickens positive for flavivirus antibodies by ELISA at each site and the identity of the infecting virus(es) are shown in Table 5. To date there have been no cases of encephalitis caused by MVE virus reported from the region.

Flavivirus activity was again detected in New South Wales in March 2001 but not in April. MVE virus antibodies were detected in 2 of the 12 flocks and Kunjin virus antibodies in 8 of the 12 flocks. The number of chickens positive for flavivirus antibodies by ELISA at each site and the identity of the infecting virus(es) are shown in Table 5. To date there have been no cases of disease caused by MVE or Kunjin viruses reported from the region.

In addition to the activity in New South Wales there have also been 12 seroconversions to Kunjin virus in 4 of the 10 Victorian sentinel chicken flocks in February 2001. Kunjin virus antibodies were detected in 4 chickens at Mildura, 5 chickens at Tooleybuc, 2 chickens at Barmah and 1 chicken at Barooga. The last Kunjin virus seroconversions in Victoria prior to this season were reported in March 1998 from the Mildura flock.

There were 7 new seroconversions, all to Kunjin virus, reported from the Victorian sentinel chicken flocks in March 2001. Kunjin virus antibodies were detected in 3 chickens at

Table 4. Flavivirus seroconversions in the Northern Territory sentinel chicken flocks in March and April 2001

	March 2001				April 2001	
Location	MVE	KUN	MVE/KUN	FLAVI	MVE	FLAVI
Howard Springs		1				
Leanyer						1
Coastal Plains					1	
Katherine		1	1	2		
Tennant Creek	6				2#	
Alice Springs					1#	

MVE antibodies to Murray Valley encephalitis virus detected by ELISA
KUN antibodies to Kunjin virus detected by ELISA
FLAVI antibodies to a flavivirus only detected by ELISA
some results not yet confirmed

Table 5. Flavivirus seroconversions in New South Wales sentinel chicken flocks, January to March 2001

	January 2001			February 2001		March 2001		
Location	MVE	KUN	MVE/KUN	MVE	KUN	MVE	KUN	MVE/KUN
Menindee	2	1	1					
Macquarie Marshes	2			1		1	5	
Wanaaring	1		1				1	
Griffith					1		1	
Bourke					1	1	5	1
Ford's Bridge					2		1	
Deniliquin							1	
Lake Cargelligo							1	
Leeton							9	

MVE antibodies to Murray Valley encephalitis virus detected by ELISA
KUN antibodies to Kunjin virus detected by ELISA
FLAVI antibodies to a flavivirus only detected by ELISA

Mildura, 3 at Tooleybuc and 1 at Kerang. Two further seroconversions to Kunjin virus were detected from Kerang in late April. There have been no cases of disease caused by either MVE or Kunjin viruses reported from the region. The sentinel chicken surveillance programs in both Victoria and New South Wales have been extended until May 2001.

Details of the locations of all chicken flocks are given in Spencer JD, Broom AK, Buick TD, Daniels PW, Doggett SL, Hapgood GD, et al. Murray Valley encephalitis virus surveillance and control initiatives in Australia. *Commun Dis Intell* 2001;25:33-48.

Communicable Diseases Surveillance

Presentation of NNDSS data

With the move to a quarterly reporting system in *Communicable Diseases Intelligence*, the summary tables have changed to fall in line with a quarterly report. Table 2 presents 'date of notification' data, which is 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. Table 3 presents the notification rate of Diseases by State or Territory for the current reporting quarter.

Table 2 now includes the following summary columns: current quarter totals, totals for the previous quarter; total for the same quarter in the previous year; a 5-year mean for the same quarter, the year to date total for each disease, the mean of the last 5 years year to date totals and the ratio of the current quarter to the mean of to the mean of the second quarter for the last 5 years.

Notifiable Diseases 2001

The Communicable Diseases Network Australia has revised the list of Diseases that are reportable to the NNDSS. All jurisdictions are working towards reporting against the new national list. Transmission of a dataset consistent with the new list will depend upon changes to public health legislation and IT system development. The following new diseases have been added to the NNDSS database: anthrax, Murray Valley encephalitis, Kunjin virus infection, cryptosporidiosis, influenza (laboratory-confirmed), Australian bat lyssavirus infection and invasive pneumococcal disease (laboratory-confirmed). Data on the following diseases will no longer be collected: chancroid infection, hydatid disease, lymphogranuloma venereum, non-TB mycobacterial infections, and yersiniosis.

Highlights for 2nd quarter, 2001

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 who have formed a Data Management Network. This additional information has enabled the reporting of more informative highlights each month.*

The NNDSS is conducted under the auspices of the Communicable Diseases Network Australia, and the CDI *Virology and Serology Laboratory Reporting Scheme (LabVISE) is a sentinel surveillance scheme. 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'.*

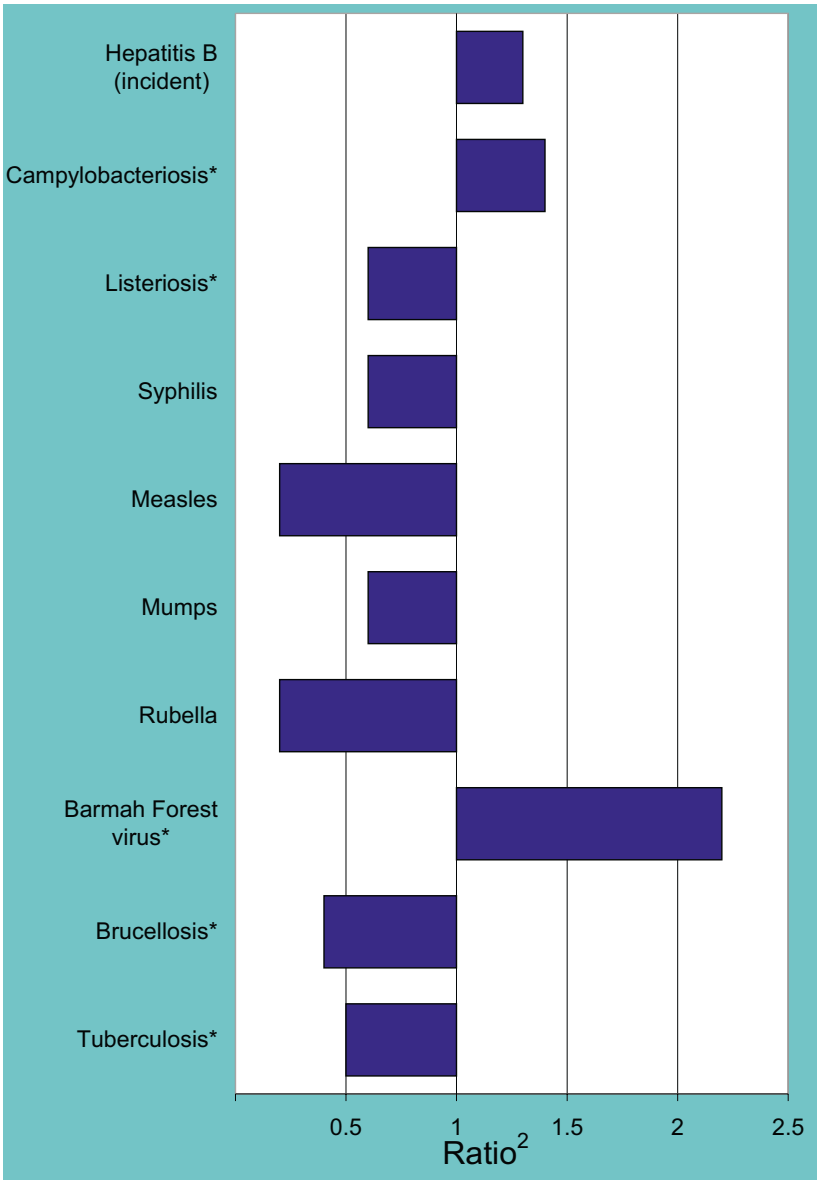
Three types of data are included in National Influenza Surveillance, 2001. These are sentinel general practitioner surveillance conducted by the Australian Sentinel Practice Research Network (ASPREN), the Department of Human Services (Victoria), the Department of Health (New South Wales) and the Tropical Influenza Surveillance Scheme, Territory Health Services (Northern Territory); laboratory surveillance data from the Communicable Diseases Intelligence *Virology and Serology Laboratory Reporting Scheme (LabVISE) and the World Health Organization Collaborating Centre for Influenza Reference and Research; and absenteeism surveillance conducted by Australia Post. For further information about these schemes, see* Commun Dis Intell *2000;24:9-10.*

Figure 1 shows the changes in disease notifications compared with the 5-year second quarter mean. Disease notifications above or below the 5-year mean, plus- or minus- two standard deviations are marked with an asterisk. These and other disease trends are discussed below.

Bloodborne diseases

Incident (acute) hepatitis B notifications were above the normal range for the second quarter compared with the 5-year mean. In this quarter, 43 notifications were received from Victoria compared with 29 in first quarter. The Victorian incident hepatitis B notification rate rose from 2.4 per 100,000 in the first quarter 2001 to 3.6 per 100,000 population in the second quarter. Injecting drug use has been identified in 65 per cent of the notified cases in Victoria (year to date). The Victorian Department of Human Services has started an enhanced acute hepatitis B surveillance program to obtain more detailed risk factor information to inform prevention strategies. A public health alert has been released through the Needle and Syringe Program to inform intravenous drug users of harm minimisation strategies and the need for primary prevention through vaccination.

Figure 1. Selected[1] Diseases from the National Notifiable Diseases Surveillance System, comparison of provisional totals for the period 1 April to 30 June 2001 with historical data[2]

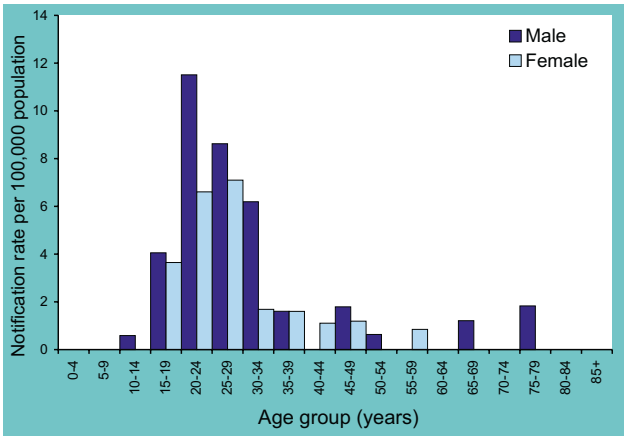


1. Selected Diseases are chosen each quarter according to current activity.
2. Ratio of current quarter total to mean of corresponding quarter for the previous five years.

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

Figure 2 shows the notification rate of incident hepatitis B infections reported in this quarter by age group and sex. Age- and sex-specific rates per 100,000 population show a male preponderance in the 20-34 year age range.

Figure 2. Notification rate of incident hepatitis B, Australia, 1 April to 30 June 2001, by age group and sex



Gastrointestinal disease

In this quarter we report for the first time, cases of shigellosis from New South Wales, where the disease has now become specifically notifiable. Previously, shigellosis cases in New South Wales were reported as 'foodborne disease' or 'gastroenteritis in an institution'. Campylobacteriosis is not a specific notifiable disease in New South Wales. In this quarter cryptosporidiosis was reported from all jurisdictions except Tasmania.

Botulism

A case of infant botulism was reported from Queensland in the second quarter. A 10 week-old infant presented with acute flaccid paralysis (prominent bulbar weakness). Subsequently, *Clostridium botulinum* type B was isolated from faeces. The infant had a history of probable consumption of honey within the 2 weeks prior to onset. New infant feeding guidelines, currently under review by the National Health and Medical Research Council, advise that infants under 1 year of age not be fed honey.

Campylobacteriosis

Notifications of campylobacteriosis in the second quarter 2001 were above the range of 5-years' data for the second quarter. Campylobacteriosis is now the major cause of sporadic gastroenteritis in Australia and is more than twice as commonly reported as salmonellosis. This pattern is found throughout industrialised countries.[1] Despite the large number of cases, outbreaks of campylobacteriosis are rarely identified (see OzFoodNet report for first quarter 2001 in this issue, pp103-106).

Cryptosporidiosis

Cryptosporidiosis became nationally notifiable with effect from January 2001. Cryptosporidiosis is spread by a faecal-oral route and includes person to person, animal to person, waterborne and foodborne transmission. The prevalence of infection is between 1 and 4.5 per cent of individuals in developed countries and 3 to 20 per cent of individuals in developing countries.[2] Children under 2 years of age, animal handlers, travellers, and men who have sex with men are recognised to be at greater risk of infection.

Infections with *Cryptosporidium* may be asymptomatic and carriers may shed oocysts in their faeces. The infective dose is very small (approx 100 oocysts) and previous exposure in immunocompetent adults is not entirely protective, although it may decrease the severity of the disease caused by subsequent infections. People with markedly impaired immune systems due to HIV/AIDS infection are susceptible to severe persistent diarrhoea caused by cryptosporidiosis and the infection may spread to the biliary tract. Declines in the prevalence of cryptosporidiosis in HIV/AIDS patients treated with highly active anti-retroviral therapy have been reported.[3]

During the early part of this quarter sporadic cryptosporidiosis infections, possibly associated with use of swimming pools, were reported from several jurisdictions in Australia. Victoria continued to observe increased notifications of cryptosporidiosis compared to previous years, predominantly confined to the Melbourne metropolitan area. (Cryptosporidiosis became notifiable in Victoria from 16 May 2001, prior to which notifications were received from doctors and laboratories on a voluntary basis.) The majority of cases reported exposure to public swimming pools before becoming ill. Small clusters were associated with several pools.

Figure 3 shows the notification rate for cryptosporidiosis by age group and sex for this quarter. More than half of all notifications in this quarter were in children aged less than 5 years. There was no difference in the notification rate between males and females.

Last summer in the United States of America, 5 outbreaks of cryptosporidiosis associated with swimming pool use were reported. The Centers for Disease Control and Queensland Health have published guidelines on the education of swimmers and pool staff, pool design modifications and improved operation and maintenance procedures in order to prevent outbreaks,[4] see also http://www.qld.health.gov.au/healthyliving/.

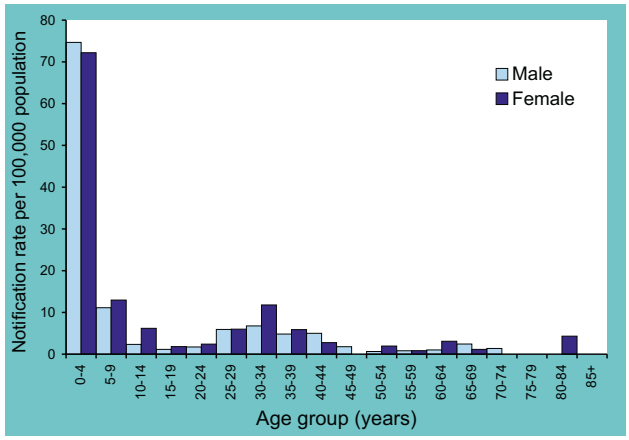
Salmonellosis

Several outbreaks of *Salmonella* Typhimurium infection were reported from around the country. In South Australia, *Salmonella* Typhimurium PT126 was recognised as a cause of gastroenteritis in 15 people in the early part of the year. This outbreak was associated with the consumption of custard fruit tarts (see OzFoodNet first quarter report, this issue). Since the beginning of May, another 34 cases have been identified in South Australia but no food source has yet been identified. Investigations are continuing.

Gary Dowse, Medical Epidemiologist from the Communicable Disease Control Branch, Health Department of Western Australia, reported on an outbreak of *Salmonella* Typhimurium PT64. 'An outbreak of Salmonellosis associated with eating fried ice cream at a Perth restaurant was reported from Western Australia in June. Over 30 patrons reported being ill, with a relatively short incubation period and several being hospitalised, indicating the food was heavily contaminated. *Salmonella* Typhimurium PT64 was isolated from faecal specimens from 20 patrons, 2 remaining serves of fried ice cream and 1 asymptomatic food handler. Cases were infected over several days, apparently from the same pre-prepared batch of fried ice cream. Preparation involved coating the ice cream with a layer of sponge cake, which was then dipped in an egg mix and frozen. Serves were removed from the freezer and deep-fried for a short period, when required. The means of contamination was not identified. This is the second outbreak of *Salmonella* food poisoning associated with eating fried ice cream reported from Western Australia in recent years. An outbreak associated with fried ice cream has also been reported previously from New South Wales.[5,]

Health Department officials in Victoria investigated a cluster of 14 cases of *Salmonella* Typhimurium PT104 that were notified between February and July 2001. Following reports of a similar outbreak in Sweden, the source was identified as 2 brands of 'Helva', a type of sweet made from sesame seeds, sugar and flavourings that had been imported from

Figure 3. Notification rate of cryptosporidiosis, Australia, 1 April to 30 June 2001, by age group and sex



Turkey. The Australia New Zealand Food Authority coordinated a national recall of the 2 products.

Two cases of *Salmonella* Typhimurium PT99 in southern Victoria were associated with an outbreak of gastroenteritis epidemiologically linked to the consumption of lambs fry at a local hotel buffet in early June. Further cases in the region are currently being investigated.

In Queensland, more than 30 cases of *Salmonella* Bovis-morbificans PT32 were reported in June. A link was made to consumption of a particular product from a major fast food chain. The outbreak is currently under investigation and further details will be provided on completion of investigations.

Quarantinable diseases

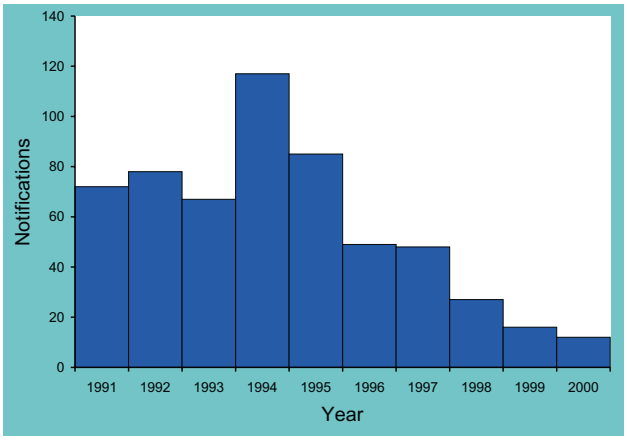
No quarantinable Diseases were reported in Australia in the second quarter of 2001.

Sexually transmitted infections

A review of syphilis notifications in Queensland is being carried out to distinguish new cases from re-tests and to remove duplicates. Hence no syphilis data were available for Queensland in this quarter which explains the low number of notifications of syphilis in this report.

Eight cases of donovanosis were reported in this quarter. All cases were from the Northern Territory, Western Australia or Queensland. Donovanosis is a notifiable disease in all jurisdictions except South Australia. Donovanosis is a chronic genital ulcer disease that generally occurs in indigenous Australians in rural and remote communities. Notifications of donovanosis have fallen significantly over the past 10 years (Figure 4), and particularly since 1994 due to the introduction of more sensitive and acceptable testing methods and more effective treatment with azithromycin. The Office for Aboriginal and Torres Strait Islander Health (OATSIH) is designing a donovanosis eradication plan based on strengthening primary health care services in rural and remote areas to provide early diagnosis and treatment for donovanosis. Laboratory confirmation using sensitive polymerase chain reaction (PCR) methods, the use of standard treatment protocols, active case follow-up and enhanced surveillance are also important aspects of the eradication plan. Enhanced surveillance for donovanosis will include continued passive surveillance in all States and Territories, active surveillance in local areas, laboratory notification in Western Australia and the Northern Territory and standardised data collection protocols. The impact of this program over the next few years may be to initially increase notifications of donovanosis to the NNDSS. There have been 10 notifications of donovanosis in total in 2001 compared with 12 notifications in all of 2000.

Figure 4. Notifications of donovanosis, Australia, 1991 to 2000, by date of notification



Vaccine preventable diseases

Laboratory-confirmed influenza and invasive pneumococcal disease are newly notifiable vaccine preventable diseases in 2001. Data were received from all jurisdictions except Victoria, Queensland and South Australia. Influenza was added to the list of notifiable diseases in these jurisdictions at the end of the first quarter and data will be available from the third quarter of 2001. Administrative changes to include influenza as a notifiable disease are under way in the Australian Capital Territory.

Invasive pneumococcal disease data were available from all jurisdictions except Tasmania and South Australia, where surveillance has only recently commenced.

Measles, mumps and rubella notifications were all reduced compared with the 5-year mean of second quarter notifications. This decline reflects the continuing impact of the Measles Control Campaign in 1998 when 1.7 million children in Australia received the measles-mumps-rubella vaccine.

No measles cases were reported from the Australian Capital Territory, the Northern Territory, South Australia or Victoria. There were single cases of measles reported from Western Australia, Queensland and Tasmania. Both the Western Australian and Queensland cases were infected overseas. A cluster of 7 cases of measles was reported from western Sydney. The first case possibly acquired the infection while travelling overseas. Five of the 7 cases were laboratory confirmed. Three cases were infants aged between 8 and 12 months, and the other 4 were in young adults aged 19 to 26 years, who were unlikely to have been vaccinated against measles.

A measles outbreak in Papua New Guinea (PNG) in late June prompted a warning from the Communicable Diseases Network Australia (CDNA) to travellers to PNG to consider measles vaccination. The media release warned doctors and health care workers to be alert for measles in people returning to Australia from PNG (CDNA media release 01/01, 23 July 2001).

Vectorborne diseases

Murray Valley encephalitis and Kunjin viral infection are now notifiable Diseases in all jurisdictions except the Australian Capital Territory, where such infections are combined under Murray Valley encephalitis.

Murray Valley encephalitis

Two cases of Murray Valley encephalitis (MVE) virus infection, which occurred in the first quarter of 2001 and were not previously reported, have been noted in reports to the Communicable Diseases Network Australia. Since the NNDSS analysis is by date of notification, delays in reporting mean that these cases do not appear in Table 1.

The first case was in a 59-year-old man from South Australia who acquired the infection in the Northern Territory. The second case, in a German tourist who was infected in the Northern Territory at the end of April, was reported on ProMED-mail in May 2001. This 23-year-old man developed viral encephalitis on his return to Germany, presented as febrile and disoriented and suffered repeated convulsions. An acute flavivirus infection was suggested and MVE was diagnosed serologically. Confirmation was provided by Dr Dominic Dwyer's laboratory at ICPMR in Sydney (ProMED Viral enceph., imported – Germany ex Australia (03) 20010524.0252).

Kunjin virus infection

Three cases of Kunjin were reported in this quarter. In 2 cases the date of notification of disease was in the first quarter and reported in the second quarter, thus not appearing in Table 1. These cases were resident in Western Australia and the Northern Territory. A third case, from Victoria, was notified to State authorities in June, but the report was not received in the NNDSS before the end of the quarter. Delays in reporting may be considerable in diseases with insidious onset or where symptoms mimic other diseases and several infections are considered in the differential diagnosis. Delays may also occur when the definitive serological tests are not widely available. Since Kunjin virus infection is a newly notifiable disease this year, delays in reporting may also occur because of a lack of awareness among reporting laboratories and doctors.

The Western Australia case presented with a 4 month history of aching joints and tiredness, so the date of onset was estimated to be around Christmas 2000. This case was confirmed serologically. The Northern Territory case occurred in a 23-year-old female. The third case reported from Victoria, had a history of travel in outback New South Wales, South Australia, Queensland and the Northern Territory during the incubation period.

Malaria

Four cases of *Plasmodium falciparum* malaria in Sudanese refugees were reported from Tasmania. These occurred in a family group who appear to have acquired the disease in Angola.

Barmah Forest virus

Reports of Barmah Forest virus (BF) infections in this quarter were above the range of notifications based on the last 5 years data. Increased numbers of notifications from New South Wales (255 YTD compared with 191 in 2000) and Queensland (440 YTD compared with 333 in 2000) were noted. National notifications for April and May were the highest ever recorded for those months and the numbers for June were the highest for that month since 1995. A comparison of notifications by month for the first 6 months shows higher BF notifications throughout this period in 2001 (Figure 5). Barmah Forest virus infections were largely in adult populations (96% of notifications in this quarter were in persons aged 20 years or more) and affected men and women equally (Figure 6).

Zoonoses

Among the zoonotic Diseases reported to NNDSS, data were available from all States and Territories for all diseases with the exception of anthrax and ornithosis. Anthrax is not yet a notifiable disease in South Australia. Ornithosis was only made a notifiable disease in Queensland at the end of June 2001.

There were only 2 cases of brucellosis reported to the NNDSS in this quarter, both from Queensland. This was a significant decrease from the 5-year mean for this quarter.

Q fever

Notifications of Q fever, though within the range of the last 5 years' notifications, show an increase in Victoria (24 YTD compared with 23 for all of 2000) and Queensland (228 YTD compared with 334 for all of 2000). There was an outbreak of Q fever linked to an abattoir in northern Victoria, with a total of 21 confirmed cases. Screening and vaccination of susceptible employees was undertaken.

This increase in Q fever notifications may be associated with increased public awareness and testing before vaccination, in occupational groups. Abattoir workers who are at high risk of infection are eligible for the vaccine, funding for which was recently provided to the States and Territories by the Commonwealth, under the National Q Fever Management Program. A breakdown of notifications by age and sex (Figure 7), shows a strong male preponderance (male to female ratio 4.4:1) and infection mainly in adults (92% in persons aged more than 20 years).

Figure 5. Notifications of Barmah Forest virus, Australia, January to June 1999 to 2001, by date of notification

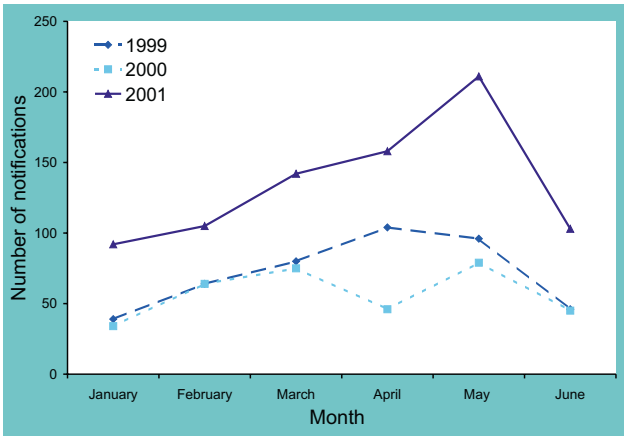


Figure 6. Notifications of Barmah Forest virus, Australia, 1 April to 30 June 2001, by age and sex

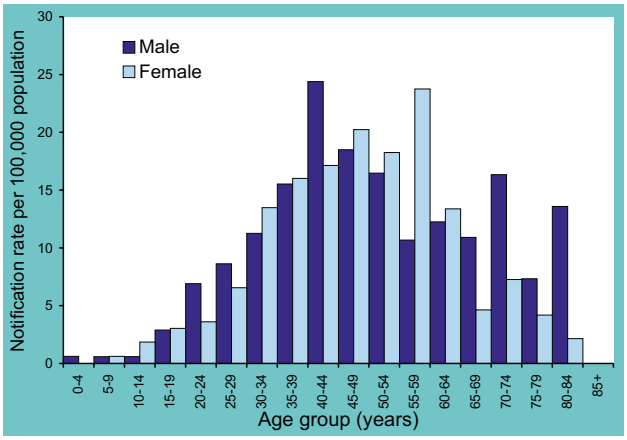
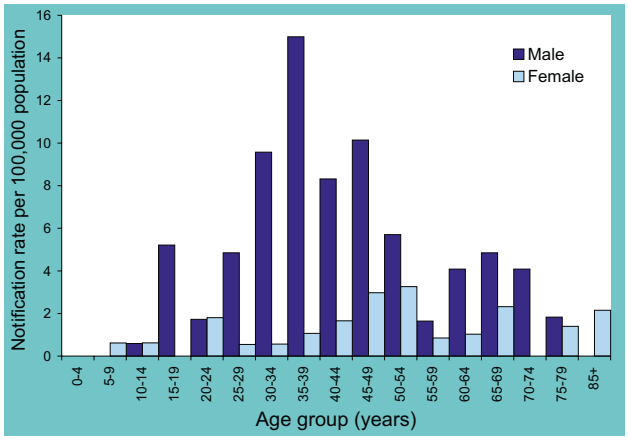


Figure 7. Notifications of Q fever, Australia, 1 April to 30 June 2001, by age and sex



Other diseases

Legionellosis

Notifications of legionellosis are well down on last year (137 YTD compared with 319 for the same period in 2000). The Melbourne Aquarium outbreak in the second quarter of 2000 had a significant impact on the burden of disease in Victoria. Victoria reported 31 cases in the second quarter of 2001, which represented more than 40 per cent of all reports of legionellosis in Australia in the same period. The Victorian Government has recently strengthened requirements for the maintenance of cooling towers to prevent contamination with the *Legionella* bacteria. Contaminated cooling towers have been implicated in outbreaks of legionellosis world-wide.

Meningococcal disease

The number of meningococcal disease notifications was slightly increased compared with the average of the last 5 years. The totals for the first half of this year (n=271) were above those reported in the first 6 months of 2000 (n=215).

LabVISE

The Laboratory Virology and Serology (LabVISE) reporting scheme is a passive surveillance scheme based on voluntary reports of infectious agents contributed to the Commonwealth Department of Health and Aged Care, by sentinel virology and serology laboratories around Australia.

Comments on second quarter 2001 LabVISE data

Reports to LabVISE were lower (9,304 reports) in this quarter, than for the same quarter last year (11,303 reports). Reports were received from all States and Territories through 13 participating laboratories (Table 4).

Data collected in the LabVISE surveillance scheme supplemented that collected in the NNDSS in the same quarter. Year to date totals of isolates of Barmah Forest virus are almost double in number (200) compared with the same period in 2000 (n=104). These were mostly from Queensland (n=95), where reports of BF infection to NNDSS were also increased compared with previous years. Similarly year to date totals reported to LabVISE of isolates of *Coxiella burnetti*, the causative organism of Q fever, are increased (n=72) compared with the same period (n=32) last year. These reports were largely from Queensland (n=20) or Victoria (n=24). Both these States also reported increased Q fever cases to the NNDSS.

All reports of Norwalk-like virus (NLV) in this quarter were notified from Victoria. This may reflect a reporting bias because the Victorian Infectious Diseases Research Laboratory, unlike other laboratories, routinely screens stool specimens for the Norwalk virus. These reports included samples from 4 recognised outbreaks of NLV in this quarter in Victoria. Two of these outbreaks were in childcare centres, one in a primary school and one in an aged care facility. The frequency and size of these outbreaks were similar to those previously seen in this season in Victoria (Joy Gregory, OzFoodNet, Department of Human Services, Victoria, personal communication). NLV is the leading cause of diarrhoea and vomiting in the United Kingdom[6] and may comprise up to 11 per cent of all episodes of acute primary gastroenteritis in the USA.[7]

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Tables

There were 20,278 notifications to the National Notifiable Diseases Surveillance System (NNDSS) with a notification date between 1 April and 30 June 2001 (Table 2). Figure 1 illustrates, for selected Diseases, the 2nd quarter 2001 totals as ratios to the mean of the 2nd quarters for the previous 5 years. A summary of Diseases currently being reported by each jurisdiction is provided in Table 1. The notification rate of Diseases per 100,000 population for each State or Territory is presented in Table 3.

There were 4,908 reports received by the *CDI* Virology and Serology Laboratory Reporting Scheme (LabVISE) in the reporting period, 1 April to 30 June 2001 (Tables 4 and 5).

The Australian Sentinel Practice Research Network (ASPREN) data for weeks 13-17 to 22-26, ending 1 July 2001, are included in this issue of *Communicable Diseases Intelligence* (Table 6).

Table 1. Reporting of notifiable diseases by jurisdiction

Disease	Data received from:*
Bloodborne	
Hepatitis B (incident)	All jurisdictions
Hepatitis B (unspecified)	All jurisdictions except NT
Hepatitis C (incident)	All jurisdictions
Hepatitis C (unspecified)	All jurisdictions
Hepatitis D	All jurisdictions
Gastrointestinal	
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	
Cholera	All jurisdictions
Plague	All jurisdictions
Rabies	All jurisdictions
Viral haemorrhagic fever	All jurisdictions
Yellow fever	All jurisdictions
Sexually transmissible	
Chlamydial infection	All jurisdictions
Donovanosis	All jurisdictions except SA
Gonococcal infection	All jurisdictions
Syphilis	All jurisdictions
Vaccine preventable	
Diphtheria	All jurisdictions
Haemophilus influenzae type b	All jurisdictions
Influenza	All jurisdictions except ACT, Qld, SA & Vic
Measles	All jurisdictions
Mumps	All jurisdictions
Pertussis	All jurisdictions
Pneumococcal disease	All jurisdictions except SA & Tas
Rubella	All jurisdictions
Tetanus	All jurisdictions
Vectorborne	
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 except SA
Australian Bat lyssavirus	All jurisdictions
Brucellosis	All jurisdictions
Leptospirosis	All jurisdictions
Ornithosis	All jurisdictions
Other lyssaviruses (NEC)	All jurisdictions
Q Fever	All jurisdictions
Other	
Legionellosis	All jurisdictions
Leprosy	All jurisdictions
Meningococcal infection	All jurisdictions
Tuberculosis	All jurisdictions

* Jurisdictions not yet reporting on Diseases either because legislation has not yet made some Diseases notifiable in that jurisdiction or data are not yet being reported to the Commonwealth

† Combined under Murray Valley encephalitis

Table 2. Notifications of diseases received by State and Territory health authorities in the period 1 April to 30 June 2001, by date of notification*

Disease	ACT	NSW	NT	Qld	SA	Tas	Vic	WA	Total 2nd quarter 2001[1]	Total 1st quarter 2001[1]	Total 2nd quarter 2000[1]	Last 5 years mean 2nd quarter	Year to date 2001	Last 5 years YTD mean	Ratio†
Bloodborne															
Hepatitis B (incident)	2	15	1	15	6	5	43	7	94	96	108	72	240	148	1.3
Hepatitis B (unspecified)[2]	14	534	NN	196	51	7	534	161	1,497	1,487	2,075	1,847	2,853	3,633	0.8
Hepatitis C (incident)	7	13	0	0	25	0	12	6	63	82	112	67	143	141	0.9
Hepatitis C (unspecified)[2]	37	1,252	36	728	136	88	1,236	376	3,889	4,309	5,007	4,445	7,918	9,107	0.9
Hepatitis D	0	4	0	1	0	0	1	0	6	5	6	3	11	7	1.9
Gastrointestinal									0						
Botulism	0	0	0	1	0	0	0	0	1	1	0	0.2	2	0	5.0
Campylobacterosis[3]	106	-	83	959	506	128	1,263	584	3,629	3,394	3,248	2,791	6,810	5,877	1.1
Cryptosporidiosis	2	35	91	80	20	NDR	108	49	385	255	NDR	n/a	599	n/a	n/a
Haemolytic uraemic syndrome	0	0	0	0	0	0	0	0	0	2	1	2	2	7	0.0
Hepatitis A	2	27	16	35	5	0	26	4	115	96	215	497	210	1,201	0.2
Hepatitis E	0	0	0	0	0	0	2	0	2	1	0	0.4	3	3	5.0
Listeriosis	0	3	0	3	2	0	0	0	8	21	17	14	29	34	0.6
Salmonellosis	10	299	85	538	126	45	249	198	1,550	2,180	1,564	1,599	3,655	4,065	1.0
Shigellosis	4	21	28	19	10	2	26	12	122	116	142	160	235	344	0.8
SLTEC,VTEC[4]	0	0	0	1	4	0	1	0	6	16	5	5	22	15	1.1
Typhoid	0	4	2	1	0	0	2	1	10	33	17	14	43	42	0.7
Quarantinable															
Cholera	0	0	0	0	0	0	0	0	0	0	0	1	0	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
Sexually transmissible															
Chlamydial infection	52	792	265	1,290	665	80	490	660	4,294	4,696	4,270	3,036	8,733	6,018	1.4
Donovanosis	0	0	4	1	NN	0	0	3	8	2	4	7	10	16	1.1
Gonococcal infection[5]	2	249	325	208	85	5	176	342	1392	1,457	1,708	1,412	2,737	2,747	1.0
Syphilis[6]	1	148	62	0	6	2	3	42	264	278	484	422	536	841	0.6

Table 2 (continued). Notifications of diseases received by State and Territory health authorities in the period 1 April to 30 May 2001, by date of notification*

Disease	ACT	NSW	NT	Qld	SA	Tas	Vic	WA	Total 2nd quarter 2001[1]	Total 1st quarter 2001[1]	Total 2nd quarter 2000[1]	Last 5 years mean 2nd quarter	Year to date 2001	Last 5 years YTD mean	Ratio[†]
Vaccine preventable															
Diphtheria	0	0	0	0	0	0	0	0	0	1	0	0	1		0.0
Haemophilus influenzae type b	0	5	3	3	2	0	0	1	14	5	6	12	19	21	1.2
Influenza*	NDR	9	2	NDR	NDR	0	NDR	13	24	12	NDR	n/a	24	n/a	n/a
Measles	0	9	0	1	0	1	0	1	12	70	32	80	82	174	0.2
Mumps	0	6	0	0	2	1	7	14	30	31	64	47	57	90	0.6
Pertussis	17	602	37	223	281	11	101	12	1,284	1,217	1,107	1,020	2,494	2,388	1.3
Pneumococcal disease*	8	84	29	97	NDR	NDR	50	58	326	87	NDR	n/a	391	n/a	n/a
Poliomyelitis	0	0	0	0	0	0	0	0	0	0	0	0	0		0.0
Rubella[7]	0	6	0	27	0	2	5	0	40	55	53	206	95	482	0.2
Tetanus	0	0	0	0	0	0	0	0	0	1	1	1	1	3	0.0
Vectorborne															
Arbovirus infection NEC	0	5	1	0	0	0	8	0	14	9	27	14	23	41	1.0
Barmah Forest virus infection	1	188	8	230	0	0	2	17	446	324	169	200	760	451	2.2
Dengue	1	9	10	19	3	0	3	5	50	33	40	37	83	140	1.3
Japanese encephalitis	0	0	0	0	0	0	0	0	0	0	NDR	n/a	0	n/a	n/a
Kunjin virus infection	NDR	0	0	0	0	0	0	0	0	0	NDR	n/a	0	n/a	n/a
Malaria	1	25	15	70	3	5	21	8	148	230	276	199	375	459	0.7
Murray Valley encephalitis	NDR	0	0	0	0	0	0	0	0	2	NDR	n/a	2	n/a	n/a
Ross River virus infection	4	323	19	659	13	1	55	56	1,130	1,577	1,408	1,608	2,681	4,482	0.7
Zoonoses															
Anthrax*	0	0	0	0	NN	0	0	0	0	0	NDR	n/a	0	n/a	n/a
Australian bat lyssavirus*	0	0	0	0	0	0	0	0	0	0	NDR	n/a	0	n/a	n/a
Brucellosis	0	0	0	2	0	0	0	0	2	6	2	5	8	13	0.4
Leptospirosis	0	17	1	34	0	1	6	1	60	70	75	65	130	127	0.9
Other lyssavirus (NEC)*	0	0	0	0	0	0	0	0	0	0	NDR	n/a	0	n/a	n/a
Ornithosis	0	6	0	NDR	2	0	14	1	23	29	23	19	52	34	1.2
Q fever	1	28	0	95	3	1	18	4	150	169	118	144	319	275	1.0

Table 2 (continued). Notifications of diseases received by State and Territory health authorities in the period 1 April to 30 June 2001, by date of notification*

Disease	ACT	NSW	NT	Qld	SA	Tas	Vic	WA	Total 2nd quarter 2001[1]	Total 1st quarter 2001[1]	Total 2nd quarter 2000[1]	Last 5 years mean 2nd quarter	Year to date 2001	Last 5 years YTD mean	Ratio†
Other															
Legionellosis	0	20	1	12	4	1	31	7	76	65	237	93	137	158	0.8
Leprosy	0	0	0	0	0	0	0	0	0	1	2	1	1	4	0.0
Meningococcal infection	0	59	5	23	7	2	36	11	143	128	131	112	271	183	1.3
Tuberculosis	1	37	0	5	0	1	67	14	125	163	214	236	287	508	0.5
Total	273	4,834	1,129	5,576	1,967	389	4,596	2,668	21,432	22,812	22,968	20,495	43,084	44,284	1.0

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. Unspecified numbers should be interpreted with some caution as the magnitude may be a reflection of the numbers of tests being carried out.
3. Not reported for NSW because it is only notifiable as 'foodborne disease' or 'gastroenteritis in an institution'.
4. Infections with Shiga-like toxin (verotoxin) producing *E. coli* (SLTEC/VTEC).
5. NT, Qld, SA , Vic and WA: includes gonococcal neonatal ophthalmia.

NA Not calculated as only notifiable for under 5 years.

6. Includes congenital syphilis.
7. Includes congenital rubella

* 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.

NDR No data received.

NN. Not Notifiable

NEC Not Elsewhere Classified.

\- Elsewhere Classified.

Table 3. Notification rates of diseases by State or Territory, 1 April to 30 June 2001. (Rate per 100,000 population)

Disease[1]	State or Territory								
	ACT	NSW	NT	Qld	SA	Tas	Vic	WA	Australia
Bloodborne									
Hepatitis B (incident)	2.6	0.9	2.0	1.7	1.6	4.3	3.6	1.5	1.9
Hepatitis B (unspecified)[2]	17.9	32.8	NN	21.8	13.6	6.0	44.5	33.9	31.3
Hepatitis C (incident)	9.0	0.8	0.0	0.0	6.7	0.0	1.0	1.3	1.3
Hepatitis C (unspecified)[2]	47.4	77.0	73.4	81.0	36.3	74.9	103.1	79.2	80.7
Hepatitis D	0.0	0.2	0.0	0.1	0.0	0.0	0.1	0.0	0.1
Gastrointestinal									
Botulism	0.0	0.0	0.0	0.1	0.0	0.0	0.0	0.0	0.0
Campylobacterosis[3]	135.7	_	169.1	106.6	134.9	108.9	105.3	123.1	113.6
Cryptosporidiosis	2.6	2.2	185.4	8.9	5.3	NDR	9.0	10.3	8.0
Haemolytic uraemic syndrome	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
Hepatitis A	2.6	1.7	32.6	3.9	1.3	0.0	2.2	0.8	2.4
Hepatitis E	0.0	0.0	0.0	0.0	0.0	0.0	0.2	0.0	0.0
Listeriosis	0.0	0.2	0.0	0.3	0.5	0.0	0.0	0.0	0.2
Salmonellosis	12.8	18.4	173.2	59.8	33.6	38.3	20.8	41.7	32.2
Shigellosis	5.1	1.3	57.1	2.1	2.7	1.7	2.2	2.5	2.5
SLTEC,VTEC[4]	0.0	0.0	0.0	0.1	1.1	0.0	0.1	0.0	0.1
Typhoid	0.0	0.2	4.1	0.1	0.0	0.0	0.2	0.2	0.2
Quarantinable									
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									
Chlamydial infection	66.6	48.7	540.0	143.4	177.3	68.1	40.9	139.1	89.1
Donovanosis	0.0	0.0	8.2	0.1	NN	0.0	0.0	0.6	0.2
Gonococcal infection[5]	2.6	15.3	662.3	23.1	22.7	4.3	14.7	72.1	28.9
Syphilis[6]	1.3	9.1	126.3	0.0	1.6	1.7	0.3	8.8	5.5
Vaccine preventable									
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	0.3	6.1	0.3	0.5	0.0	0.0	0.2	0.3
Influenza*	NDR	0.6	4.1	NDR	NDR	0.0	NDR	2.7	1.1
Measles	0.0	0.6	0.0	0.1	0.0	0.9	0.0	0.2	0.2
Mumps	0.0	0.4	0.0	0.0	0.5	0.9	0.6	2.9	0.6
Pertussis	21.8	37.0	75.4	24.8	74.9	9.4	8.4	2.5	26.6
Pneumococcal disease	10.2	5.2	59.1	10.8	NDR	NDR	4.2	12.2	7.5
Poliomyelitis	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
Rubella[7]	0.0	0.4	0.0	3.0	0.0	1.7	0.4	0.0	0.8
Tetanus	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
Vectorborne									
Arbovirus infection NEC	0.0	0.3	2.0	0.0	0.0	0.0	0.7	0.0	0.3
Barmah Forest virus infection	1.3	11.6	16.3	25.6	0.0	0.0	0.2	3.5	9.3
Dengue	1.3	0.6	20.4	2.1	0.8	0.0	0.3	1.1	1.0
Japanese encephalitis	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
Kunjin virus infection	NDR	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
Malaria	1.3	1.5	30.6	7.8	0.8	4.3	1.8	1.7	3.1
Murray Valley encephalitis	NDR	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
Ross River virus infection	5.1	19.9	38.7	73.3	3.5	0.9	4.6	11.8	23.4

Table 3 (continued). Notification rates of diseases by State or Territory, 1 April to 30 June 2001. (Rate per 100,000 population)

Disease[1]	State or Territory								
	ACT	NSW	NT	Qld	SA	Tas	Vic	WA	Australia
Zoonoses									
Anthrax*	0.0	0.0	0.0	0.0	NN	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	0.2	0.0	0.0	0.0	0.0	0.0
Leptospirosis	0.0	1.0	2.0	3.8	0.0	0.9	0.5	0.2	1.2
Other lyssavirus (NEC)	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
Ornithosis	0.0	0.4	0.0	NDR	0.5	0.0	1.2	0.2	0.6
Q fever	1.3	1.7	0.0	10.6	0.8	0.9	1.5	0.8	3.1
Other									
Legionellosis	0.0	1.2	2.0	1.3	1.1	0.9	2.6	1.5	1.6
Leprosy	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
Meningococcal infection	0.0	3.6	10.2	2.6	1.9	1.7	3.0	2.3	3.0
Tuberculosis	1.3	2.3	0.0	0.6	0.0	0.9	5.6	2.9	2.6

1. Rates are subject to retrospective revision.
2. Unspecified numbers should be interpreted with some caution as the magnitude may be a reflection of the numbers of tests being carried out.
3. Not reported for NSW because it is only notifiable as 'foodborne disease' or 'gastroenteritis in an institution'.
4. Infections with Shiga-like toxin (verotoxin) producing *E. coli* (SLTEC/VTEC).
5. NT, Qld, SA , Vic and WA: includes gonococcal neonatal ophthalmia.
6. Includes congenital syphilis.
7. Includes congenital rubella.

NDR No data received.
NN Not Notifiable
NEC Not Elsewhere Classified.
- Elsewhere Classified.

Table 4. Virology and serology laboratory reports by laboratories for the reporting period 1 April to 30 June 2001[1]

State or Territory	Laboratory	April 2001	May 2001	June 2001	Total this period
Australian Capital Territory	The Canberra Hospital	-	-	-	-
New South Wales	Institute of Clinical Pathology & Medical Research, Westmead	127	42	108	277
	New Children's Hospital, Westmead	73	108	136	317
New South Wales	Repatriation General Hospital, Concord	-	-	-	0
	Royal Prince Alfred Hospital, Camperdown	25	44	53	122
	South West Area Pathology Service, Liverpool	105	130	209	444
Queensland	Queensland Medical Laboratory, West End	556	665	436	1,657
	Townsville General Hospital	1	17	-	18
South Australia	Institute of Medical and Veterinary Science, Adelaide	427	513	-	940
Tasmania	Northern Tasmanian Pathology Service, Launceston	2	19	10	31
	Royal Hobart Hospital, Hobart	-	-	-	0
Victoria	Monash Medical Centre, Melbourne	19	49	22	90
	Royal Children's Hospital, Melbourne	64	91	98	253
	Victorian Infectious Diseases Reference Laboratory, Fairfield	117	122	99	338
Western Australia	PathCentre Virology, Perth	-	-	-	-[2]
	Princess Margaret Hospital, Perth	52	103	182	337
	Western Diagnostic Pathology	30	38	16	84
Total		1,598	1,941	1,369	4,908

1. The complete list of laboratories reporting for the 12 months, January to December 2001, will appear in every report from January 2000 regardless of whether reports were received in this reporting period. Reports are not always received from all laboratories.
2. Data received from PathCentre Virology, Perth from October 2000 to August 2001 is awaiting processing by the Department of Health and Aged Care. A special report on these data will appear in a later edition of *Communicable Diseases Intelligence* (*CDI*). The CDI Editorial staff apologise for any inconvenience this may cause.

- Nil reports

Table 5. Virology and serology laboratory reports by State or Territory[1] for the reporting period 1 April to 30 June 2001, and total reports for the year[2]

	State or Territory[1]								This period 2001	This period 2000	Year to date 2001[3]	Year to date 2000
	ACT	NSW	NT	Qld	SA	Tas	Vic	WA				
Measles, mumps, rubella												
Measles virus	-	2	-	2	-	1	1	-	6	14	91	26
Mumps virus	-	-	-	1	-	-	2	-	3	8	11	31
Rubella virus	-	1	-	10	-	1	-	-	12	10	25	23
Hepatitis viruses												
Hepatitis A virus	-	1	4	15	3	-	3	1	27	41	39	94
Hepatitis D virus	-	-	-	-	1	-	1	-	2	2	3	3
Hepatitis E virus	-	-	-	-	-	-	1	-	1	1	3	2
Arboviruses												
Ross River virus	-	11	11	260	17	-	8	3	310	492	659	1,065
Barmah Forest virus	-	2	1	95	1	-	-	-	99	41	200	104
Dengue not typed	-	-	-	-	-	-	1	-	1	30	1	164
Flavivirus (unspecified)	-	-	1	3	-	-	8	-	12	6	15	37
Adenoviruses												
Adenovirus type 1	-	-	-	-	1	-	-	-	1	2	1	4
Adenovirus type 2	-	-	-	-	-	-	1	-	1	3	2	6
Adenovirus type 3	-	-	-	-	-	-	1	-	1	3	3	12
Adenovirus type 4	-	-	-	-	-	-	1	-	1	-	2	4
Adenovirus type 7	-	-	-	-	-	-	3	-	3	2	9	4
Adenovirus type 8	-	-	-	-	-	-	4	-	4	-	8	-
Adenovirus not typed/pending	2	37	-	-	87	-	45	7	178	263	368	546
Herpes viruses												
Cytomegalovirus	-	60	-	38	92	2	48	6	246	303	596	621
Varicella-zoster virus	3	32	9	100	28	-	55	6	233	325	602	749
Epstein-Barr virus	-	22	15	161	140	-	13	60	411	664	802	1,207
Other DNA viruses												
Parvovirus	-	1	-	26	17	-	17	-	61	73	103	167
Picornavirus family												
Coxsackievirus A16	-	-	-	-	-	-	1	-	1	1	2	3
Echovirus type 9	-	14	-	-	-	1	-	-	15	-	49	3
Echovirus type 11	-	-	-	-	-	-	1	-	1	2	4	6
Echovirus type 13	-	2	-	-	-	-	-	-	2	-	8	-
Echovirus type 18	-	1	-	-	-	-	-	-	1	-	4	-
Echovirus type 30	1	1	-	-	1	-	-	-	3	32	23	107
Echovirus not typed/pending	-	-	-	-	-	-	3	-	3	3	4	4
Poliovirus type 1 (unchar)	-	3	-	-	-	-	-	-	3	1	9	4
Poliovirus type 2 (unchar)	-	3	-	-	-	-	-	-	3	1	7	3
Poliovirus type 3 (unchar)	-	1	-	-	-	-	-	-	1	2	2	3
Poliovirus - mixed strain (unchar)	-	-	-	-	-	-	1	-	1	-	1	-
Rhinovirus (all types)	1	62	-	1	-	-	1	-	65	116	141	212
Enterovirus type 71 (BCR)	-	2	-	-	-	-	-	-	2	-	20	-
Enterovirus not typed/pending	-	10	-	2	-	1	15	-	28	282	93	583

Table 5 (continued). Virology and serology laboratory reports by State or Territory[1] for the reporting period 1 April to 30 June 2001, and total reports for the year[2]

	State or Territory[1]								This period 2001	This period 2000	Year to date 2001[3]	Year to date 2000
	ACT	NSW	NT	Qld	SA	Tas	Vic	WA				
Ortho/paramyxoviruses												
Influenza A virus	1	19	-	1	21	-	1	6	49	161	144	357
Influenza B virus	-	2	-	-	7	-	6	1	16	70	49	97
Parainfluenza virus type 1	-	1	-	-	5	-	2	1	9	130	15	180
Parainfluenza virus type 2	-	3	-	2	5	-	3	5	18	15	24	21
Parainfluenza virus type 3	-	17	-	4	25	-	8	28	82	39	165	110
Respiratory syncytial virus	2	599	-	67	50	10	128	98	954	1,032	1,112	1,285
Other RNA viruses												
Rotavirus	-	77	1	-	50	5	74	173	380	234	527	365
Astrovirus	-	-	-	-	-	-	1	-	1	-	1	-
Reovirus (unspecified)	-	-	-	-	-	-	1	-	1	-	1	1
Norwalk agent	-	-	-	-	-	-	34	-	34	3	115	4
Other												
Chlamydia trachomatis not typed	13	119	26	293	155	12	2	5	625	869	1,347	1,748
Chlamydia psittaci	-	2	-	-	-	-	18	-	20	25	41	50
Chlamydia species	-	1	-	-	-	-	-	-	1	3	4	6
Mycoplasma pneumoniae	-	30	4	83	26	2	31	1	177	150	356	299
Coxiella burnetii (Q fever)	2	2	-	20	-	-	24	-	48	8	72	32
Rickettsia - Spotted fever group	-	-	-	-	-	1	1	-	2	-	2	1
Streptococcus group A	-	13	6	58	-	-	17	-	94	72	192	181
Brucella species	-	-	-	2	-	-	-	-	2	1	2	4
Bordetella pertussis	-	35	3	58	93	-	48	-	237	110	451	275
Legionella pneumophila	-	1	-	-	-	-	22	-	23	12	28	15
Legionella longbeachae	-	-	-	-	-	-	1	-	1	19	1	35
Legionella species	-	-	-	-	-	-	7	-	7	1	7	1
Cryptococcus species	-	4	-	3	7	-	-	-	14	7	25	8
Leptospira species	-	-	-	8	3	-	-	-	11	24	26	33
Treponema pallidum	-	35	73	130	102	-	-	-	340	237	654	367
Entamoeba histolytica	-	-	-	1	-	-	4	-	5	1	7	9
Toxoplasma gondii	-	1	-	-	-	-	7	-	8	4	16	8
Echinococcus granulosus	-	-	-	-	6	-	1	-	7	11	10	14
Total	25	1,229	154	1,444	943	36	676	401	4,908	5,961	9,304	11,303

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 6. Australian Sentinel Practice Research Network reports, weeks 13-17 to 22-26, 2001

Week number	13-17		18-21		22-26	
Ending on	29 April 2001		27 May 2001		01 July 2001	
Doctors reporting	288		259		317	
Total encounters	31,597		29,688		36,934	
Condition	Reports	Rate per 1,000 encounters	Reports	Rate per 1,000 encounters	Reports	Rate per 1,000 encounters
Influenza	137	4.3	181	6.1	333	9.1
Influenza with culture	5	0.2	9	0.3	5	0.1
Chickenpox	60	1.9	43	1.4	104	2.8
Shingles	50	1.6	38	1.3	61	1.7

The NNDSS is conducted under the auspices of the Communicable Diseases Network Australia. The system coordinates the national surveillance of more than 50 communicable diseases or disease groups endorsed by the Communicable Diseases Network Australia and the National Public Health Partnership. Notifications of these diseases are made to State and Territory health authorities under the provisions of their respective public health legislations. De-identified core unit data are supplied fortnightly for collation, analysis and dissemination. For further information, see Commun Dis Intell *2000;24:6-7.*

LabVISE is a sentinel reporting scheme. Currently 17 laboratories contribute data on the laboratory identification of viruses and other organisms. This number may change throughout the year. Data are collated and published in Communicable Diseases Intelligence *monthly. These data should be interpreted with caution as the number and type of reports received is subject to a number of biases. For further information, see* Commun Dis Intell *2000;24:10.*

ASPREN currently comprises about 120 general practitioners from throughout the country, not all of whom report each week. Between 7,000 and 8,000 consultations are reported each week, with special attention to 12 conditions chosen for sentinel surveillance in 2001. Communicable Diseases Intelligence *reports the consultation rates for four of these. For further information, including case definitions, see* Commun Dis Intell *2001;25:106.*

Additional Reports

Australian encephalitis: Sentinel Chicken Surveillance Programme

Sentinel chicken flocks are used to monitor flavivirus activity in Australia. The main viruses of concern are Murray Valley encephalitis (MVE) and Kunjin which cause the potentially fatal disease encephalitis, in humans. Currently 30 flocks are maintained in the north of Western Australia, 9 in the Northern Territory, 12 in New South Wales and 10 in Victoria. The flocks in Western Australia and the Northern Territory are tested year round but those in New South Wales and Victoria are tested only from November to March, during the main risk season.

Results are coordinated by the Arbovirus Laboratory in Perth and reported bimonthly. For more information and details of the location of sentinel chicken sites see Commun Dis Intell *2000;24:8-9.*

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5. Berrimah Agricultural Research Centre, Northern Territory
6. PathCentre, Western Australia
7. Territory Health Services, Northern Territory

May/June 2001

Sentinel chicken serology was carried out for 26 of the 30 flocks in Western Australia in May and June 2001. The number of seroconversions to flaviviruses have decreased in the north of Western Australia but Murray Valley encephalitis (MVE) and Kunjin virus (KUN) activity was still detected in both the Kimberley and Pilbara regions. In May there were 10 seroconversions from the Kimberley and 22 from the Pilbara. The majority of these were to MVE virus. Flavivirus activity decreased significantly in June and there was only one seroconversion to MVE from the Aboriginal community of Kalumburu in the far north Kimberley and 4 seroconversions (2 KUN, 2 MVE/KUN) from Marble Bar, Paraburdoo and Ophthalmia Dam (near Newman) in the Pilbara. The number of chickens positive for flavivirus antibodies by ELISA at each site and the identity of the infecting virus(es) are shown in Table 7. There have been no cases of disease caused by MVE virus reported from Western Australia during the 2001 wet season.

Serum samples from 7 of the 8 Northern Territory sentinel chicken flocks were tested at the University of Western Australia in May and June 2001. There were 4 new seroconversions to flaviviruses in May (3 KUN, 1 Flavi only) and one to MVE virus from the Alice Springs flock in June 2001. In May, Kunjin virus seroconversions were reported from Howard Springs, Beatrice Hill Farm and the new flock at Gapuwiyak. The single seroconversion to a flavivirus (not MVE or Kunjin) was from Leanyer. A media warning was sent out by the Territory Health Services in May warning of continuing flavivirus activity, particularly in the Top End of the Northern Territory.

Flavivirus activity was not detected in New South Wales or Victoria in May 2001 and the sentinel chicken surveillance programs in these States have now finished for the season.

The State health departments provide funding for the sentinel chicken surveillance programs in Western Australia, the Northern Territory, New South Wales and Victoria.

Table 7. Flavivirus seroconversions in Western Australian sentinel chicken flocks, May and June 2001

	May 2001				June 2001		
Location	MVE	KUN	MVE/KUN	FLAVI	MVE	KUN	MVE/KUN
Kimberley							
Kalumburu			1	1	1#		
Kununurra	2			1			
Derby*	3			1			
Broome*		1					
Pilbara							
Port/South Hedland*	1						
Harding Dam*	2	1					
Marble Bar	2						1#
Tom Price	4	1	1				
Paraburdoo	2	1	1			1#	
Ophthalmia Dam	3	1				1	1#
Newman town		1					
Onslow	1#						

MVE antibodies to Murray Valley encephalitis virus detected by ELISA
KUN antibodies to Kunjin virus detected by ELISA
FLAVI antibodies to a flavivirus only detected by ELISA
Some results not yet confirmed
* Two flocks at this town

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.

Reporting period 1 January to 31 March 2001

The AGSP laboratories examined a total of 938 isolates in this quarter, virtually the same number as in the past two years. About 36 per cent of this total was from New South Wales, 19 per cent from Victoria, 18 per cent from Queensland, 12 per cent from the Northern Territory, 8 per cent from Western Australia and 6 per cent from South Australia. Isolates from other centres were few.

Penicillins

Figure 1 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 else penicillinase producing (PPNG) aggregated for Australia and by State and Territory. A high proportion those strains classified as PPNG or else resistant by chromosomal mechanisms fail to respond to treatment with penicillins (penicillin, amoxycillin, ampicillin) and early generation cephalosporins.

In this quarter about 23 per cent of all isolates were penicillin resistant by one or more mechanisms, 9 per cent PPNG and 14 per cent by chromosomal mechanisms (CMRNG). The proportion of penicillin resistant strains ranged from 7 per cent in the Northern Territory to 34 per cent in New South Wales.

The number of PPNG isolated across Australia (85) was slightly less in this quarter than in the corresponding period in 2000 (91). The highest proportion of PPNG was found in isolates from Victoria (16%) and Western Australia (11%). PPNG were present in all jurisdictions including 5 (4.5%) in the Northern Territory. South East Asian countries were the main source of external acquisition, but local acquisition was prominent in New South Wales.

More isolates were resistant to the penicillins by separate chromosomal mechanisms (132). These CMRNG were especially prominent in New South Wales (27%) and Queensland (11%). Three CMRNG were detected in the Northern Territory.

Ceftriaxone

Low numbers of isolates with decreased susceptibility to ceftriaxone (MICs 0.06/0.12 mg/L) were present in New South Wales, Victoria, Queensland and South Australia.

Spectinomycin

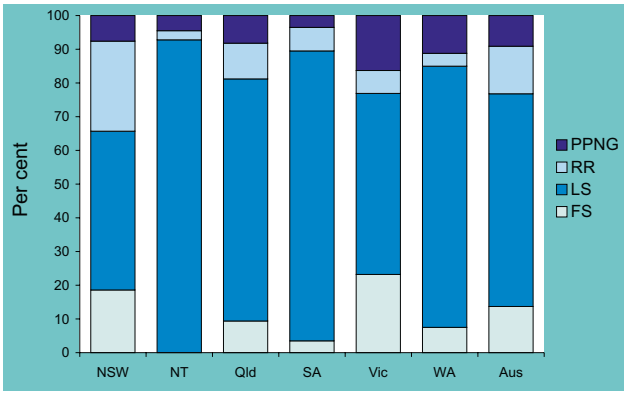
All isolates susceptible to this injectable agent.

Quinolone antibiotics

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

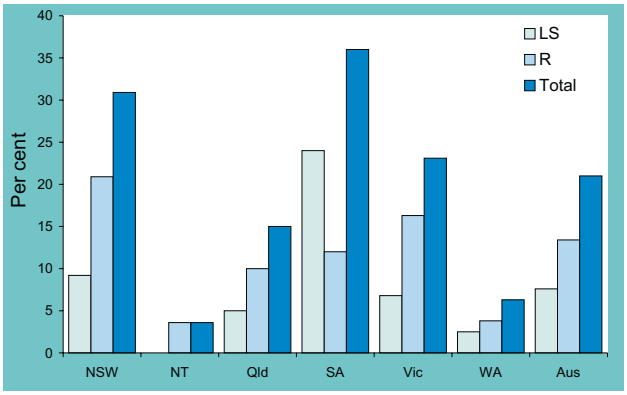
The total number (197) and proportion (21%) of all QRNG was again high and little changed from the first quarter of 2000 (183 isolates, 20%). QRNG were again widely distributed. High rates were maintained in South Australia (36%), New South Wales (31%), Victoria (22%) and

Figure 1. Categorisation of gonococci isolates, Australia, 1 January to 31 March 2001, 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*

Figure 2. Distribution of *N. gonorrhoeae* showing quinolone resistance, Australia, 1 January to 31 March 2001



LS QRNG Ciprofloxacin MICs 0.06 – 0.5 mg/L
R QRNG Ciprofloxacin MICs 1 mg/L

Queensland (15%). Six per cent of Western Australian isolates were QRNG. Seventy-two of the New South Wales, 23 of the Victorian and 17 of the Queensland QRNG exhibited high level resistance (MIC ciprofloxacin 1 mg/L) and higher level QRNG were also seen in the Northern Territory, South Australia and Western Australia. Local acquisition became increasingly prominent and MICs ranged up to 16mg/L. The majority of QRNG (126 of 197, 64%) are now in the high level category and this is a shift from the situation at this time last year.

High level tetracycline resistance (TRNG)

The number (73) and proportion (7.8%) of TRNG detected declined. TRNG represented 14 per cent of isolates from Queensland and Victoria, 7 per cent from South and Western Australia and 6 per cent from the Northern Territory.

Reference

1. Anon. 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 and related Diseases 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: (02) 9332 4648. Facsimile: (02) 9332 1837.

HIV and AIDS diagnoses and deaths following AIDS reported for 1 January to 31 march 2001, as reported to 30 June 2001, are included in this issue of Communicable Diseases Intelligence *(Tables 8 and 9).*

Table 8. New diagnoses of HIV infection, new diagnoses of AIDS and deaths following AIDS occurring in the period 1 January to 31 march 2001, by sex and State or Territory of diagnosis

										Totals for Australia			
		ACT	NSW	NT	Qld	SA	Tas	Vic	WA	This period 2001	This period 2000	Year to date 2001	Year to date 2000
HIV diagnoses	Female	0	6	0	7	1	0	5	0	19	21	19	21
	Male	0	26	1	21	8	2	22	1	81	198	81	198
	Sex not reported	0	0	0	0	0	0	0	0	0	0	0	0
	Total[1]	0	32	1	28	9	2	28	1	101	220	101	220
AIDS diagnoses	Female	0	1	0	0	0	0	1	0	2	7	2	7
	Male	0	5	1	9	0	0	9	0	24	69	24	69
	Total[1]	0	6	1	9	0	0	11	0	27	76	27	76
AIDS deaths	Female	0	1	0	0	0	0	1	0	2	3	2	3
	Male	0	3	0	3	0	0	5	0	11	29	11	29
	Total[1]	0	4	0	3	0	0	6	0	13	32	13	32

1. Persons whose sex was reported as transgender are included in the totals.

Table 9. Cumulative diagnoses of HIV infection, AIDS and deaths following AIDS since the introduction of HIV antibody testing to 31 March 2001, by sex and State or Territory

		State or Territory								
		ACT	NSW	NT	Qld	SA	Tas	Vic	WA	Australia
HIV diagnoses	Female	27	640	10	170	64	5	233	123	1272
	Male	229	11261	109	2092	702	80	4050	946	19,469
	Sex not reported	0	242	0	0	0	0	24	0	266
	Total[1]	256	12164	119	2269	766	85	4322	1075	21,056
AIDS diagnoses	Female	9	202	0	50	25	3	73	26	388
	Male	87	4750	37	865	351	45	1699	359	8193
	Total[1]	96	4964	37	917	376	48	1781	387	8606
AIDS deaths	Female	4	115	0	33	16	2	51	17	238
	Male	68	3254	24	582	234	29	1302	255	5748
	Total[1]	72	3377	24	617	250	31	1360	273	6004

1. Persons whose sex was reported as transgender are included in the totals.

In case you missed it

Hand, foot & mouth disease in the Asia- Pacific region: role of enterovirus 71

Source: Journal of Virology, August 2001

Enterovirus 71 (EV71) is a frequent cause of hand, foot, and mouth disease (HFMD) epidemics associated with severe neurological sequelae in a small proportion of cases. There has been a significant increase in EV71 epidemic activity throughout the Asia-Pacific region since 1997. Recent HFMD epidemics in this region have been associated with a severe form of brainstem encephalitis associated with pulmonary edema and high case fatality rates. In this study, we show that 4 genetic lineages of EV71 have been prevalent in the Asia-Pacific region since 1997, including 2 previously undescribed genogroups (B3 and B4). Furthermore, we show that viruses belonging to genogroups B3 and B4 have circulated endemically in Southeast Asia during this period and have been the primary cause of several large HFMD or encephalitis epidemics in Malaysia, Singapore, and Western Australia.

VAERS dataset available online

Information related to adverse events following immunisation with US licensed vaccines is now available on the Vaccine Adverse Event Reporting System's (VAERS) Website, at http://www.vaers.org. The data represent voluntary surveillance information reported by health professionals, vaccine manufacturers, and the public. VAERS accepts all reports of adverse events following vaccination, whether they are coincidental or actually caused by the vaccine. VAERS is a cooperative program jointly operated by the CDC and the Food and Drug Administration (FDA).

Bulletin Board

Public Health Association of Australia (PHAA)
PHAA Annual Conference
23–26 September 2001 Hilton Hotel Sydney
Contact: Annette Mellick
Telephone: +61 2 6285 2373
Facsimile: +61 2 6282 5438
E-mail: conference@phaa.net.au
Website: http://www.phaa.net.au

The Emerging Infectious Diseases of the Indian Ocean Rim EIDIOR
Workshop
26–29 September 2001
Perth, Western Australia
Contact: Secretariat
Telephone: +61 8 9322 6906,
Facsimile: +61 8 9322 1734
E-mail: conwes@congresswest.com.au

Australasian Epidemiological Association (AEA)
10th Annual Scientific Meeting of the Australasian Epidemiological Association
27-28 September 2001
University of Sydney
Contact: International Conferences & Events (ICE) Aust. Pty Ltd

The Australian Society for Microbiology
Annual Scientific Meeting
30 September – 6 October 2001
Burswood Resort and Convention Centre
Burswood, Perth
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Annual Conference
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The Australasian Society for HIV Medicine
13th Annual Conference
11-14 October 2001
Melbourne Convention Centre, Victoria
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The NSW Infection Control Association
24th Annual Conference
1-2 November 2001
Star City Casino, Sydney, New South Wales
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Association for Professionals in Infection Control and Epidemiology
29th Annual Conference
16-23 May 2002
Nashville Convention Center
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Contributions to the Bulletin Board are invited from those organisations with forthcoming events relevant to communicable disease control.

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.

Yellow fever in Liberia

WHO has reported 3 suspected cases of yellow fever in the south-eastern part of the country. One case with disease onset on 1 August 2001 has been confirmed (IgM positive) by the Institut Pasteur in Abidjan, Côte d'Ivoire. All 3 cases have died. The Ministry of Health's Epidemic Management Committee is planning a vaccination campaign in the affected county, but additional vaccine stocks will be needed to implement the emergency immunisation program.

Cholera

Chad

As of 21 August 2001, WHO has reported a total of 2458 cases of cholera in Chad, including 88 deaths, with a case-fatality rate of 3.5 per cent in the south-western part of the country. The Ministry of Health, with the assistance of WHO and Médecins sans Frontières, is continuing its surveillance and health education activities.

India

Since 7 July 2001 the Government of Orissa has reported 34,111 cases of diarrhoea including 33 deaths, in 24 districts in Orissa State. The cases related to the floods that occurred at that time and were detected through its early warning surveillance system. Orissa has a population of 37 million people, of which 8 million were affected by the floods. Among the cases of severe, acute diarrhoea in a cluster of 121 samples (taken from 5 districts) positive for *Vibrio cholerae,* 46 per cent were positive for serogroup O139. This proportion of O139 is high compared to the rates found in neighbouring Bangladesh, where there were 24 per cent positive isolates for O139 in non-coastal areas and 7.2 per cent in coastal areas in 2000. WHO is assisting the national health authorities in continuing surveillance.

Afghanistan

As of 25 July 2001, WHO has reported a total of 4499 cases of cholera, including 114 deaths. WHO and Médecins sans Frontières are assisting the Ministry of Public Health of Afghanistan to co-ordinate the response to the outbreak.

United Republic of Tanzania

WHO has reported 109 cases of cholera with 3 deaths between 18 May and 20 July 2001, in Dar es Salaam. The Tanzanian Ministry of Health is implementing control measures including chlorination of all water sources, provision of medical supplies to all cholera treatment centres and health education measures.

Meningococcal disease in Angola

Since the third week of May 2001, 77 cases and 17 deaths (a case-fatality rate of 22%) have been reported to WHO in the Balombo district of Angola. *Neisseria meningitidis* serogroup A has been laboratory confirmed. The cumulative attack rate since the beginning of the year is 212 per 100,000 population. Data from other districts are unavailable at the present time. On 13 August 2001, a mass vaccination campaign was launched targeting the population of Balombo district over 2 years of age.

Meningococcal disease, serogroup W135 - update 2

During 2001 the following countries have reported cases of W135 meningococcal disease to WHO.

Most cases are associated with international travel or contact with travellers to Saudi Arabia.

Burkina Faso

Following a joint mission of the Institut Pasteur, Paris and Association pour la Medecine Preventive (AMP) to investigate the epidemic meningitis situation, 10 additional cases of *N. meningitidis* W135 were laboratory confirmed. The samples were taken from documented cases between 10 and 24 April 2001 and the proportion of *N. meningitidis* isolates belonging to the W135 serogroup was found to be 37 per cent of the total *N. meningitidis* isolates identified by PCR on collected specimens. None of the cases had any relationship with the 2001 Haj pilgrimage (travel or contact history). Among 4 strains that were cultured from the same period, 3 were W135:2a:P1-2,5 and belong to the ET-37 complex.

Niger

The joint Institut Pasteur AMP mission to investigate the epidemic meningitis situation identified 10 laboratory confirmed cases of *N. meningitidis* W135. The samples were taken from documented cases between 10 and 16 April 2001 and the proportion of *N. meningitidis* isolates belonging to the W135 serogroup was found to be 40 per cent of the total *N. meningitidis* isolates identified by PCR on collected specimens. None of the cases had any relationship with the 2001 Haj pilgrimage (travel or contact history).

Central African Republic

Three reported cases of meningococcal disease in Haj pilgrims have been laboratory confirmed as *N. meningitidis* serogroup W135.

Denmark

Two cases (one case close contact with Haj pilgrims, the travel/contact history of the second case is not yet known) have been reported. *N. meningitidis* serogroup W135 has been laboratory confirmed.

France

Two cases (close contacts with Haj pilgrims) have been reported. *N. meningitidis* serogroup W135 has been laboratory confirmed.

Norway

Four cases (2 contacts with Haj pilgrims) have been reported. *N. meningitidis* serogroup W135 has been laboratory confirmed.

Saudi Arabia

Between 9 February and 22 March 2001 109 cases, predominantly Haj pilgrims from outside Saudi Arabia, including 35 deaths have been reported. *N. meningitidis* serogroup W135 has been laboratory confirmed in more than half of the cases.

Singapore

Four cases (3 close contacts with Haj pilgrims, one with history of travel to Saudi Arabia), including one death have been reported. Two of the cases occurred in January 2001, before the main period of pilgrimage to Saudi Arabia. *N. meningitidis* serogroup W135 has been laboratory confirmed.

United Kingdom

Forty-one cases (8 pilgrims returning from the Haj, 19 cases in close contacts and data outstanding on the remaining cases) including 11 deaths of laboratory confirmed invasive *N. meningitidis* serogroup W135 have been reported. WHO recommends that chemoprophylaxis be given to close contacts of the cases, such as persons sleeping in the same dwelling. In most countries rifampicin is recommended.

In preparation for the Umrah and the Haj seasons for next year, the Ministry of Health of the Government of Saudi Arabia has notified the Ministries of Health of all countries from which pilgrims arrive, that the vaccination against meningococcal meningitis with the quadrivalent vaccine (serogroups A, C, Y & W135) has been added to the health requirements for arrivals coming to the Umrah and Haj. WHO encourages national reference laboratories to closely monitor meningococcal disease.

For further information, see the Control of Epidemic Meningococcal Disease. WHO Practical Guidelines. <http://www.who.int/emc-documents/meningitis/docs/whoemcbac983.pdf>.

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.

Variant CJD cases in UK

Source: Reuters Health Online, 8 August 2001 (edited)

London: On 6 August 2001, the Department of Health reported that the number of 'definite and probable' cases of variant Creutzfeldt-Jakob disease (vCJD), thought to be the human form of bovine spongiform encephalopathy (BSE) or 'mad cow' disease, had risen to 106 in the United Kingdom (UK).

A monthly update on the disease from the National CJD Surveillance Unit showed that as of 3 August 2001, 14 people have died and been confirmed to have had vCJD this year. One other person thought to have the disease has died, but the brain biopsy needed to confirm vCJD has not yet been performed. Another 7 people have symptoms of the fatal condition, which classifies them as 'probable' cases. That brings the overall number of definite and probable cases of vCJD recorded by the surveillance unit since 1995, to 106.

Earlier this year, a member of the Spongiform Encephalopathy Advisory Committee, which advises the government on BSE, told BBC radio that the average incubation period for the disease in humans could 'well be in the region of 30 years. He said thousands or tens of thousands more cases could emerge. The director of the UK's National CJD Surveillance Unit, which monitors the epidemic, told Reuters Health that the latest figures are in line with current predictions that in the short term, the number of vCJD cases in the UK will double every 3 years. As to what will happen in the longer term, he said, 'My own personal view is that we can't say at the moment. We don't know for how long these trends will be maintained.'

CJD diagnostic test 'Ready in a year'

Researchers in Israel claim to have developed a simple diagnostic test for variant Creutzfeldt-Jakob disease (vCJD), the human form of bovine spongiform encephalopathy (BSE). They say their method can reliably distinguish between urine samples from healthy humans and samples preserved from people who died of vCJD. Ruth Gabizon, who leads the team at the Hadassah Medical Organisation in Israel, said that the procedure was developed using urine from humans, hamsters, and cattle. British specialists said the work is interesting, but raised questions about its accuracy.

vCJD, which is a fatal degenerative disease affecting the brain, is hard to distinguish from other degenerative Diseases like Alzheimer's disease. Doctors often cannot be sure that a person has vCJD until after death. So a urine test would make diagnosis much simpler and might even be used to work out how far and how quickly the disease has spread through the general population.

vCJD in humans, BSE in cows, and scrapie in hamsters are believed to be related forms of the same disease, known as transmissible spongiform encephalopathies. 'We had urine samples from all the cases of CJD in Israel in the last year,' she told BBC News Online. The team compared these samples with urine from non-infected people, and carried out the same procedure on urine from British BSE-infected cattle and scrapie-infected hamsters. 'We were able to detect all the positives as positives and all the negatives as negatives,' she said, adding that in some cases the team could detect scrapie infection in hamsters which had not yet shown any symptoms.

The debate about the test arises from the difficulty in detecting CJD. Ms Gabizon says that there is only one substance which all scientists agree is evidence of CJD infection. This substance is known as PrPSc and forms part of the prion or rogue protein believed to cause the disease. Her test detects a related substance that she calls UPrPSc. She says that she is confident that the 2 are linked because her test produced reliable results. Researchers from

Britain's MRC Prion Research unit, who are world leaders in the field, however, have their doubts. They do not believe that there have been sufficient controls to show that the Israeli team really are detecting prions. (Since there have been no deaths from vCJD in Israel, the precise identity of the human samples utilised by the Israeli group is unclear from this press statement. - Mod. CP)

The question is whether prions really do move from the brain to urine. If prions move into urine, the British team thinks it is likely that the rogue proteins would accumulate in the kidneys, where they would be found by doctors conducting postmortem examinations.

Canada (British Columbia) - primary multi-drug resistant HIV cases

Source: The Vancouver, 9 August 2001

Doctors at St. Paul's Hospital in Vancouver, British Columbia have seen about 6 cases of multi-drug resistant HIV in the past year, raising concerns that this may signal a new epidemic of resistant strains of HIV. While it is not uncommon for patients to develop a form of resistance to some of the drugs they are taking, there have only been a few reported cases worldwide where patients are newly infected with a type of HIV that is resistant to all 3 classes of antiretroviral drugs.

Patients infected with a multi-drug resistant strain are less likely to respond to different combinations of the drugs. Doctors at St. Paul's who recently treated 2 patients infected with multi-drug resistant HIV stated that in both cases, the virus spread very rapidly within a few months. It is not currently clear whether these strains are more aggressive than those that have been reported in other parts of the world.

Salmonella Typhimurium outbreak in Sweden from contaminated jars of helva (or halva)

From: Birgitta de Jong <birgitta.de.jong@smi.ki.se>

In early June 2001, at least 10 people resident in the south of Sweden were found to be infected with *Salmonella Typhimurium* definitive phage type (DT)104. They were mostly children with a predominance of Arabic names. An earlier outbreak of *S. Typhimurium* infection, involving both DT9, DT30, and probably also not specifically typable (NST) strains of *S. Typhimurium* that had a common phage type pattern, was associated with the consumption of tahini (sesame paste), with most cases also in the south of Sweden. It was therefore suspected that the new cases had acquired the infection in a similar way.

The first interviews showed, however, that the cases had not been eating tahini, but imported helva (or halva)- a type of dessert or sweet made from sesame seeds. The first case in the recent outbreak of *S. Typhimurium* DT104 infection fell ill on 13 April 2001 and the latest reported case on 19 June 2001. The investigation showed that 27 people (23 from the south of Sweden - 2 of them asymptomatic - and 4 from another county) had become infected after consuming helva. Of the 4 people in the nearby county, 3 belonged to the same family.

Salmonella of the same type has also been directly isolated from 5 jars of helva, 4 with pistachio and one with cocoa flavouring. Information about salmonella isolated from helva (pistachio flavour) was first disseminated by the Swedish Food Administration on 11 June 2001, with notification on 20 June that cocoa-flavoured helva was also contaminated.

In recent years, Smittskyddsinstitutet (SMI, Swedish Institute for Infectious Disease Control) has seen an increase of salmonella infection from outbreaks and from food samples associated with different types of imported vegetables, spices, and seeds, including tahini, fresh and dry spices, banana leaves, and bean sprouts.

Malaria emerging in former southern Soviet republics

Source: WHO regional office Europe, 4 June 2001 (edited)

Data extracted from the database of notified Diseases held by the WHO Regional Office for Europe, show that malaria has been emerging in the former Soviet republics of Armenia, Azerbaijan, Georgia, Russia proper, Tajikistan and to a lesser degree Turkmenistan and Uzbekistan. It seems that the number of recorded cases are decreasing in Armenia, Azerbaijan and Tajikistan (Table). Almost all cases are *Plasmodium vivax.*

Table. Notifications of malaria, former southern Soviet republics, 1992 to 2000

	2000	1999	1998	1997	1996	1995	1994	1993	1992
Armenia	141	329	542	567	149	0	1	0	0
Azerbaijan	1526	2311	5157	9911	13,135	2840	667	23	27
Belarus	-	1	2	0	0	0	0	0	0
Georgia	164	35	14	0	3	0	0	0	0
Kazakstan	6	1	4	0	1	0	1	0	1
Kyrgyzstan	7	0	5	0	1	0	0	0	0
Russia	47	77	63	31	10	4	1	1	0
Tajikistan	18,446	13,493	19,351	29,794	16,561	6103	2411	619	404
Turkmenistan	18	10	115	4	3	0	1	1	5
Uzbekistan	46	7	0	0	0	0	0	0	0

Linezolid-resistant MRSA isolated from a patient in the United States

Source: Eurosurveillance Weekly (edited)

A strain of methicillin-resistant *Staphylococcus aureus* (MRSA) that was resistant to the new antibiotic linezolid was isolated from an 85-year-old patient undergoing peritoneal dialysis in the United States, according to a report published in the *Lancet* last week.[1]

MRSA infections are a major problem in many hospitals, and treatment with other antibiotics, usually vancomycin, may be indicated. Linezolid is a new antibiotic that may be an alternative to vancomycin.[2] It prevents the formation of functional ribosomal complexes, thus inhibiting protein synthesis, and it is active against MRSA, *S. epidermidis*, streptococci (including penicillin-resistant strains of *S. pneumoniae*), and enterococci (including vancomycin-resistant strains of *Enterococcus faecalis* and *E. faecium*). It is bacteriostatic against most susceptible bacteria.

Over a 3-week period, 11 linezolid-susceptible isolates were recovered from the patient's peritoneal dialysis fluid, during which time the patient (who was intolerant of vancomycin) received treatment with linezolid. The isolates had identical susceptibility profiles and were indistinguishable by pulsed field gel electrophoresis (PFGE). Of the subsequent isolates, 3 were resistant to linezolid and differed from earlier isolates in their other antimicrobial susceptibilities. Minimum inhibitory concentrations of linezolid were 2 mg/L for linezolid-susceptible and >32 mg/L for linezolid-resistant isolates.

By PFGE, the linezolid-resistant isolates were unrelated to the earlier susceptible isolates. Of the resistant isolates, two were indistinguishable from each other, and a third differed by just one band. Linezolid treatment was discontinued when the resistant isolate was identified, and during the remainder of his hospital stay, the patient received ampicillin, azithromycin, gentamicin, levofloxacin, and quinupristin-dalfopristin for the MRSA and for *E. faecalis*, which was grown from blood cultures, and *Pseudomonas aeruginosa*, which was also isolated from peritoneal fluid. All cultures were negative within one week, but 3 weeks after the last positive cultures, the patient died of his underlying disease.

Possible explanations for this unexpected finding include the acquisition of an unrelated linezolid-resistant MRSA isolate from an external source; the appearance of a previously undetected linezolid-resistant clone within the patient; or the emergence of resistance to linezolid in a previously undetected susceptible clone that was coinfecting the patient. No linezolid-resistant *S. aureus* was recovered from any other patient at the institution, suggesting that the third of these possibilities is the most plausible. The emergence of resistance to linezolid in MRSA is an unwelcome development, and future cases will have to be watched closely. Strict infection control measures are essential as and when such strains are encountered in the future.

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Increased Aedes aegypti mosquitoes in Indian ports

Source: The Lancet, 28 April 2001 (edited)

Indian health experts have been alarmed by rising numbers of *Aedes aegypti* mosquitoes, the key vector of yellow fever, around sea and airports. Experts are concerned that the virus may be introduced into India if precautions are not taken. India and other Asian countries have so far been free from the virus, which is found in Africa and South America. Studies by the National Institute of Communicable Diseases (NICD) and the National Anti-Malaria Programme in different parts of India have pointed towards this increasing trend.

Under the International Health Regulations of 1969, all international airports and seaports should be kept free from larvae and adult mosquitoes and their index should be less than 1 per cent. According to a report by India's NICD, the larval premises index at international seaports increased from 8.8 per cent in 1997 to 29.6 per cent in 2000 in Calcutta, from 0 per cent in 1964 to 22.8 per cent in 1999 in Chennai, and from 0 per cent in 1961 to 12.2 per cent in 1995 in Bombay. The larval index has also been rising at international airports: from 0 per cent in 1978 to 26.9 per cent in 2000 in Calcutta, 13.5 per cent in 1998 to 38.8 per cent in 1999 in Chennai, 0 per cent in 1956 to 9.2 per cent in 1995 in Bombay. In Delhi, it rose from 0 per cent in 1977 to 60.7 per cent in 2000.

Experts point out that yellow fever could be introduced to India through the unnoticed arrival of any subclinically infected patient or mosquitoes infected with yellow fever (virus) carried on aircraft or ships. NICD officials claim that it is the responsibility of the civil aviation and seaport authorities to keep the mosquito populations in check. They also say that India, with an unvaccinated and susceptible population, is a yellow fever 'receptive area'. *Aedes aegypti* mosquitoes are seen in abundance in both urban and rural areas. 'The only missing link in the chain of disease transmission is the yellow fever virus,' stated the director of NICD.

'This situation is a cause of serious concern,'say the WHO. 'Given the eastward movement of the virus, we are very concerned about the introduction of yellow fever into this part of the world'. 'The *Aedes aegypti* population in India is definitely increasing,' said the NICD. 'If this rising trend is not checked via regular monitoring, the chance of introduction of yellow fever will increase,' he added.

Man hospitalised in Kazakhstan with plague

Source: ITAR/TASS News Agency

A 41-year-old man has been hospitalised with plague in Aralsk, Kyzyl-Orda region of Kazakhstan. Medics are taking anti-epidemic measures in the place of his residence, the Agency for Emergency Situations told Itar-Tass on Saturday. The first death from plague in the last 25 years was reported in Kazakhstan in 1999 when a 13-year-old boy died from bubonic plague in Aralsk. Natural plague foci are found in the western and southern regions of the country.

Deaths following yellow fever vaccination

Source: Eurosurveillance Weekly 26 July 2001 (edited)

The *Lancet* of 14 July 2001 collated reports of 6 deaths following yellow fever vaccination in Brazil, the USA, and Australia in the period 1996-2001.[1,2,3] The clinical pictures were not consistent and different vaccine strains had been used on each continent.

The Brazilian cases (aged 5 and 22) were confirmed as vaccine-derived, but represent 2 deaths in over 85 million vaccinations over a 10-year period. The investigators were surprised by the clinical features of the vaccine-related Diseases, which included significant organ damage normally only associated with wild-type yellow fever. Since neither the vaccine nor its stabiliser had changed, it is possible that the 2 deaths were due to unidentified host factors.

The 4 US cases were all more than 62 years of age and had co- morbidity. Although the clinical features and their timing suggest yellow fever vaccine as the cause of the 3 deaths and one severe illness, only one of these cases had antigenic evidence of vaccine-derived virus. Unlike the Brazilian cases, the US cases showed significant central nervous system involvement and a lesser degree of organ damage.

The one Australian case was aged 56 years and died of multi-system disease. His death can be attributed by virus isolation to vaccine- derived yellow fever. The same day, however, 20 other people were immunised with yellow fever vaccine from the same batch and remained well.

It is a feature of all 7 cases that death or severe illness occurred in a small number of people among a much larger population who received the same batch of vaccine. No obvious risk factors, apart from age in the American cases, could be identified, and an accompanying *Lancet* commentary concludes that 'the use of 17D vaccination remains highly advisable for people living in or travelling to endemic and epidemic zones, but that these reports raise questions about the mechanisms of attenuation of yellow fever virus that should be urgently investigated'.[4] This view is supported by WHO. However, WHO cautions that 'travellers should be carefully assessed regarding their need for the vaccine and their personal level of risk'.[5]

Yellow fever is caused by a flavivirus and produces an acute disease with a mortality rate of up to 50-60 per cent in non-immunes. An anthropozoonosis, it is transmitted to man by mosquitoes within the endemic zones of Africa and South America. The main reservoirs of infection are mosquitoes and vertebrates (usually monkeys) in forest areas (forest or sylvan yellow fever), except for when there is transmission in towns from person to person by mosquitoes (urban yellow fever) when humans become part of the reservoir.[6]

A live attenuated vaccine based on a 17D version of the virus was developed (by Nobel prize winner Max Theiler of The Rockefeller Foundation) in 1937, and all the currently produced vaccines are based on this. The strains of vaccine used around the world are essentially similar, but contain at least 2 different substrains, 17DD and 17D-204. The vaccine is highly protective, and until recently has been regarded as extremely safe. (It should still be regarded as such; as few as 6 deaths, while regrettable, in 150 million doses is a safety record unmatched by any other highly effective live attenuated vaccine except, perhaps, the Sabin vaccine for polio.)

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