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CONTENTS

Contributions covering any aspect of communicable diseases are invited. Instructions to authors can be found in CDI 1995; 20: 13.

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MENINGOCOCCAL DISEASE IN AUSTRALIA

Rosemary Munro[1] *and John Tapsall*[2]

Abstract

Although the overall incidence if invasive meningococcal disease in Australia is not high by international standards, *Neisseria meningitidis* is a major cause of bacterial meningitis and septicaemia in a number of patient subgroups. Clusters of cases are not infrequently recorded. This situation presents significant challenges to public health responses in both the immediate and longer term. Resolution of these problems would be advanced by more accurate and complete data on disease patterns, invasive meningococcal subtypes and antibiotic susceptibility patterns. The prospect of more efficacious conjugate vaccines gives further impetus to the need for improvement of these data. ***Comm Dis Intell* 1996;20:368-371.**

Introduction

Neisseria meningitidis is a strictly human, but highly versatile pathogen responsible for invasive disease throughout the world. Meningococcal infection is manifest most frequently as a meningitis or as an often fulminant septicaemia. It occurs in epidemics, smaller outbreaks and sporadically without apparent linkage to other cases. Rapid and accurate characterisation of isolates is an essential part of the preventative public health response to a possible outbreak.

This review will summarise features of N. meningitidis which allow differentiation of strains, what is known about the epidemiology of meningococcal disease in Australia, antimicrobial susceptibility patterns and their influence on treatment recommendations, outcome of infection, and potential roles of new vaccines in Australia.

Epidemiology of meningococcal disease in Australia

Incidence of disease

Data on many aspects of meningococcal disease in Australia are incomplete. The National Notifiable Diseases Surveillance System provides information on sex, age, onset date and place of occurrence of cases, but provides no data on strains causing disease, clinical presentation and outcome. Meningococcal disease was not a separate notifiable disease in some States until the late 1980s. It is an infection with cyclical peaks of incidence. Notification of 'meningitis' showed a peak of 33.1 cases per 100,000 in 1942 (2,371 cases); this was followed by a slow decline in incidence until another peak in the early 1970s. By the late 1970s notifications were categorised nationally as meningococcal disease[1]. There was then another decline to less then 0.5 cases per 100,000 until 1987, when an increase was again seen. The increase has been sustained with annual rates of 1.6 to 2.2 per 100,000 1991-94[1,2,3]. The highest attack rates are seen in children less than five years of age, particularly in Aboriginal communities in central and northern Australia. In Aboriginal children less than five years of age in the Northern Territory, Western Australia and New South Wales, the incidence since 1991 is estimated to be 66 per 100,000[4]. The incidence decreases with age, but in Australia, as in many other developed countries, there is a second smaller peak of incidence in the 15-20 years age group. The peak incidence for meningococcal disease is during winter, July to September.

Knowledge of strains causing disease in Australia

The National Neisseria Network (NNN), a laboratory-based meningococcal surveillance system commenced providing standardised national information on serogroups, antibiotic susceptibility patterns, patterns of disease and mortality rates in 1994[5]. National data on serotypes and subtypes were provided from 1995. Otherwise, data on strains and patterns of disease in Australia have been sporadic and regional. Large-scale outbreaks of meningococcal disease have been confined to Aboriginal communities in central and northern Australia.

In 1971 to 1973, there was an epidemic in central Australia affecting predominantly Aboriginal communities with an annual attack rate of 321 per 100,000[6]. Organisms which were serogrouped from this outbreak were serogroup A. In 1987 to 1991, another epidemic, predominantly serogroup A, recurred in central Australia[7]. Both these outbreaks had characteristics of epidemics of serogroup A seen regularly in Africa and other developing countries. Apart from these outbreaks, serogroup A organisms are rarely isolated[5,8,9]. Serogroup B organisms have been the main cause of sporadic disease in Australia, as in other developed countries[5,8,9]. From 1987 to the early 1990s, there were reports from different areas of Australia of an increase in the frequency of serogroup C isolations.

Meningococcal strain differentiation

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Strain differentiation is undertaken to determine isolate relatedness and identify possible outbreaks, to map the spread of virulent strains, and to assist in determining vaccine efficacy.

Serogroup, serotype, subtype

The most universal methods of strain differentiation involve the sequential serological procedures of serogrouping, serotyping and subtyping. Organisms isolated from systemic sites have polysaccharide capsules which allow differentiation into 13 distinct serogroups. Serogroups A, B, and C account for 90% of invasive infections. There are five different structural classes of outer membrane proteins (OMPs) located in the cell wall of the organism. *Neisseria meningitidis* is a transformable organism capable of frequent recombination events causing alterations in antigens exposed on its surface. Variations in these OMPs can be used to further differentiate organisms within serogroups. Meningococci of different serogroups can be divided into 20 serotypes with six common ones, using monoclonal antibodies directed against class 2/3 OMPs. Similarly, variations in class 1 OMPs form the basis of a subtyping system involving 13 common subtypes[10].

Most laboratories perform serogrouping of invasive meningococcal isolates. This basic information will provide some valuable epidemiological information. The epidemic forms of meningococcal disease affecting many thousands of people in Africa and other developing countries are almost always serogroup A. In developed countries where endemic and hyperendemic disease is more usual, there are geographical and temporal variations in the occurrence of serogroup B and C strains.

Monoclonal antibodies for serosubtyping were developed over ten years ago, but only in the last three to four years they have been widely available in a standardised form. However, a significant proportion of strains (about 40%) will be nontypable by one or other method. This is partly related to the titre of monoclonal antibody attainable, but more importantly to the inability of monoclonal antibodies to recognise many variants of OMPs caused by recombination events, or to react differently with strains with only very minor genetic differences.

Multilocus enzyme electrophoresis (MLEE)

This is a phenotypic method which identifies 14 to 20 intracellular enzymes by electrophoretic separation and action on a range of substrates[11]. Because these enzymes are less susceptible to genetic variation than cell surface proteins, their electrophoretic mobilities can be used to group organisms into clones or clusters called electrophoretic complexes. All isolates can be assigned an electrophoretic type (ET) and grouped into genetically related ET complexes. The method has been used in many countries, allowing study of the global spread of some clones and changing incidence of particular ETs within countries. Unfortunately, there is very little information about ETs of isolates in Australia.

DNA typing techniques

A range of DNA typing techniques has been applied to meningococci; the most widely used is pulsed field gel electrophoresis (PFGE)[12]. PFGE, which examines the whole chromosome, has mainly been used to examine whether epidemiologically related strains collected during an outbreak are genetically related or are the same strain. It complements the epidemiological data. Interpretation is subjective, and small genetic variations which occur frequently in meningococci which are closely related may further make interpretation difficult. Recent recommendations for criteria for PFGE typing, recognising that evidence for clonality is relative rather than absolute, will assist with these problems[13]. Other methods which examine total chromosomal DNA include restriction fragment length polymorphisms (RFLP) with cloned meningococcal DNA as a probe or with ribosomal RNA as a probe (ribotyping)[14,15].

There are also DNA typing techniques applied to meningococci which examine variation in single genes or small numbers of genes. Polymerase chain reaction (PCR) amplification of the *por* A gene encoding the class 1 OMP with RFLP of PCR products and random amplified polymorphic DNA PCR are two such techniques[16,17].

DNA typing techniques have the advantage over serological techniques in that all strains are typable. In general, DNA typing correlates with results obtained by MLEE and will also discriminate amongst organisms in specific ET complexes. Reports on the use of these techniques have mainly been retrospective analyses of collections of organisms: a number of techniques has reliably identified epidemiologically related strains[18].

Choice of strain differentiation techniques

The choice of a typing technique will depend in part on practical considerations such as cost, technical complexity, and time to obtain results, but will also be influenced by the reason strain differentiation is being undertaken.

Outbreak investigations

If rapid identification of epidemiologiccally related cases is required so that a public health response can be mounted to control a possible outbreak, a DNA typing technique is ideal. Serogrouping and serosubtyping will be helpful, but only if strains are typable. MLEE, although typing all strains, is too slow and complex for outbreak identification. *Por* A PCR amplification with RFLP of products is one of the most rapid DNA typing techniques. Random amplification polymorphic DNA (RAPD) PCR, another rapid typing technique, has lacked reproducibility in our hands. PFGE or RFLP with meningococcal DNA or rRNA probes take several days to perform.

Studying the epidemiology of meningococcal disease over time

Serotyping and MLEE are standardised strain differentiation techniques used in many countries over a number of years. Valuable information can be obtained about the spread of strains in different geographic areas over time and comparisons made with other countries by characterisation of all meningococcal isolates using these two methods.

There has been little work using DNA typing techniques to study prospectively meningococci causing sporadic disease. Ongoing characterisation of serogroup B meningococci in New Zealand has indicated that the marked increase in disease incidence since 1991 coincided with an increase in frequency of one particular genotype as defined by RFLP, this type being uncommon before 1991[19]. DNA typing techniques examining total chromosomal DNA may be more applicable to epidemiological investigations of spread of sporadic strains than techniques examining variation in smaller numbers of genes. Combinations of techniques may be required to give optimal information.

In addition, there were reports of clustering of cases of serogroup C disease, both in urban and rural settings[20,21,22,23]. At that time there was increased serogroup C activity in a number of countries throughout the world. However, data from the NNN in 1994-95 indicate that serogroup B organisms again predominate and have not been associated with clusters or outbreaks of disease in Australia[5].

Data are limited about serotypes and subtypes of meningococci causing disease in Australia, and there has been considerable geographical variation in the prevalence of particular serotypes and subtypes. In South Australia and the Northern Territory in 1971 to 1989, the predominant serotypes were 4, 2a, 15, and 14, with subtypes P1.2, P1.1, and P1.10 in South Australia. In the Northern Territory all serogroup A isolates were 4:P1.10. Serogroup B and C strains showed considerable heterogeneity[24]. In south-western Sydney in 1990-94[25], serogroup C accounted for 60.8% (31 / 51) of invasive meningococcal isolates. Eighty per cent of these serogroup C isolates were serotype 2b and 70% subtype P1.2. Thus during this five year period the phenotype C:2b:P1.2 was the commonest sporadic isolate and was also associated with the cluster of cases described in 1991[20]. The phenotype C:2b:P1.2 has been involved in all the serogroup C outbreaks mentioned above (J. Jelfs, personal communication). Outbreaks many thousands of kilometres apart occurred in 1990-1991. Sporadic cases of the C:2b:P1.2 phenotype continued to occur in New South Wales over subsequent years, but have become less common, along with an overall decrease in serogroup C strains in New South Wales in 1994-95[26]. Molecular analysis of outbreak and sporadic strains of this phenotype is currently being undertaken to investigate whether one or more clones are involved.

Antibiotic susceptibility patterns

Many countries have noted the appearance of meningococcal isolates with altered penicillin susceptibilities in recent years. Laboratories of the National Neisseria Network have determined the antibiotic susceptibility of more than 450 invasive isolates, using standardised agar dilution techniques since 1994. Although 72.5% of isolates showed a decreased susceptibility to penicillin (minimal inhibitory concentration (MIC) $\leq$0.06mg / l), the data indicate that penicillin-based treatment regimes remain suitable for use in Australia. A study in Victoria of invasive strains isolated over six years from 1988 found little or no increase in resistance[27]. No beta lactamase-producing isolates have been detected in Australia.

Outcome of meningococcal infection

Factors influencing the outcome of meningococcal infection include the clinical syndrome on presentation, age, timing of commencement of antibiotic treatment and strain causing infection. In the study from south-western Sydney where clinical and laboratory criteria were used to classify patients as having predominantly meningitis (20.7%), meningitis / septicaemia (53.4%), or septicaemia (22.4%), the mortality rate in the meningitis group was 0, in the meningitis / septicaemia group it was 6.5% and in the septicaemia group it was 30.8%[25]. An attempt to prevent rapid bacterial multiplication is the rationale for the early use of parenteral penicillin to improve outcome[28]. This recommendation remains controversial, because there is a subset of patients with meningococcal disease who present with shock and rapidly developing multi-organ failure in whom there is a high mortality rate despite early appropriate antibiotic therapy. However, early recognition and treatment of meningococcal meningitis remains an important goal.

It is not common for Australian general practitioners to use parenteral antibiotics before transferring the patient to hospital. A delay in diagnosis and administration of appropriate intravenous therapy of more than two hours occurred in 36.2% of patients in the south-western Sydney study[25]. By comparison, there was a median delay to treatment in an Auckland, New Zealand, survey of 80 minutes[19]. There is room for improvement in the time taken to diagnose meningococcal infection and institute appropriate intravenous treatment. It is particularly important that intravenous antibiotics are not delayed while investigations such as lumbar puncture or CT scan are performed. The prior use of antibiotics does decrease the number of positive cerebrospinal fluid (CSF) cultures; however, there is very often other laboratory evidence of meningococcal disease[25]. Polymerase chain reaction (PCR) has also been used in blood and CSF to make a non-culture diagnosis of meningococcal disease[29].

Meningococcal vaccines

Meningococcal vaccines currently available in Australia are polysaccharide vaccines directed against serogroup A, C, W_{135} and Y strains. Polysaccharide vaccines are poorly immunogenic and unable to generate immunological memory. Thus response rates to the current serogroup vaccine are poor in young children, antibody titres are of short duration, and there is failure to respond to subsequent vaccination. Polysaccharides can be changed to T cell-dependent antigens by structural modification of the polysaccharide sialic acid polymer subunit so that it can be conjugated to a protein carrier such as tetanus toxoid, CRM (a nontoxigenic mutant diphtheria toxin), or a meningococcal OMP. Immunogenicity trials of these conjugate vaccines in infants and toddlers have shown high levels of bactericidal antibodies with good immunological memory. Similar modifications of the polysaccharide sialic acid polymer subunit of serogroup B organisms coupled with a protein carrier have produced a vaccine giving good levels of bactericidal antibodies and immunological memory[30].

The use of capsular polysaccharide vaccines is preferred because of the lack of strain variability in polymer subunits and the production of protective bactericidal antibodies post-vaccination. Another approach to vaccination, especially against serogroup B organisms whose capsular polysaccharide is poorly immunogenic, is the use of outer membrane protein antigen vaccines. Vaccine antigens may be prepared from outer membrane vesicles and combined with meningococcal capsular polysaccharide for improved solubility. Alternatively multivalent

vaccines against a number of class 1 OMPs can be prepared by insertion of OMP genes into meningococcal strains and subsequent purification of outer membrane vesicles. The limitation of these vaccines against group B meningococci is that the protection induced is serotype and subtype specific. Thus vaccines appropriate for one country or particular geographical area may not be suitable in another area.

The polysaccharide serogroup A, C, W_{135} and Y vaccines formerly have been used successfully to control large outbreaks of serogroup A disease in Australia and for control of some of the smaller outbreaks and clusters of serogroup C disease[7,20,21,23]. It is doubtful whether there would ever be a case for universal vaccination against meningococcal disease in Australia with the newer, more effective vaccines. The overall incidence of meningococcal disease of less than 2 per 100,000 places Australia in the category of a low-incidence country. However, attack rates in Aboriginal communities in central and northern Australia are much higher, approaching the incidence of *Haemophilus influenzae* in Aboriginal children less than five years of age before mass vaccination programs began. In this group, universal vaccination with new meningococcal vaccines effective against serogroups A, B, C, W_{135} and Y would be very worthwhile.

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CHANGES AT THE *CDI* DESK

Charles Watson, Chair, CDI Editorial Advisory Board and Ana Herceg, Acting Editor, CDI

After two and a half years, Dr Helen Longbottom has moved on from her position as Editor of *Communicable Diseases Intelligence.* Her dedication and hard work have ensured that *CDI* is a publication of high quality. Just as importantly she leaves behind a committed team to continue the work. Helen's departure gives us occasion to reflect on her accomplishments as Editor and to look forward to further developments.

During the time that Helen was Editor, a number of significant changes were made to *CDI*, including the establishment of the Editorial Advisory Board. The Board has contributed to the development of *CDI*, as has an ongoing review process which included the readership survey conducted last year (see *CDI* 1996;20:39-41).

Perhaps the most important change that occurred under Helen's stewardship was the commencement of a peer review process. In addition a process of soliciting articles and editorials on specific topics was commenced.

New sections of *CDI* have been introduced, including *Correspondence* to allow debate, and an *Outbreaks* section to report on current events. A number of changes have also been made to the layout of the sections. Since November 1995, *CDI* has been accessible through the Internet on the World Wide Web.

Improvements have also been made to the *Communicable Diseases Surveillance* section of *CDI*, both in explaining the significance of the different surveillance systems and highlighting the important trends identified from the data in these systems.

The *CDI* team is committed to further developing our publication, in the process of continuous improvement commenced by Helen in her time as Editor. Our aim is to maintain *CDI* as a valuable national resource for those working in communicable diseases. We would very much appreciate receiving comments from readers on ways to improve the publication. We hope you will continue to use and contribute to *CDI.*

OUTBREAK

Two linked cases of Legionnaires' disease, South Australia

Martyn Kirk[1,2], *Rachel Wells*[1], *Carolyn Walker*[1] *and Jan Lanser*[3]

In the week of 14-20 July 1996, the South Australian Health Commission was notified of two cases of Legionnaires' disease, both of whom had stayed in the same hotel during the incubation period. The patients, a 54 year old female and a 49 year old male, were both smokers with no other obvious risk factors. Both patients had *Legionella pneumophila* serogroup 1 isolated from clinical specimens and subsequently died. An environmental assessment of the hotel revealed that the spa pool was inadequately disinfected and that recent renovations had interrupted the hot water supply to hotel rooms. No epidemiological link with these sources was confirmed, although one of the cases had been in the room housing the spa pool while the spa was operating. The other case had not been near the spa, although he may have worked near an exhaust vent for the spa pool room.

As a precautionary measure, the hotel spa pool was closed and the hot water system heat disinfected. Active surveillance for Legionnaires' disease, initiated as a result of this cluster, did not detect any other cases associated with the hotel. A media release alerted people who had stayed at the hotel since the beginning of July to seek medical attention if they had symptoms consistent with legionellosis.

Culture of a water sample from the spa pool grew *L. pneumophila* serogroup 1 pontiac strain. *Legionella* isolates from the cases and the spa pool were identical by restriction fragment length polymorphism testing. This particular strain of *L. pneumophila* serogroup 1 has been found previously in South Australia; from an outbreak in 1986 and from sporadic cases, including two epidemiologically unrelated cases this year. No *Legionellae* were cultured from samples of water entering the hotel or from showers in guests' rooms.

In South Australia this year, there have been seven notified cases of *L. pneumophila* serogroup 1 infection with four deaths. One case notified in New South Wales had been in South Australia during the incubation period.

Except for the two cases visiting the same hotel, no epidemiological links between the cases were identified. Five of the eight cases investigated had been in or near (separate) spa pools during the incubation period.

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Legionellae are opportunistic pathogens ubiquitous in aquatic and soil environments. *Legionellae* can amplify in spa pools, as water is maintained at temperatures optimal for growth. Spa pools also generate aerosols, making them particularly effective vehicles for transmission of *Legionella* infections. However, traditional disinfection of spa pool water with chlorine or bromine is effective against *Legionella*, provided the disinfectant is continuously applied[1]. Most States and Territories have legislation covering the disinfection of public spa pools. The National Health and Medical Research Council has published guidelines for the maintenance of private spa pools, which are currently being revised[2].

In this instance a source of infection for the two cases was not confirmed, although *L. pneumophila* serogroup 1 was isolated from the hotel spa pool. However, spas have been shown to be a source of Legionnaires' disease in outbreak settings, even where people had not entered the spa pool but had been in the vicinity of the implicated pool[3]. As a result of the possible association of legionellosis with spa pools, the South Australian Health Commission has reinforced to local government and the spa pool industry the need to be vigilant in maintaining effective disinfection of spa pools.

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NOTICES TO READERS

Comments invited on proposed Australian Standard Vaccination Schedule

Proposed changes to the NHMRC Standard Childhood Vaccination Schedule are now available for public consultation. The new schedule will be called the Australian Standard Vaccination Schedule. The Schedule is updated as new vaccines come onto the market, and as research offers additional information about the efficacy of vaccines.

The NHMRC Immunisation Working Party, following wide consultation, has agreed on a number of proposed changes to the current vaccine schedule including:

- the use of DTaP (diphtheria, tetanus and the new acellular pertussis vaccine or DTwP (whole cell pertussis) for the 18-month and four to five year old booster injections;
- postponement of the introduction of DTaP for primary vaccination pending vaccine registration for that purpose, clarification of the clinical efficacy of the difference, if any, of DTwP and DTaP for primary vaccination, and availability of the vaccine at an acceptable price;
- the introduction of a three-dose series of hepatitis B vaccination for pre-adolescents;
- postponement of the introduction of universal hepatitis B vaccination for infants, pending the availability of multivalent vaccines very soon. However, recognising that the National Health and Medical Research Council has endorsed the use of hepatitis B vaccine for all infants, advice on when this vaccine should be administered is provided;
- no change to the current schedule for Hib vaccines;
- inclusion of milestones for adults.

The draft Australian Vaccination Schedule is open for public consultation until 5 September 1996. Copies are available from the Working Party Secretariat on (06) 289 9319.

Submissions should be sent to:
The Secretary, Immunisation Working Party, National Health and Medical Research Council, GPO Box 9848, MDP 13, Canberra ACT 2601, Attention: Monica Johns.

Review of *CDI* mailing list - a reminder

The flyers that you received with the previous issue and this issue of *Communicable Diseases Intelligence* have an important notice printed on them regarding our mailing list.

If you wish to continue receiving *CDI*, you must complete one of the flyers and return it to this office by Monday, 16 September 1996. If you do not notify this office by that date, your name will be removed from the mailing list.

Please return only one form

Would you also check that your name and address are correct. Mark any changes needed to help us bring our records up to date. Please return the flyer to Surveillance and Epidemiology Section, Department of Health and Family Services, MDP 15, GPO Box 9848 Canberra ACT 2601, or fax it to (06) 289 7791 before 16 September 1996. You can fold the flyer, tape it together and post it to the address shown.

OVERSEAS BRIEFS

Source: World Health Organization

Cholera in Mongolia and Liberia

The Ministry of Health in Mongolia reported an outbreak of cholera on 9 August 1996. This was the first official confirmation of cholera in the country. As of 13 August, 52 cases had been reported with six deaths.

In Liberia, the Ministry of Health and Social Welfare has announced a steady upward trend in cholera figures in the city of Monrovia and its environs since April 1996. This increase was initially in areas of inadequate sanitation and water supply but has recently been apparent across the city, changing an endemic situation to a cholera epidemic.

Dengue, Venezuela

The number of cases of dengue and dengue haemorrhagic fever has increased markedly since mid-July, particularly in the Federal District, Barina, Lara and Tachira. In the week ending 4 August 1996, 293 cases of dengue were reported (including 33 cases of dengue haemorrhagic fever). A major epidemic occurred in 1995 causing 5,098 cases.

Viral meningitis, Cyprus

An outbreak of viral meningitis has affected 223 persons in Cyprus since 5 July 1996. Of these, 193 were children under 14 years of age. The causal virus has been identified as coxsackie B5. The outbreak is centered in the district of Limassol (170 cases); cases were also reported from Larnaca, Famagusta (29), Nicosia (19) and Paphos (5). Of the 223 patients, 190 have been discharged from hospital; no complications were reported.

Venezuelan equine encephalitis diagnosed in Mexico

Thirty-two cases of Venezuelan equine encephalitis have been diagnosed among 2,500 horses in three foci in the State of Oaxaca. These were in rural areas often flooded during rainy periods. The first case was detected on 16 June and the last occurred on 23 July 1996. Ten horses died. No human cases have been detected.

Venezuelan equine encephalitis virus was isolated from two horses. The virus isolates were further identified as sub-type 1, variant E which is enzootic in the south-east coast of Mexico and in Central America. The transmission cycle involves small mammals and mosquitos.

Typhoid fever, Tajikistan

As at 5 August, 7,516 cases of typhoid fever had been reported. Of these, 2,515 (34%) were children under 14 years of age. Of the 2,096 hospitalised cases, 597 (28%) were children under 14 years of age.

COMMUNICABLE DISEASES SURVEILLANCE

National Notifiable Diseases Surveillance System

The NNDSS is conducted under the auspices of the Communicable Diseases Network Australia New Zealand. The system coordinates the national surveillance of 41 communicable diseases or disease groups endorsed by the National Health and Medical Research Council (NHMRC). Notifications of these diseases are made to State and Territory health authorities under the provisions of their respective public health legislation. De-identified core unit data are supplied fortnightly for collation, analysis and dissemination. For further information, see CDI 1996;20:9-10.

Reporting period 21 July to 3 August 1996

There were 1,658 notifications received for this two-week period (Tables 1, 2 and 3). The numbers of reports for selected diseases have been compared with average data for this period in the previous three years (Figure 1).

The number of notifications of **chlamydial infection** was 303 for the current reporting period. Notifications have shown a slight upward trend in recent years (Figure 2). It should be noted that inclusion of notifications from South Australia commenced in 1992 and from Western Australia in 1993. The condition is not yet notifiable in New South Wales. For the 10,750 cases notified since the beginning of 1995, the male:female ratio was 1:2.2. The age spectrum of cases has not altered significantly. About 85% of cases in females are in the age range 15 to 29 years; about 70% of male cases are in the same age in range (Figure 3).

There were 28 notifications of **meningococcal infection** received for the current fortnight, compared to 19 for the previous period. Cases have been reported recently from all States and Territories.

The number of notifications of **rubella** has declined in recent months, although the annual total of notifications remains higher than for 1995. Most recent notifications have been from the south-eastern corner of Queensland. Over the last two months, 88% of rubella laboratory reports in the LabVISE surveillance system have also been received from Queensland. The seasonal pattern of the previous years suggests that a rise in notifications can be expected within the next two months.

Table 1. Notifications of diseases preventable by vaccines recommended by the NHMRC for routine childhood immunisation, received by State and Territory health authorities in the period 21 July to 3 August 1996[1]

DISEASE	ACT	NSW	NT	Qld	SA	Tas	Vic	WA	TOTALS FOR AUSTRALIA[2]			
									This period 1996	This period 1995	This period 1996	This period 1995
Diphtheria	0	0	0	0	0	0	0	0	0	0	0	0
Haemophilus influenzae B infection	0	1	0	0	0	0	0	0	1	2	38	47
Measles	0	3	0	4	0	0	0	1	8	32	274	941
Mumps	1	1	1	NN	0	0	0	0	3	5	62	88
Pertussis	0	28	0	24	16	0	0	3	71	162	1739	2503
Rubella	1	3	0	26	0	1	0	0	39	83	1488	1383
Tetanus	0	0	0	0	0	0	0	0	0	0	1	3

NN Not Notifiable.

1. No notifications of poliomyelitis have been reported since 1986.

2. 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.

Table 2. Notifications of other diseases[1] received by State and Territory health authorities in the period 21 July to 3 August 1996

DISEASE	ACT	NSW	NT	Qld	SA	Tas	Vic	WA	TOTALS FOR AUSTRALIA[2]			
									This period 1996	This period 1995	This period 1996	This period 1995
Arbovirus Infection (NEC)[3,4]	0	0	1	0	0	0	0	3	4	5	132	374
Barmah Forest virus infection	0	1	-	13	0	0	-	-	14	24	600	324
Ross River virus infection	0	4	0	32	0	-	0	2	38	57	7260	2221
Dengue	0	0	0	2	0	-	0	0	2	1	26	16
Campylobacteriosis[5]	9	-	11	126	137	18	0	55	356	426	6368	6105
Chlamydial infection (NEC)[6]	9	NN	33	149	0	11	68	33	303	186	4393	3687
Donovanosis	0	NN	2	0	NN	0	0	0	2	2	32	50
Gonococcal infection[7]	0	13	28	60	0	0	30	26	157	101	2256	1800
Hepatitis A	1	18	8	14	1	0	0	1	43	36	1442	954
Hepatitis B incident	0	0	0	2	0	0	0	0	2	14	128	229
Hepatitis B unspecified	6	0	0	29	0	2	0	13	50	54	906	1012
Hepatitis C incident	0	0	2	-	0	-	-	-	2	12	17	69
Hepatitis C unspecified	12	NN	0	123	NN	17	0	34	186	394	4511	5407
Hepatitis (NEC)	0	1	0	0	1	0	0	NN	2	0	13	17
Legionellosis	0	2	0	1	1	0	0	1	5	6	110	125
Leptospirosis	0	1	0	18	0	0	0	0	19	7	154	76
Listeriosis	0	1	0	0	0	0	0	0	1	2	32	42
Malaria	0	6	0	33	1	0	0	3	43	29	521	410
Meningococcal infection	2	8	1	14	1	0	0	2	28	21	205	211
Ornithosis	0	NN	0	0	0	0	0	0	0	3	55	81
Q fever	0	4	0	3	0	0	0	2	9	23	308	275
Salmonellosis (NEC)	5	38	24	41	12	2	0	16	138	166	3808	4196
Shigellosis[5]	1	-	18	13	0	0	0	6	38	29	419	512
Syphilis	0	34	18	15	0	0	8	1	76	54	1007	1170
Tuberculosis	1	3	1	6	2	0	0	0	13	58	648	689
Typhoid[8]	0	0	0	1	0	0	0	0	1	3	51	42
Yersiniosis (NEC)[5]	0	-	0	0	1	0	0	2	3	13	154	222

1. For HIV and AIDS, see Tables 4 and 5. For rarely notified diseases, see Table 3 .

2. 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.

3. Tas: includes Ross River virus and dengue.

4. NT, Vic and WA: includes Barmah Forest virus.

5. NSW: only as 'foodborne disease' or 'gastroenteritis in an institution'.

6. WA: genital only.

7. NT, Qld, SA and Vic: includes gonococcal neonatal ophthalmia.

8. NSW, Vic: includes paratyphoid.

NN Not Notifiable.

NEC Not Elsewhere Classified.

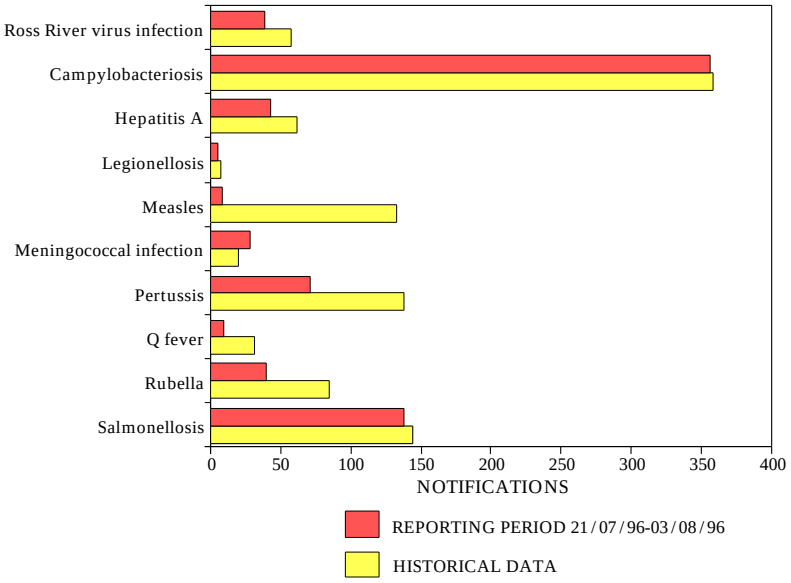
- Elsewhere Classified.

Table 3. Notifications of rare[1] diseases received by State and Territory health authorities in the period 21 July to 3 August 1996[2]

DISEASES	Total this period	Reporting States or Territories	Year to date 1996
Brucellosis	0		22
Chancroid	0		1
Cholera	0		4
Hydatid infection	0		24
Leprosy	1	WA	8

1. Fewer than 60 cases of each of these diseases were notified each year during the period 1988 to 1995.
2. No notifications have been received during 1996 for the following rare diseases: botulism; lymphogranuloma venereum; plague; rabies; yellow fever; or other viral haemorrhagic fevers.

Figure 1. Selected National Notifiable Diseases Surveillance System reports, and historical data[1]



1. The historical data are the averages of the number of notifications in 9 previous 2-week reporting periods: the corresponding periods of the last 3 years and the periods immediately preceding and following those.

Figure 2. Chlamydial infection, notifications 1991 to 1996, by month of onset

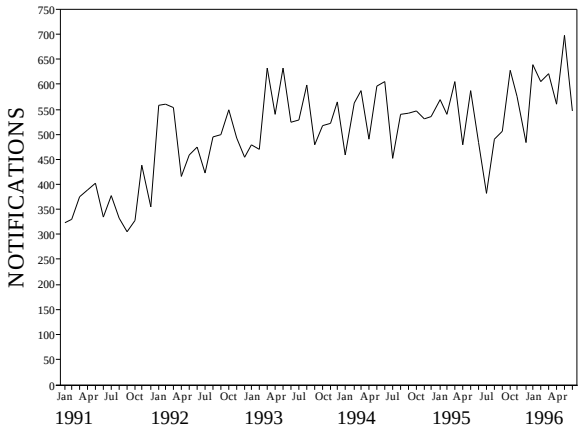


Figure 3. Chlamydial infection, notifications 1995 and 1996, by sex and age group

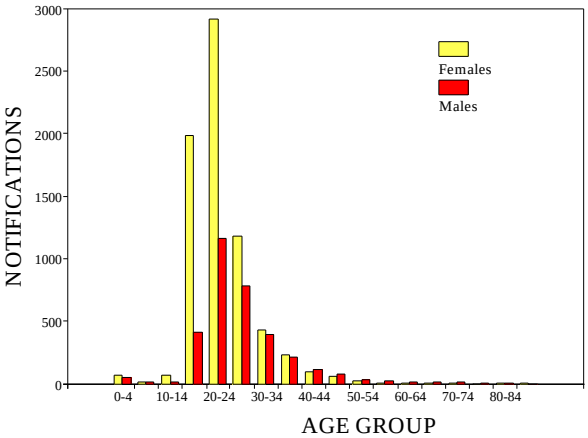


Table 4. New diagnoses of HIV infection, new diagnoses of AIDS and deaths following AIDS occurring in the period 1 to 29 February 1996, by sex and State or Territory of diagnosis

		ACT	NSW	NT	Qld	SA	Tas	Vic	WA	TOTALS FOR AUSTRALIA			
										This period 1996	This period 1995	Year to date 1996	Year to date 1995
HIV diagnoses	Female	0	3	0	2	0	0	1	1	7	7	12	15
	Male	1	21	1	12	1	0	16	6	58	80	120	143
	Sex not reported	0	0	0	0	0	0	0	0	0	1	2	4
	Total[1]	1	24	1	14	1	0	17	7	65	88	134	163
AIDS diagnoses	Female	0	0	0	0	0	0	0	0	0	4	0	7
	Male	0	6	0	3	1	0	6	3	19	66	49	125
	Total[1]	0	6	0	3	1	0	6	3	19	70	49	132
AIDS deaths	Female	0	0	0	1	0	0	3	0	4	4	6	6
	Male	0	19	0	2	5	0	12	0	38	66	62	121
	Total[1]	0	19	0	3	5	0	15	0	42	70	68	127

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

Table 5. Cumulative diagnoses of HIV infection, AIDS and deaths following AIDS since the introduction of HIV antibody testing to 29 February 1996, by sex and State or Territory

		ACT	NSW	NT	Qld	SA	Tas	Vic	WA	AUSTRALIA
HIV diagnoses	Female	15	553	4	96	44	4	159	71	946
	Male	168	9,954	81	1,575	563	70	3,355	755	16,521
	Sex not reported	0	2,049	0	0	0	0	42	0	2,091
	Total[1]	183	12,563	85	1,676	607	74	3,565	828	19,581
AIDS diagnoses	Female	5	130	0	28	18	2	47	18	248
	Male	71	3,744	25	638	267	32	1,320	275	6,372
	Total[1]	76	3,884	25	668	285	34	1,374	295	6,641
AIDS deaths	Female	2	96	0	22	13	2	36	11	185
	Male	50	2,664	20	443	186	21	1,041	201	4,626
	Total[1]	52	2,769	20	467	199	23	1,083	213	4,826

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

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 (ACT, 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, available from the National Centre in HIV Epidemiology and Clinical Research, 376 Victoria Street, Darlinghurst NSW 2010. Telephone: (02) 332 4648 Facsimile: (02) 332 1837.

HIV and AIDS diagnoses and deaths following AIDS reported for February 1996, as reported to 31 May 1996, are included in this issue of *CDI* (Tables 4 and 5).

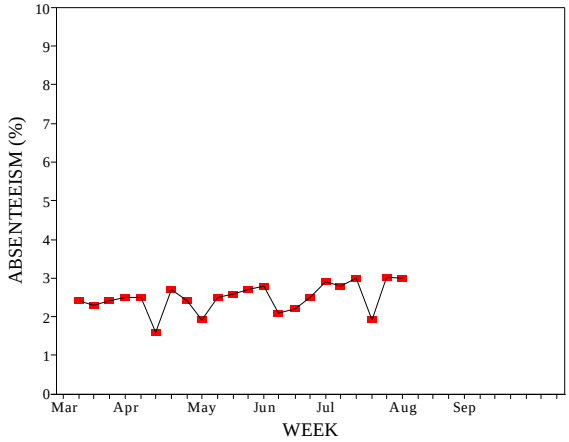
National Influenza Surveillance

Australian Sentinel Practice Research Network; Communicable Diseases Intelligence Virology and Serology Reporting Scheme Contributing Laboratories, New South Wales Department of Health; Victorian Department of Health; World Health Organisation Collaborating Centre for Influenza Reference and Research.

National Influenza Surveillance is conducted from May to September each year. Data are combined from a number of sources to provide an indication of influenza activity. Included are sentinel general practitioner surveillance, absenteeism data from a national employer, and laboratory data from LabVISE and the World Health Organization Collaborating Centre for Influenza Reference and Research. For further information, see CDI 1996;20:9-12.

The absenteeism rate for Australia Post has remained between 2.8 and 3.0% for the last six weeks, except for the third week in July when it was lower (Figure 4). Over the same period, the consultation rate for influenza-like illness increased in New South Wales and the Northern Territory, but the ASPREN reports fluctuated between 23 and 28 per 1,000 encounters (Figure 5).

Figure 4. Australia Post absenteeism, 1996, by week



As many laboratory reports of influenza A were received in the last fortnight as had been received for the year to date. While the number of reports by week of specimen collection is declining, it is still above the peak recorded last year (Figure 6). In all, 389 reports were received this fortnight, diagnosed by virus isolation (228), antigen detection (105), single high titre (45) and four-fold rise in titre (11). Of these, 54% (211 / 389) were for children under five years of age and 7% (26 / 389) were for adults over 65 years of age.

Twenty-three reports of influenza A (H3N2) were received this fortnight. Sixteen reports (70%) were for children under five years of age.

Three reports of influenza B were received this fortnight, two were diagnosed by antigen detection, the other by single high titre (Figure 7).

Australian Sentinel Practice Research Network

The Australian Sentinel Practice Research Network (ASPREN) comprises 99 sentinel general practitioners from throughout the country. A total of approximately 9,000 consultations are recorded each week for 12 conditions. Of these, CDI reports the consultation rate for influenza, rubella, measles, pertussis and gastroenteritis. For further information including case definitions see CDI 1996;20:98-99.

Data for weeks 30 and 31 ending 28 July and 4 August respectively are included in this issue of *CDI* (Table 6). The consultation rate for gastroenteritis in the most recent three reporting weeks is 30% higher than the rate seen in June and early July. The consultation rate for chickenpox has remained level over the last two months. The rates of reporting of rubella, measles and pertussis continue at low levels.

Figure 5. Sentinal general practitioner influenza-like illness consultation reports per 1,000 encounters, 1996, by week

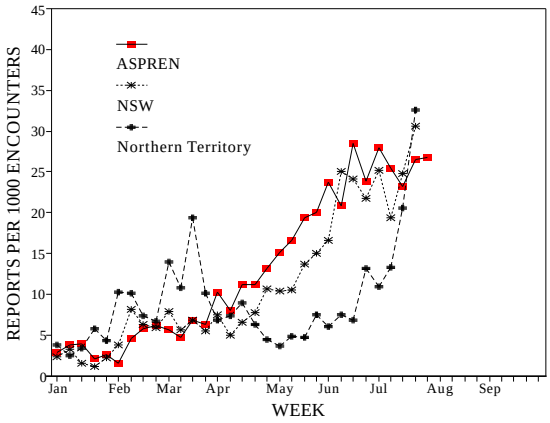


Figure 6. Influenza A laboratory reports, 1995 and 1996, by week of specimen collection

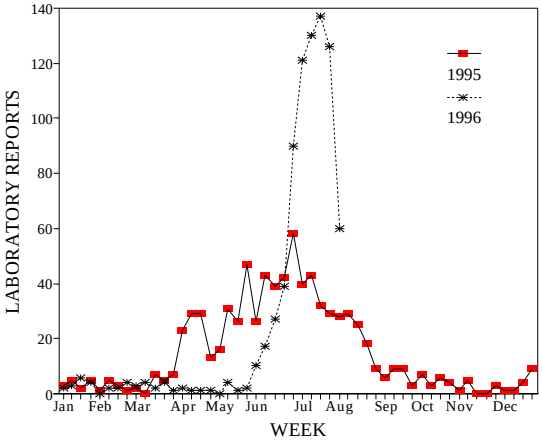


Figure 7. Influenza B laboratory reports, 1996, by method of diagnosis and week of specimen collection

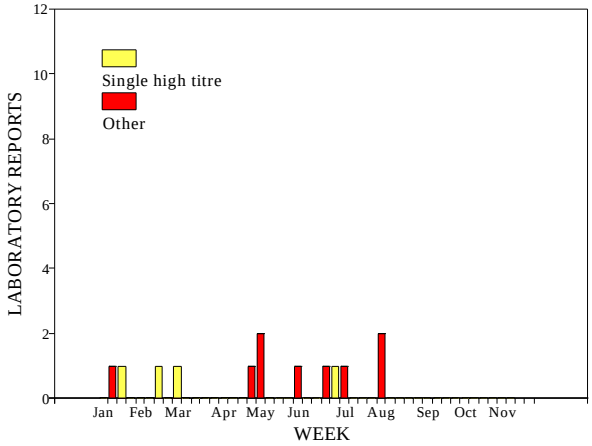


Table 6. Australian Sentinel Practice Research Network reports, weeks 30 and 31, 1996

Condition	Week 30, to 28 July 1996		Week 31, to 4 August 1996	
	Reports	Rate per 1,000 encounters	Reports	Rate per 1,000 encounters
Influenza	235	26.5	230	26.7
Rubella	3	0.3	4	0.5
Measles	1	0.1	1	0.1
Chickenpox	9	1.0	17	2.0
Pertussis	5	0.6	4	0.5
Gastroenteritis	116	13.1	125	14.5

Australian Encephalitis: Sentinel Chicken Surveillance Programme

AK Broom[1], *JS Mackenzie*[2], *L Melville*[3], *DW Smith*[4] *and PI Whelan*[5]

The Sentinel Chicken Surveillance Programme is coordinated by the Arbovirus Research Laboratory in the Department of Microbiology at the University of Western Australia. The Programme provides an early warning of increased flavivirus activity by monitoring flavivirus seroconversions in chickens in sentinel flocks in Western Australia, the Northern Territory, Victoria and Queensland. Information on seroconversions from this scheme is published every two months.

1. Department of Microbiology, The University of Western Australia
2. Department of Microbiology, The University of Queensland
3. Berrimah Agricultural Research Centre, Darwin, NT
4. PathCentre, Perth
5. Medical Entomology Branch, Department of Health and Community Services, Darwin, NT.

Sentinel chicken serology was carried out for 19 of the 22 flocks in Western Australia in May and June 1996. There were no seroconversions during this period.

Four flocks of sentinel chickens from the Northern Territory were also tested in May and June. During this period there were two seroconversions to Murray Valley encephalitis in May in the flock from Coastal Plains Research Station near Darwin. There were no seroconversions to flaviviruses in June.

Correction to March-April 1996 data

In the Northern Territory chicken flocks there were no seroconversions in March 1996 and two from Coastal Plains Research Station in April 1996. One chicken seroconverted to a flavivirus (probably Kunjin) and one to Murray Valley encephalitis virus.

LabDOSS

LabDOSS is a passive surveillance scheme that reports on significant bacterial and fungal isolates from normally sterile sites. Twenty laboratories currently forward reports of sterile site isolates to the Department of Health and Family Services. LabDOSS is published in alternate issues of CDI. Data from the LabDOSS scheme should be interpreted with caution. There is a potential for geographical, testing and referral pattern biases. In addition, risk factors and clinical information are not consistently provided by laboratories. For further information, see CDI 1996;20:9-10.

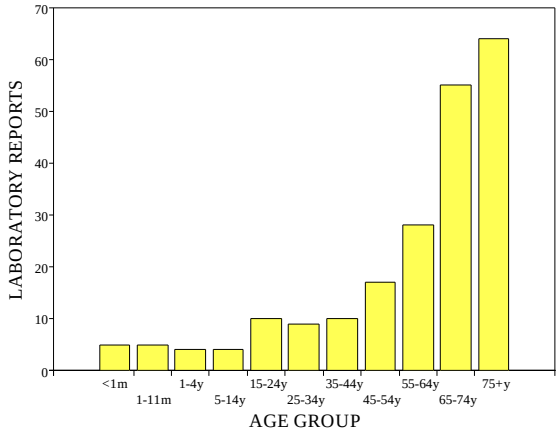
Data for this four weekly period have been provided by 6 laboratories. There were 219 reports of significant sepsis:
New South Wales: Prince of Wales Hospital 32; Royal North Shore Hospital 40.
Tasmania: Royal Hobart Hospital 20.
Queensland: Sullivan and Nicholaides and Partners 52.

Table 7. LabDOSS reports of blood isolates, by organism and clinical information

Organism	Clinical information						Risk factors					Total[1]
	Bone/Joint	Lower respiratory	Endocarditis	Gastrointestinal	Urinary tract	Skin	Surgery	Immunosuppressed	IV line	Hospital acquired	Neonatal	
Acinetobacter species		1						1		4	1	5
Enterobacter faecalis					2			2				6
Escherichia coli		3		3	8		4	7		5		29
Klebsiella oxytoca				1				1		1		5
Klebsiella pneumoniae				1	2				2	3		9
Proteus mirabilis					4			3		2		5
Pseudomonas aeruginosa	1				1		1	3		2		6
Staphylococcus aureus	1	3	1		2	12	5	7	6	15	1	30[2]
Staphylococcus coagulase negative				1	1	1	3	7	5	7	2	30[3]
Streptococcus pneumoniae		10					1	1				15
Streptococcus species				2		1		1	1			7

1. Only organisms with 5 or more reports are included in this table.
2. MRSA 7.
3. Includes *Staphylococcus epidermidis.*

Figure 8. LabDOSS reports of blood isolates, by age group



Western Australia: Sir Charles Gairdner Hospital 29.
South Australia: Institute of Medical and Veterinary Science 46.

Blood isolates
Organisms reported 5 or more times from blood are detailed in Table 7. Other blood isolates not included in Table 8 were:
Gram-positive: 1 *Bacillus cereus*, 1 *Corynebacterium* species, 2 *Enterococcus faecium*, 1 *Enterococcus* species, 1 *Listeria monocytogenes*, 2 *Streptococcus* Group B, 4 *Streptococcus* Group G, 1 *Streptococcus mitis*, 1 *Streptococcus sanguis* and 1 *Streptococcus viridans*.
Gram-negative: 1 *Aeromonas* species, 1 *Alcaligenes xylosoxidans*, 1 *Campylobacter jejuni*, 1 *Campylobacter* species, 2 *Citrobacter freundii*, 3 *Citrobacter* species, 2 *Enterobacter aerogenes*, 4 *Enterobacter cloacae*, 3 *Enterobacter* species, 2 *Haemophilus influenzae*, 2 *Klebsiella* species, 1 *Leclercia adecarboxylata*, 1 *Morganella morganii*, 1 *Pseudomonas fluorescens*, 2 *Pseudomonas* species, 1 *Salmonella paratyphi*, 1 *Salmonella* species, 3 *Salmonella typhi*, 1 *Serratia marcescens* and 1 *Xanthomonas maltophilia*.
Anaerobes: 1 *Bacteroides* species, 1 *Clostridium perfringens*, 1 *Fusobacterium* species, and 2 *Propionibacterium* species.
Fungi: 1 *Candida albicans* and 1 other *Candida* species.

There were 147 (70% of total) blood isolates reported for patients over the age of 54 years (Figure 8).

Isolates from sites other than blood
CSF: Two reports of isolates from CSF or causing meningitis were received involving *Neisseria meningitidis* and *Streptococcus pneumoniae*. Both were in children under 4 years of age.
Peritoneal dialysate: Six reports were received this period. Included was 1 *Bacteroides ovatus*, 2 *Escherichia coli*, 1 *Klebsiella pneumoniae* and 2 *Pseudomonas aeruginosa*.

LabVISE

The Virology and Serology Reporting Scheme, LabVISE, is a sentinel reporting scheme. Twenty-one laboratories contribute data on the laboratory identification of viruses and other organisms. Data are collated and published in Communicable Diseases Intelligence each fortnight. 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 CDI 1996;20:9-12.

There were 1,797 reports received in the *CDI* Virology and Serology Reporting Scheme this period (Tables 8 and 9).

In the last fortnight, 472 laboratory reports of **respiratory syncytial virus** were received. Reports have continued to increase but are below the expected peak for July (Figure 9). The July data may be incomplete so the number of reports could increase further. Diagnoses for the reports received this fortnight were by antigen detection (280), virus isolation (189), single high titre (2) and four-fold rise in titre (1). Ninety per cent of reports (427 / 472) were for children under five years of age and of these 91% (390 / 427) were under one year of age.

Reports of **parainfluenza virus type 1** have continued to decline since a peak in April (Figure10). In Australia, epidemics of parainfluenza virus type 1 occur in the autumn-winter months of alternate years. The number of reports received so far this year is similar to that for the same period in 1992 but much less than 1994. Eighteen reports were received this period, 11 were for children under five years of age.

Figure 9. Respiratory syncytial virus laboratory reports, 1991 to 1995 average and 1996, by month of specimen collection

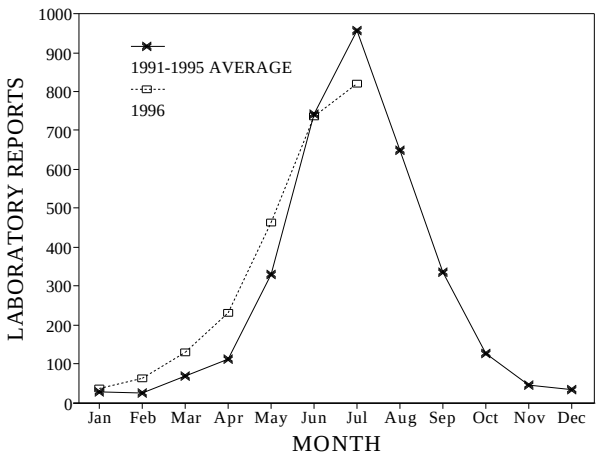


Figure 10. Parainfluenza virus type 1 laboratory reports, 1992, 1994 and 1996 by month of specimen collection

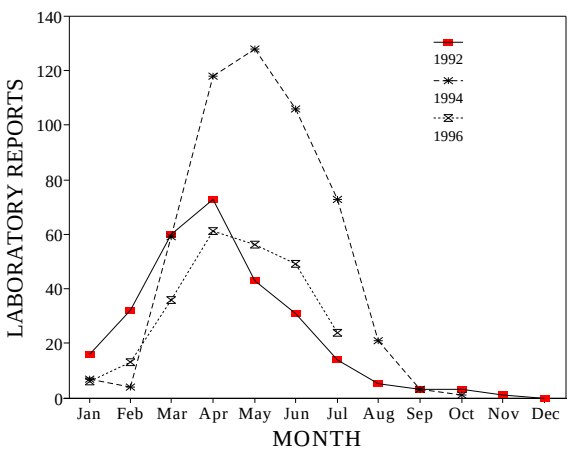


Table 8. Virology and serology laboratory reports by State or Territory[1] for the reporting period 25 July to 7 August 1996, historical data[2], and total reports for the year

	State or Territory [1]								Total this fortnight	Historical data [2]	Total reported this year
	ACT	NSW	NT	Qld	SA	Tas	Vic	WA			
MEASLES, MUMPS, RUBELLA											
Measles virus				1					1	16.3	35
Mumps virus				1					1	3.5	28
Rubella virus				30				1	31	15.5	352
HEPATITIS VIRUSES											
Hepatitis A virus		1	6	4					11	15.5	305
ARBOVIRUSES											
Ross River virus		2	5	20				4	31	11.5	3,029
Barmah Forest virus				10				1	11	4.8	165
ADENOVIRUSES											
Adenovirus type 40							1		1	.0	24
Adenovirus not typed / pending		2		29			10	15	56	41.5	890
HERPES VIRUSES											
Herpes simplex virus type 1							3		3	189.7	2,707
Herpes simplex virus type 2							1		1	207.8	2,676
Herpes simplex not typed / pending							1	2	3	22.2	290
Cytomegalovirus		7		46		3	8	8	72	70.7	1,088
Varicella-zoster virus		5		36			3	9	53	40.3	791
Epstein-Barr virus		13	2	40			4	9	68	63.2	1,302
OTHER DNA VIRUSES											
Molluscum contagiosum								1	1	.0	3
Parvovirus				9					9	3.8	103
PICORNA VIRUS FAMILY											
Rhinovirus (all types)		1		28			2	3	34	35.5	464
Enterovirus not typed / pending				25			3	8	36	40.0	578
ORTHO / PARAMYXOVIRUSES											
Influenza A virus		24		267		1	27	70	389	96.8	817
Influenza A virus H3N2				23					23	7.0	45
Influenza B virus				1				2	3	22.2	34
Parainfluenza virus type 1		2		9			4	3	18	15.5	251
Parainfluenza virus type 2				3				1	4	6.8	54
Parainfluenza virus type 3		2		8			3	10	23	29.2	354
Parainfluenza virus type 4				1					1	.0	7
Parainfluenza virus typing pending								1	1	4.3	10
Respiratory syncytial virus	1	50		223		4	87	107	472	464.5	2,690
Paramyxovirus (unspecified)							1		1	.0	12
OTHER RNA VIRUSES											
Rotavirus		31	1			5	49	19	105	144.3	808
OTHER											
Chlamydia trachomatis not typed		17	27	96		2		28	170	104.2	2,523
Chlamydia species				1					1	1.0	64
Mycoplasma pneumoniae		10		30	1		7	2	50	22.7	419
Coxiella burnetii (Q fever)		2		1				3	6	7.7	116
Rickettsia australis		1							1	.7	12
Rickettsia spp - other								1	1	.5	5
Neisseria gonorrhoeae			1					47	48	.0	104
Bordetella pertussis							18	1	19	12.2	310
Bordetella species		2		18					20	4.8	189
Legionella longbeachae								1	1	.0	12
Legionella species				3				1	4	.3	8
Leptospira hardjo				1					1	.0	14
Leptospira australis				1					1	.0	5
Leptospira species		1		7					8	.5	42
Schistosoma species								3	3	3.3	193
TOTAL	1	173	42	972	1	15	232	361	1,797	1,730.3	23,928

1. State or Territory of postcode, if reported, otherwise State or Territory of reporting laboratory.
2. The historical data are the averages of the numbers of reports in 6 previous 2 week reporting periods: the corresponding periods of the last 2 years and the periods immediately preceding and following those.

Table 9. Virology and serology laboratory reports by contributing laboratories for the reporting period 25 July to 7 August 1996

STATE OR TERRITORY	LABORATORY	REPORTS
New South Wales	Institute of Clinical Pathology & Medical Research, Westmead	43
	Royal Prince Alfred Hospital, Camperdown	45
	South West Area Pathology Service, Liverpool	60
Queensland	Queensland Medical Laboratory, West End	473
	State Health Laboratory, Brisbane	544
Tasmania	Northern Tasmanian Pathology Service, Launceston	2
	Royal Hobart Hospital, Hobart	13
Victoria	Monash Medical Centre, Melbourne	41
	Royal Children's Hospital, Melbourne	191
Western Australia	PathCentre Virology, Perth	185
	Princess Margaret Hospital, Perth	165
	Western Diagnostic Pathology	35
TOTAL		1797

Contributions covering any aspects of communicable disease are invited. Instructions to authors can be found in *CDI* 1996;20:13.

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