COMMUNICABLE DISEASES INTELLIGENCE

ISSN 0725 - 3141 VOLUME 20 NUMBER 13 24 June 1996

CONTENTS

ARTICLES Page

Editor : Helen Longbottom
Deputy Editor : Graham Andrews

COMMONWEALTH
DEPARTMENT OF
HEALTH AND FAMILY SERVICES

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

CDI is produced fortnightly by the AIDS/Communicable Diseases Branch, Department of Health and Family Services, GPO Box 9848 Canberra ACT 2601, Fax: (06) 289 7791 Telephone : (06) 289 1555

Opinions expressed in CDI are those of the authors and not necessarily those of the Department of Human Services and Health or other Communicable Diseases Network - Australia affiliates. Figures given may be subject to revision.

CDI is available on the CDI Bulletin Board System on (06) 281 6695, and via Internet on 'ftp://ftp.health.gov.au' in directory /pub/CDI and on 'http://www.health.gov.au' in '/hfs/pubs/cdi/cdihtml.htm.'

ROTAVIRUS INFECTION - THE NEED FOR A VACCINE

Ruth Bishop and Graeme Barnes, Department of Gastroenterology and Clinical Nutrition, Royal Children's Hospital, Parkville, Victoria 3052

Abstract

Primary rotavirus infection in young children can cause severe acute diarrhoea requiring hospitalisation for treatment of dehydration. Morbidity and mortality rates in children throughout the world are sufficient to justify the development of a vaccine. Current strategies are aimed at the development of a live oral attenuated rotavirus vaccine to be given in the first two to three months of life, preferably in association with oral poliomyelitis vaccine.

Introduction

Acute diarrhoea in young children is one of the major world health problems, with an average incidence of 2.5, 2.3 and 3.9 episodes per child per year in Africa, Asia and Latin America respectively. In the United States of America, there are up to 2.3 episodes per child per year[1,2,3]. It has been estimated that diarrhoea results in up to 3.7 million physician visits and 220,000 hospitalisations of children under five years old annually in the United States of America. Rotaviruses are a leading cause of diarrhoea in young children throughout the world, with the peak age for severe disease varying between six and 12 months in developing countries and 12 to 24 months in developed countries.

Rotaviruses cause 20 to 40 per cent of the episodes of diarrhoea requiring hospitalisation of children in both developed and developing countries. It is not often realised that the disease can be as severe as cholera in adults, and that rotavirus infections cause the death of approximately one million children annually in developing countries. Mortality can be controlled by early access to rehydration therapy. It is unlikely that morbidity will be controlled by improvements in hygiene, water supply or sewage disposal, as the incidence of rotavirus diarrhoea is similar in developing and developed countries, despite high standards of community hygiene in the latter.

The World Health Organization has given a high priority to the development of a rotavirus vaccine for use worldwide[4]. Recent results of field trials make it likely that at least one candidate rotavirus vaccine may be licensed for use in the near future[5]. Decisions to use rotavirus vaccines must be based on the demonstration of the importance of the disease, the likelihood that it can be controlled by vaccination and the calculated cost effectiveness of a vaccine.

Clinical symptoms, transmission and sources of rotavirus infection

Rotavirus infections are ubiquitous in the young of all mammalian and avian species. Strains are adapted to infect individual animal species, and cross-species infections appear to be rare in nature. In humans, transmission is direct from person-to-person via the faecal-oral route. Rotaviruses can contaminate, and survive for long periods in sewage and water supplies, but these do not appear to be major sources of infection within most communities[2,3].

Rotaviruses are highly infectious. Excretion of high levels of infectious particles in faeces (10^6 tissue culture infectious dose, $TCID_{50}$ / ml) (tissue culture infectious dose), stability of the virus in the environment and the low dose needed to initiate infection (1-10 $TCID_{50}$ / ml) ensures that rotavirus infections can be transmitted and sustained over long periods of time in most communities. High levels of transmission of rotavirus within families have been recorded once infection is introduced by a family member. Mild to moderate symptoms of enteric infection can develop in most contacts, including older children and adult family members.

Rotaviruses infect the mature absorptive epithelial cell lining the villi of the small intestine, producing symptoms of enteric infection after a period of 24 to 48 hours. In young children undergoing primary infection, symptoms include acute onset of vomiting and watery diarrhoea, often accompanied by fever. Symptoms can persist for several days or weeks while the gut mucosal lesions heal. In rare cases, such as in immunocompromised children, virus infection persists, causing prolonged diarrhoea. In the majority of patients, dehydration from rotavirus diarrhoea can be readily corrected by early commencement of oral rehydration therapy (ORT). Nevertheless, for a variety of reasons, some young children still die from rotavirus diarrhoea, even in Australia, the United States of America and other developed countries. Antibiotics and antimotility drugs should not be used in treatment.

Epidemiology of rotavirus infection

Rotaviruses are one of the most common infectious agents encountered throughout life. Primary infection occurs early in life, and serum surveys imply that all children in both developed and developing countries have experienced rotavirus infection by five years of age. Repeated infections are common throughout life, and have the potential to cause disease at any age. In general, most rotavirus infections are only mildly symptomatic. However severe disease associated with rotavirus infections is not uncommon and accounts for between one-third and one-half of hospitalisations for severe diarrhoea in young children worldwide

The burden of rotavirus disease across a community has seldom been assessed. A longitudinal study of rotavirus infection and gastroenteritis in families regis-

tered with one paediatric medical practice involved a middle class suburban population in the United States of America[6]. This study produced evidence that rotaviruses infected one or more members in 51% of 65 families, including 28% (35 / 126) of children and 67% (16 / 24) of adults, during 29 months of surveillance. Rotavirus infection in both children and adults was symptomatic in 75 to 80% of cases. Similar results have been recorded in longitudinal surveillance studies involving 140 children and their families over three years in Melbourne.

The natural history of rotavirus infection supports the belief that effective active immunity could be achieved using a vaccine to protect against severe disease. Longitudinal surveillance studies in Melbourne and elsewhere show that primary rotavirus infection does not protect against reinfection, but is protective against the development of clinical symptoms on reinfection[2].

Rotavirus disease in Australia

Communicable Diseases Intelligence (CDI) publishes fortnightly reports on the diagnosis of rotaviruses made by contributing sentinel laboratories throughout Australia. It is apparent that rotaviruses, together with respiratory syncytial viruses (RSV), account for many of the laboratory reports to this scheme.

The seasonal occurrence of severe rotavirus infection varies from year to year and from State to State[7]. As observed in temperate countries worldwide, rotavirus enteritis occurs predominantly in the colder months of the year. Peak months can occur as early as May or June or as late as September. In any year, the peak of infection appears to occur earlier in Perth than in the eastern States. Peak months in the Northern Territory appear unpredictable and have even included the summer months.

A recent study published in the *Medical Journal of Australia* estimates that rotavirus is a major cause of morbidity among young children in New South Wales. The annual number of admissions to hospitals is approximately 3,700 children under five years of age, at an estimated cost of 4.6 million dollars[8]. The study concluded that routine infant vaccination against rotavirus could reduce this morbidity and the resulting health care costs. A further study of the direct and indirect costs of a rotavirus outbreak in a child-care centre in New South Wales suggests that large, indirect costs can also accrue from parents' lost work time and from illness due to secondary household cases[9].

The epidemiology of rotavirus disease in Australia and elsewhere is complex[2,3]. Rotaviruses comprise a genus within the family Reoviridae and can be subdivided into at least five antigenic groups (Group A, B, C, D and E). The majority of symptomatic infections in humans and other animals are caused by Group A rotaviruses. Group A rotaviruses are subdivided further into P and G serotypes based on identification of the two outer capsid proteins VP4 and VP7 respectively, both of which stimulate production of neutralising antibody in serum and intestinal contents post-infection. Antibodies to VP4 and VP7 have been shown to be protective in animal models. Although the protective response in humans is still not precisely identified, current hypotheses implicate involvement of neutralising antibody to VP4 and VP7.

In addition to recently developed specialised assays to determine G and P type, it is possible to study genetic variation in rotaviruses excreted by individuals. Rotaviruses contain a core of genetic material of doubled stranded RNA (dsRNA) that can be extracted and subjected to gel electrophoresis to reveal 11 distinct bands, each of which represents a gene that codes for a separate viral protein. The overall pattern formed by the 11 bands is designated the electropherotype of a particular strain. There appears to be an almost infinite variety of differing electropherotypes worldwide. The number coexisting in a particular location appears to be limited, with usually one or two patterns dominant. These dominant electropherotypes may persist, but are usually replaced in any one location after 12 to 18 months. Determination of electropherotypes provides a ready means of studying genetic variation in rotavirus strains, and of tracking transmission or spread of strains.

We have conducted comprehensive studies of the serotype and electropherotype of rotavirus strains causing severe enteritis in Australian children for the past three years, and earlier studies in Melbourne since 1973[2]. Results show that serotype G1 infections are common and persistent in all States as they are worldwide and show great genetic diversity. Unpredictable epidemics of G2, G3 and G4 strains have appeared in all States. At least one unusual strain that appears to be a combination of two pre-existing strains (by reassortment of genes) caused a widespread epidemic in children in Central Australia and the Northern Territory in 1994[10]. Rotaviruses of each major serotype have also been recorded as causes of endemic nosocomial infections in obstetric hospital nurseries in Melbourne and elsewhere, frequently for long periods of time[2].

The ability of one human serotype to cross-protect against disease due to other serotypes is the subject of continuing research. Longitudinal surveillance studies of Melbourne children infected as neonates with a G3 strain has shown that they were not immune to reinfection with rotaviruses of different serotypes one to three years later[11]. However, they were protected against moderate to severe disease on reinfection. This seminal finding has been confirmed in other developed and in developing countries. It provides the basis for an approach to vaccination using live oral attenuated vaccines derived from human rotavirus strains.

Vaccine development

Although rotavirus infection can occur in breast-fed infants, there is evidence that the use of oral immune supplements (passive protection) delays, but does not prevent, later rotavirus infection. Nevertheless there are numerous clinical circumstances in which passive protection could be advantageous, for example in prolonged nosocomial outbreaks, particularly among

young children requiring special care, children undergoing major surgery, and in day-care or other nursery situations.

To date vaccine development has centred around the development of live oral vaccines capable of conferring protection against clinical disease on reinfection. At present this is most likely to be achieved by the use of a live oral attenuated rotavirus vaccine capable of stimulating a mucosal immune response (or other immune response) effective against the four major human rotavirus serotypes. Since rotavirus disease can occur under six months of age and is most common between six and 24 months of age, the vaccine should be given within the first few months of life, preferably in association with oral poliomyelitis vaccine[12].

The first candidate oral vaccine to have completed phase 1 and phase 2 field trials is a mixture of four live viruses derived from an animal rotavirus strain. This includes a G3 simian rotavirus (RRV) together with three reassortant viruses in which the gene coding for VP7 of human G1, G2 and G4 serotypes has replaced the corresponding simian gene coding for G3. This tetravalent vaccine (RRV-TV) has been tested in children in 23 centres in the United States of America and shows a relative efficacy of 57% against all rotavirus diarrhoea, and of 82% against very severe rotavirus enteritis[4]. Incorporation of this vaccine into immunisation schedules in the United States of America has been assessed as likely to be cost effective[13].

Rotavirus was first discovered in 1973 by scientists at the Royal Children's Hospital (RCH) and Melbourne University. Research since then at the Royal Children's Hospital has focussed on the epidemiology of rotavirus infection, together with the immune response observed after natural infection. This research has led to the identification of a candidate oral rotavirus vaccine by adapting to culture the strain of human rotavirus (RV3) shown to immunise newborn babies after natural infection[2,4]. Early results of phase 1 trials in adults, children aged three to four years and infants aged three months have shown no adverse symptoms after one dose of virus (6×10^5 $TCID_{50}$ / ml), and have resulted in immune responses in a proportion of recipients. It is hoped that funding for further testing of this candidate vaccine will be made available.

Alternative approaches are also being explored in Australia and elsewhere. This includes the use of virus-like particles incapable of replication but bearing the major outer capsid proteins of rotavirus serotypes, genetically engineered vaccines using avirulent *Salmonella* species or other bacteria or viruses as vectors for genes expressing rotavirus proteins[3].

It now seems within our grasp to control this common life-threatening 'cholera' of young children. Efforts to do so deserve wide support from scientists and funding bodies.

References

1. Brandt C, Glass RI. Impact of diarrheal diseases worldwide. In: Kapikian AZ, ed. Viral infections of the gastrointestinal tract, 2nd ed. New York: Marcel Dekker, 1994:1-26.
2. Bishop RF. Natural history of human rotavirus infection. In: Kapikian AZ, ed. Viral infections of the gastrointestinal tract, 2nd ed. New York: Marcel Dekker, 1994: 131-167.
3. Kapikian AZ, Chanock RM. Rotaviruses. In: Fields BN *et al*. Field's Virology, 3rd ed. Philadelphia: Lippincott-Raven, 1996:1657-1708.
4. Bishop RF. Development of candidate rotavirus vaccines. *Vaccine* II: 1993;247-254.
5. Bernstein DI, Glass RI, Rodgers G *et al.* Evaluation of rhesus rotavirus monovalent and tetravalent reassortant vaccines in US children. *JAMA* 1995; 273:1191-1196.
6. Rodriguez WJ, Kim HW, Brandt CD *et al.*Longitudinal study of rotavirus infection and gastroenteritis in families served by a paediatric medical practice: clinical and epidemiologic observations. *Pediatr Infect Dis* 1987;6:170-176.
7. Masendycz PJ, Unicomb LE, Kirkwood CD, Bishop RF. Rotavirus serotypes causing severe acute diarrhoea in young children in six Australian cities 1989 to 1992. *J Clin Microbiol* 1994; 32:2315-2317.
8. Ferson MJ. Hospitalisations for rotavirus gastroenteritis among children under 5 years of age in New South Wales. *Med J Aust* 1996; 164:273-277.
9. Ferson MJ. Direct and indirect costs of a rotavirus outbreak in child care. *Comm Dis Intell* 1995;19:4-6.
10. Palombo EA, Bugg HC, Masendycz PJ. Multiple gene rotavirus reassortants responsible for an outbreak of gastroenteritis in central and northern Australia. *J Gen Virol* 1996 (in press).
11. Bishop RS, Barnes GL, Cipriani E, Lund JS. Clinical immunity after neonatal rotavirus infection. A prospective longitudinal study in young children. *N Engl J Med* 1983;309:72-76.
12. Crawley JMS, Bishop RF, Barnes GL. Rotavirus gastroenteritis in infants aged 0-6 months in Melbourne, Australia: implications for vaccination. *J Paediatr Child Health* 1993; 29:219-221.
13. Smith JC, Haddex AC, Teutsch SM, Glass RI. Cost-effectiveness analysis of a rotavirus immunization program for the United States. *Pediatrics* 1996; 96:609-615.

THREE CASES OF DIARRHOEA FROM PENANG, MALAYSIA

David L. Paterson, Colleen P. Brenton and Jennifer M.B. Robson, Drs JJ Sullivan, NJ Nicolaides & Partners, 134 Whitmore Street, Taringa, Qld 4068

Abstract

Three persons who had travelled to Penang, Malaysia during the outbreak of cholera in May presented with diarrhoea on their return to Australia. *Vibrio cholerae*, serotype Ogawa, biotype El Tor was isolated from one case. *Salmonella* group C and *Salmonella enteritidis* were isolated from the other cases. These reports highlight the need for travellers to follow precautions regarding consumption of food and beverages when travelling overseas.

Background

Between 10 May and 31 May 1996 there was an outbreak of cholera originating in Penang, Malaysia. More than 1,000 cases were reported. We describe our experiences with Australian travellers returning from Penang with diarrhoea since that time.

Case 1

A 54 year old female from Brisbane travelled to Penang, Phuket and Kuala Lumpur between mid-April and mid-May 1996. Five days after her return she presented with watery diarrhoea (five loose bowel motions per day) and nausea. Contacts in Malaysia had informed her of the cholera outbreak, so she presented to her general practitioner for testing to exclude cholera. Faeces were collected on 21 May 1996. Microscopy revealed no leucocytes or erythrocytes. There was no growth on thiosulphate-citrate-bile salts-sucrose (TCBS) agar. *Salmonella* group C, however, was isolated. The organism was resistant to tetracycline but was susceptible to amoxycillin, cotrimoxazole and norfloxacin. She improved without any specific antimicrobial therapy.

Case 2

A 49 year old female from Brisbane travelled to Penang between early and mid-May 1996. Two days after leaving Penang, she developed watery diarrhoea, with eight motions per day. She was mildly dehydrated but did not require hospitalisation. She was treated with tinidazole but showed no improvement. Her diarrhoea lasted for one week. Faeces were collected five days after her diarrhoea commenced. Microscopy revealed no leukocytes or erythrocytes. Yellow colonies grew on TCBS agar. These grew in broth with one percent sodium chloride, and with no sodium chloride. Automated tests (Vitek GNI) were also consistent with the identification of *Vibrio cholerae.* Agglutination with O1 antiserum was achieved. The organism was identified as serotype Ogawa, biotype El Tor. The organism was susceptible to amoxycillin, tetracycline and cotrimoxazole.

The faeces of family members were screened and were negative for *V. cholerae*. The patient was treated with tetracycline, although her symptoms were almost completely settled by the time treatment was commenced.

Case 3

A 58 year old male travelled to Malaysia, including Penang, in early May 1996. He had watery diarrhoea on his return. Empiric therapy with norfloxacin was commenced after a faeces sample was collected. Microscopy of faeces on 30 May 1996 showed 2+ leukocytes but no red cells. *Giardia intestinalis* was identified on microscopic examination of a wet preparation of the faeces. *Salmonella enteritidis* was grown from his faeces.

Discussion

It is not surprising that an Australian tourist has been involved in the outbreak of cholera occurring in Malaysia. The outbreak was significant, with more than 1,000 cases confirmed since the first people were hospitalised on 10 May[1]. Although cases have been reported from Kuala Lumpur and other states of Malaysia, all cases shared a common history of having visited Penang. All isolates so far identified have been *Vibrio cholerae* biotype El Tor, serotype Ogawa.

The outbreak of cholera and the two cases of other gastrointestinal diseases acquired in Penang have at least four implications for Australian public health physicians and microbiologists:

1. The diversity of pathogens identified in the cases described above emphasises the need for pretravel education concerning prevention of food- and water-borne illnesses. The cholera outbreak reinforces the need for travellers to avoid ice in their drinks, as this may have been the source of this outbreak. It is also advisable to boil all water for five minutes prior to drinking. Chemical disinfection with iodine (which is more reliable than chlorine), is a suitable alternative when boiling is not feasible. Hot water from a tap may still contain pathogenic organisms and cannot be considered safe for drinking or for brushing teeth. Cold foods and salads should be avoided. These precautions will assist in preventing each of the pathogens identified in the three patients described above from being acquired.
2. Should cholera vaccine be recommended for travellers going to Penang or other parts of Malaysia in the near future? The presently available cholera vaccine is a heat-killed suspension of the Inaba and Ogawa serotypes of classical *V. cholerae*, serotype O1. It has been most extensively evaluated in Asian communities where some prior immunity would be expected. In these studies, its efficacy was 50 to 70%

and duration of protection was three to six months[2]. There are no good data on its efficacy for travellers, although in view of the lack of prior immunity, it would be expected that its efficacy would be even lower. It would appear that much greater protection would be gained by adhering strictly to precautions pertaining to water and food consumption than falsely relying on a poorly effective vaccine.

3. Does a case of cholera have public health implications? It is recommended that household contacts of an affected person should have faeces samples taken to exclude the possibility of carriage of the organism. There is no value, though, in vaccinating household members or other contacts of the patient. Chemoprophylaxis of household contacts with tetracycline is considered in situations where there is a high likelihood of secondary transmission[3]. As the incubation period of the disease is five days at the most, prophylaxis for cotravellers is probably pointless, as the incubation period will have been exceeded by the time the index case has been identified. Enteric precautions would be advisable if a traveller with cholera is hospitalised. As in the case above, however, there is a broad spectrum of diseases with many cases requiring no specific therapy.
4. Should TCBS plates be used routinely by microbiology laboratories in Australia? Our laboratory uses a vibrio enrichment broth and TCBS plates on all faeces samples received. In part, this reflects concern about missing locally acquired *Vibrio* species. Indeed, on the same day as the *V. cholerae* serotype O1 was isolated from faeces in the patient described above, another patient with no history of travel outside of Australia was found to be positive for non-O1 *V. cholerae.* However, there are considerable concerns about the cost effectiveness of using TCBS routinely[4]. Laboratories that do not incorporate the routine use of TCBS have to rely on good clinical notes regarding overseas travel or ingestion of raw seafood before considering the use of these selective plates.

Acknowledgments

John Bates, Dr Sarah Friel, Dr Brad McCall, Denise Murphy, Dr Kathryn Myers and Dr Gregory Williams are to be thanked for their help in the preparation of this report.

References

1. Overseas briefs. *Comm Dis Intell* 1996;20:263.
2. NHMRC. *The Australian immunisation procedures handbook.* 5th ed. Canberra: Australian Government Publishing Service, 1994:90.
3. Benenson AS, ed. *Control of communicable diseases manual.* Washington: American Public Health Association, 1995:140-144.
4. McLaughlin JC. *Vibrio.* In: Murray PR, ed. *Manual of clinical microbiology.* 6th ed. Washington: ASM Press, 1995:468.

CHOLERA INFORMATION FOR THE AUSTRALIAN TRAVELLER

Adapted from Health Information for International Travel and The Australian Immunisation Procedures Handbook, 5th edition.

Cases of cholera in Australia almost always occur in individuals who have been infected in Asia, Africa, the Middle East, South America and parts of Oceania.

Despite the prevalence of cholera in some tourist destinations, vaccination with the currently available cholera vaccine is not recommended due to its low efficacy and short duration of action. The careful selection of food and water while overseas is of far greater importance to the traveller.

In places where the standard of hygiene is less reliable than in Australia, travellers should be advised to take the following precautions with food and drink.

- Avoid eating cold meat, salads, raw or cold seafood (including shellfish), precooked food, unpasteurised milk and dairy products. Ice made from contaminated water is not sterilised by freezing, so avoid ice in drinks, ice-cream and flavoured ice blocks. Fruit that you have peeled yourself is usually safe.
- Boiling for a minimum of ten minutes is the most reliable method of ensuring that water of uncertain purity is made safe for drinking. Alternative methods of water purification include chemical disinfection with iodine or chlorine. Filters are not recommended as none can remove all pathogenic viruses.

In general, the following beverages are usually safe from contamination:

- tea and coffee made with freshly boiled water, and
- commercially canned or bottled carbonated beverages, beer and wine.

At all times, personal hygiene is of the utmost importance, and travellers should be advised to be scrupulous about washing their hands after using the toilet and before eating.

CHOLERA

Adapted from World Health Organization Fact Sheet N107 of March 1996

Cholera is an acute intestinal infection caused by the bacterium *Vibrio cholerae*. It has a short incubation period, from less than one day to five days, and produces an enterotoxin that causes a copious, painless, watery diarrhoea that can quickly lead to severe dehydration and death if treatment is not promptly given. Vomiting also occurs in most patients.

Most persons infected with *V. cholerae* do not become ill, although the organism is present in their faeces for 7 to 14 days. When illness does occur, more than 90% of episodes are of mild or moderate severity and are difficult to distinguish clinically from other types of acute diarrhoea. Less than ten per cent of ill persons develop typical cholera with signs of moderate or severe dehydration.

Background

The vibrio responsible for the seventh pandemic, now in progress, is known as *V. cholerae* O1, biotype El Tor. The pandemic began in 1961 when the vibrio first appeared as a cause of epidemic cholera in Celebes (Sulawesi), Indonesia. The disease then spread rapidly to other countries of eastern Asia and reached Bangladesh in 1963, India in 1964, and the USSR, Iran and Iraq in 1965-1966.

In 1970, cholera reached West Africa, which had not experienced the disease for more than 100 years. The disease quickly spread to a number of countries and eventually became endemic in most of the continent. In 1991, cholera struck Latin America, where it had also been absent for more than a century. Within the year it spread to 11 countries, and subsequently throughout the continent.

Until 1992, only *V. cholerae* serogroup O1 caused epidemic cholera. Some other serogroups could cause sporadic cases of diarrhoea, but not epidemic cholera. Late that year, large outbreaks of cholera began in India and Bangladesh that were caused by a previously unrecognised serogroup of *V. cholerae*, designated O139, synonym Bengal. Isolation of this vibrio has now been reported from ten countries in South Asia. It is still unclear whether *V. cholerae* O139 will extend to other regions, and careful epidemiological monitoring of the situation is being maintained.

Transmission

Cholera is spread by contaminated water and food. Sudden, large outbreaks are usually caused by a contaminated water supply. Only rarely is cholera transmitted by direct person-to-person contact. In highly endemic areas, it is mainly a disease of young children, although breastfed infants are rarely affected.

Marine shellfish and plankton are the main reservoirs of *V. cholerae*. The El Tor strain can also survive in fresh water for long periods. Persons with asymptomatic infections play an important role in carrying *V. cholerae* from place to place, causing epidemics to spread.

Treatment

When cholera occurs in an unprepared community, case-fatality rates may be as high as 50%, usually because there are no facilities for treatment, or because treatment is given too late. In contrast, a well organised response in a country with a well established diarrhoeal disease control program can limit the case-fatality rate to less than one per cent.

Most cases of diarrhoea caused by *V. cholerae* can be treated adequately by giving a solution of oral rehydration salts. During an epidemic, 80 to 90% of diarrhoea patients can be treated by oral rehydration alone, but patients who become severely dehydrated must be given intravenous fluids.

In severe cases, an effective antibiotic can reduce the volume and duration of diarrhoea and the period of vibrio excretion. Tetracycline is the usual antibiotic of choice, but resistance to it is increasing. Other antibiotics that are effective when *V. cholerae* are sensitive to them include cotrimoxazole, erythromycin, doxycycline, chloramphenicol and furazolidone.

Epidemic control and preventive measures

When cholera appears in a community, it is essential to ensure three things: hygienic disposal of human faeces, an adequate supply of safe drinking water, and good food hygiene. Effective food hygiene measures include cooking food thoroughly and eating it while it is still hot; preventing cooked foods from being contaminated by contact with raw foods, contaminated surfaces or flies; and avoiding raw fruits or vegetables unless they are first peeled.

Routine treatment of a community with antibiotics or mass chemoprophylaxis has no effect on the spread of cholera, nor does restricting travel and trade between countries or between different regions of a country. Setting up a cordon sanitaire at frontiers uses personnel and resources that should be devoted to effective control measures, and hampers collaboration between institutions and countries that should unite their efforts to combat cholera.

The only cholera vaccine that is widely available at present is killed vaccine administered parenterally, which confers only partial protection (50% or less) and for a limited period of time (three to six months maximum). Use of this vaccine to prevent or control cholera outbreaks is not recommended because it may give a false sense of security to vaccinated subjects and to health authorities who may then neglect more effective measures.

In 1973, the World Health Assembly deleted from the International Health Regulations the requirement for presentation of a cholera vaccination certificate. Today, no country requires proof of cholera vaccination as a condition for entry, and the International Certificate of Vaccination no longer provides a specific space for recording cholera vaccinations.

THE ROLE OF A DIAGNOSTIC REFERENCE LABORATORY IN MALARIA SURVEILLANCE

John Walker, Centre for Infectious Diseases and Microbiology, Westmead Hospital, Westmead, NSW 2145

Abstract

Australia was declared free of malaria by the World Health Organization in 1981, but the infection has been endemic here in the past. Some tropical regions of the country are still considered to be receptive to its reintroduction. Although the risk of reintroduction is small, it cannot be dismissed. About 700-800 imported cases of malaria are notified in Australia each year. Most of these are imported from neighbouring countries such as Papua New Guinea and the Solomon Islands. A person who had arrived from the Solomon Islands was believed to be the source of a small outbreak of *Plasmodium vivax* at Cape Tribulation in north Queensland in 1986. There were at least four introduced cases and, almost certainly, one indigenous infection. The clustering of cases was detected because of the existence of a national surveillance system based on notified laboratory diagnoses. Without such a system, the relationship between a number of infections diagnosed in different parts of the country would almost certainly have gone unrecognised. Apart from supporting a surveillance system, reference laboratories can also provide a system of quality assurance for routine laboratories, many of which have difficulty in diagnosing infrequently seen infections such as malaria.

Background

With the inclusion of New South Wales in 1969, malaria became notifiable in all States and Territories of Australia. At the time, a national register of cases was established and maintained by the late Professor R. H. Black at the School of Public Health and Tropical Medicine (SPHTM), University of Sydney. An important function of the register was the checking of all malaria diagnoses by the medical parasitology department of the SPHTM. Because of the impossibility of unequivocally diagnosing malaria clinically, no case could be included in the register unless it was based on a verified blood film identification. This checking also ensured that the data on malaria species in the annual reports of the register were accurate. As will be discussed later, Australian laboratories can make significant errors in malaria diagnosis (Walker, unpublished data on New South Wales register of malaria cases).

An important function of the register, which is now maintained at the Tropical Health Program, University of Queensland, is to provide the World Health Organization (WHO) with data on the number of cases of malaria occurring in Australia. As a result of an assessment based mainly on these data, WHO declared Australia to be free of endemic malaria in 1981[1]. This status has important connotations for tourism and development projects in the north, and its maintenance is in the national interest. In the past, epidemics of malaria, some with significant case mortality, were associated with mining projects in the Northern Territory and Kimberley region[2].

Malaria cases at Cape Tribulation, 1986

At least four cases of introduced malaria and one possible case of indigenous malaria were reported from Cape Tribulation in north Queensland in 1986. The cluster was detected because of the existence of a national register of cases.

The original case in this outbreak (probably case 5) would be classified as *imported*, as the infection was acquired outside Australia. The cases involving individuals infected by mosquitoes which had fed on the original case (cases 1,2,3 and 6) would be classed as *introduced*. Any cases derived by mosquito transmission from one of the introduced cases would be defined as *indigenous*.

The cases are discussed in chronological order with reference to the dates of the blood film diagnosis.

Cases 1, 2 and 3

Plasmodium vivax infections in three patients who had not travelled outside Australia were diagnosed at Cairns and Mossman in north Queensland between 6 and 9 November 1986. These patients had been living at Cape Tribulation for several months before becoming ill. Because of delays in the case details reaching the central register in Sydney, it was not immediately clear that they were related. At the time, the checking of blood films from Queensland cases was not being performed at the SPHTM.

Case 4

This 21 year old male had become ill on 17 October 1986 at Cape Tribulation in north Queensland, where he had been living for at least several months, and possibly for a number of years. No blood films were made at the time, but following travel to Sydney, a diagnosis of *Plasmodium vivax* infection was made by a private pathology laboratory on 14 November 1986. The blood films were examined at the SPHTM on the same day and the diagnosis was confirmed. As there was no

history of recent travel out of Australia, authorities in Queensland were immediately notified of a possible case of local transmission of malaria at Daintree River, near Cape Tribulation.

Case 5

This man, who arrived in Brisbane from the Solomon Islands on 1 September 1986, is thought to have been the primary source of the outbreak. He had been taking Fansidar prophylaxis when in the Solomon Islands. He visited Cape Tribulation for one week from 3 October. On the 10th he left the area and was treated in Cairns with quinine for a febrile illness, clinically diagnosed as malaria. *P. vivax* was subsequently diagnosed by blood film at Katherine in the Northern Territory on 17 November 1986.

There is uncertainty about case 4 mentioned above. His febrile illness began on 17 October 1986. If this was due to the malaria infection that was diagnosed in Sydney on 14 November, it was too early to have resulted from mosquito-borne infection originating from case 5. It is possible that the initial illness was not malaria, or that it was a relapse from an infection acquired outside Australia months or years before. If the latter possibility is correct, this individual could have been the source of the subsequent infections.

Case 6

The most significant case in this series was that of a 22 year old woman whose *P. vivax* infection was diagnosed at Gosford, New South Wales on 8 December 1986[3,4]. This patient had spent one night at Cape Tribulation on 21 October 1986 and had become ill at Cairns on 3 November. She had never been out of Australia. When the blood films were reviewed at the SPHTM, Queensland authorities were immediately notified of a definite case of introduced malaria originating in the Daintree River area. An epidemiological investigation of the region identified the most probable site of transmission. Larvae of *Anopheles farauti* were found nearby[3].

A possible indigenous case

Case 7

A further infection, which occurred after the investigation was concluded, has not been included in past discussions of this outbreak (Walker,.unpublished data, School of Public Health and Tropical Medicine, University of Sydney). This woman, who had never been out of Australia, became ill at Burnie in Tasmania on 31 December 1986 and *P. vivax* infection was diagnosed on 2 January 1987. She had arrived at Cape Tribulation in late November 1986, after the other individuals had left the area. Either she was infected by a mosquito which had survived from the first week of October to late November, or she was bitten by a mosquito which had fed on one of the other individuals before they left.

On the basis of the time of the onset of symptoms in cases 1,2,3 and 6, it is presumed that mosquitoes were infected on or about 7 October, most probably by feeding on case 5, and that these mosquitoes transmitted the infection to the other individuals on or around 18 October. One person (case 3) became ill at Cape Tribulation on 3 November and his infection was confirmed by blood film at Mossman on the 9th. If there were gametocytes in his blood, he could have been a source of infection for mosquitoes during the first week of November. Around 75% of all cases of *P. vivax* infection diagnosed in Australia have gametocytes in the peripheral blood at the time of diagnosis (Walker, unpublished data on New South Wales register of malaria cases).

It is possible, given the climate at Cape Tribulation, that a mosquito had survived from early October to late November and transmitted the infection to case 7. This is less likely, however, than a second round of transmission involving mosquitoes which had fed on one of the other infected individuals. In studies on the survival of Australian anophelines at Darwin, Russell found that only between 3% and 4.3% of *Anopheles farauti* survived long enough for sporozoite development[5]. Survival of mosquitoes in the rainforest at Cape Tribulation would probably be longer than at Darwin, but even a relatively low daily mortality rate of ten per cent would leave only 0.45% of mosquitoes alive after 50 days, the period involved in this instance.

If this infection was the result of a second round of transmission, it becomes the most recent indigenous case of malaria diagnosed on the Australian mainland. This was previously thought to have occurred at the Roper River Mission in the Northern Territory in 1962[2].

Advantages of a centralised surveillance system

Had it not been for the verification of malaria diagnoses by a single laboratory, the connection between the infections originating at Cape Tribulation would not have been made until the details of the individual cases were collated at the central register. This was often a lengthy process because of the slow return of case information sheets.

In situations where surveillance of an infection is important, and in which the diagnosis of that infection is laboratory based, it is logical to structure the system around a single laboratory or a network of laboratories. A major reason for this is the significant error rate in malaria diagnosis when performed by routine pathology laboratories in Australia (Walker, unpublished data on New South Wales register of malaria cases).

Quality assurance of malaria diagnosis

The error rate in the diagnosis of malaria species by Australian laboratories has been analysed from data collected by the New South Wales malaria register from 1989 until March 1996 (Walker, unpublished data on New South Wales register of malaria cases). Only cases having a definite suggestion of the species identity by the original diagnostic laboratory are included. Infec-

tions initially detected by the State reference laboratory are excluded. Of 1,386 cases which fit these criteria, there were 211 incorrect diagnoses, an error rate of 15%. For diagnoses of *P. vivax*, the rate of incorrect diagnosis was 10%.

For *P. falciparum* infections however, the error rate is much higher. Of 315 cases, 68 were incorrectly diagnosed, an error rate of 21.6%. The most common single error involves the identification of *P. falciparum* as *P. vivax*. This occurred 44 times. There is a highly significant difference (p=0.004) between the error rate for private pathology laboratories (32.5%) and non-private laboratories (17.8%) in the diagnosis of *P. falciparum* infections. The lower error rate for non-private laboratories is influenced by the performance of several large hospitals which diagnose numerous malaria cases. The staff of these hospitals, therefore, have more experience than most in recognition of these parasites. There is no difference between private and public laboratories in the identification of other species. Such a high error rate for *P. falciparum* is unacceptable and needs to be addressed. In five instances involving this species, the infection was missed altogether, although subsequent checking of the films by the reference laboratory revealed parasites.

On this basis alone, it is clear that relying on unconfirmed diagnoses of malaria species would provide extremely inaccurate epidemiological data, with an underestimate of the amount of *P. falciparum* and an overestimate of the amount of *P. vivax*. Even greater rates of error occur when mixed infections and other species are involved.

Climatic factors limit the region of Australia which is considered to be receptive to the reintroduction of malaria in the far north. Bryan, Foley and Sutherst discuss the possible influence of future climate change in extending the range of *Anopheles farauti* down the Queensland coast as far south as Gladstone[6]. This extension of range would include popular tourist destinations such as the Whitsunday Passage. Although the establishment of malaria on the coast of Queensland is unlikely, the experience from Cape Tribulation demonstrates that it could occur and that surveillance is necessary to prevent the reintroduction of an infection which was endemic in Cairns as recently as 1943[2].

References

1. Sleigh A, Srinivasa M, Cooper A *et al. Report of the Australian malaria register for 1991*. Tropical Health Program, The University of Queensland 1992.
2. Black RH. *Malaria in Australia.* Service publication 9, School of Public Health and Tropical Medicine, The University of Sydney. Canberra: Australian Government Publishing Service, 1972.
3. Streatfield R. Report and Recommendations: Vivax malaria, Cape Tribulation October-November, 1986. NHMRC Malaria subcommittee, 3-4 November 1987.
4. Musgrave IA. Malaria outbreak in Queensland. *Med J Aust* 1987;146:278.
5. Russell RC. Seasonal abundance, longevity and population age composition of potential malaria vectors in northern and southern Australia. *Aust J Zool* 1987; 35:289-306.
6. Bryan JH, Foley DH, Sutherst RW. Malaria transmission and climate change in Australia. *Med J Aust* 1996; 164:345-347.

EDITORIAL: MALARIA SURVEILLANCE IN AUSTRALIA

Graeme Oliver, Department of Health and Family Services, GPO Box 9848, Canberra ACT 2601

Every year 700-800 cases of imported malaria are notified in Australia[1,2]. Many of these infections are acquired overseas by Australian residents travelling in malaria endemic areas; other cases occur in migrants and visitors from countries with endemic malaria. Traditional movement between Papua New Guinea, the islands of the Torres Strait and northern Australia is an important factor in malaria importation[3].

Although in recent years the highest population rates for the notification of malaria have been recorded for Far North Queensland and other parts of northern Australia[1,2], cases are diagnosed in all parts of the country.

Disproportionate numbers of cases occur in males, with a male:female ratio of 2.4:1 over the last five years. Especially predominant are males in the age range 15-29 years. It is not clear whether this is related to greater numbers of young male travellers from endemic areas (including residents returning from abroad and students from countries to our near north coming to educational institutions in northern Queensland and the Northern Territory) or to behavioural factors affecting exposure and prophylaxis.

Conditions suitable to maintain endemic malaria continue to exist in parts of Australia north of latitude 19°S. Consequently, effective surveillance measures must be maintained, especially in these 'malaria-receptive' parts of the country, to rapidly detect and appropriately manage cases of malaria. In northern Queensland and the Northern Territory, procedures are in place to ensure timely notification of cases and thorough follow-up, accompanied by appropriate measures to control possible vectors.

In addition to facilitating effective control in 'malaria-receptive' areas, surveillance also serves a number of other important functions in Australia as a whole. International obligations require regular reporting to the World Health Organization of the number of cases of

malaria detected, how many are locally acquired or imported, and which *Plasmodium* species are involved. Surveillance information, including species identification and drug susceptibility, informs policy on prophylaxis and management schedules and underpins health advice to travellers.

A meeting of the Australian Malaria Register Network in October 1995 examined current surveillance practices and other aspects of malaria control, including vector surveillance and vector control, active case finding in high-risk groups, and supervised therapy of cases. Recommendations were made that States and Territories introduce a national minimum data-set, to be collated and reported upon fortnightly, and that extra information be gathered to enhance regional management at State and Territory level. Implementation of these recommendations is anticipated in the near future.

References

1. Annual Report of the National Notifiable Diseases Surveillance System, 1994. *Comm Dis Intell* 1995;19(22): 542-574.
2. Longbottom, H. Epidemiology of Malaria in Australia 1991-1995. *Comm Dis Intell* 1996;20(4):84-87.
3. Report of the Australian Malaria Register for 1991. *Comm Dis Intell* 1993;17(7):134-142.

OVERSEAS BRIEFS

In the past fortnight the following information has been provided by the World Health Organization (WHO).

Dengue/Dengue haemorrhagic fever

Malaysia: The dengue season has started in Malaysia, with 2,450 cases reported throughout the country by 1 June 1996, according to information from the WHO Collaborating Centre for Dengue / Dengue Haemorrhagic Fever in Kuala Lumpur. The States most affected are the Federal Territory, Selangor, Perak, Johor and Pahang. The present season is expected to continue for the next few months, reaching a peak in July-August. Active vector control measures are in place in all States.

Indonesia: Over the last five months there have been 12,093 cases of dengue / dengue haemorrhagic fever and 260 deaths in 23 of the 27 provinces of Indonesia. The highest number of cases occurred in East Java (3,346 cases, 62 deaths). Dengue usually occurs in densely populated areas. A campaign to educate the public on keeping the environment clean and free of stagnant water, the breeding place for mosquitoes, has been stepped up.

Lassa fever update

Sierra Leone: In collaboration with the Ministry of Health and Sanitation and the Lassa Fever Research Project, the WHO support team, with a specialist from Centres for Disease Control (CDC) has assessed the extent and severity of the outbreak after making field trips to Kailahun and Kenema Districts during 26 and 27 May. The conclusion of the WHO support mission is that a major epidemic of Lassa fever has occurred in Kailahun and Kenema Districts and is continuing as unusually large numbers of severely ill patients are still being admitted to hospital. A large number of deaths are still occurring. A total of 167 cases and 60 deaths have been recorded during the period 1 January to 28 May 1996.

Cholera

Philippines: An outbreak has been reported in Baganga, Davao Oriental Province, where 214 cases and four deaths occurred during the period 15 to 30 May. Measures to control the outbreak at provincial and local levels are being undertaken.

Other countries reporting cholera in the past fortnight are Cameroon, Kenya, Liberia and Niger.

Malaysia: The outbreak of cholera in Penang has been contained and no new cases have been reported in this area since 31 May. There have, however, been some cases reported in the neighbouring State of Kedah. No deaths have occurred during this outbreak and all preventive and control measures according to the WHO guidelines are being carried out.

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 legislations. De-identified core unit data are supplied fortnightly for collation, analysis and dissemination. For further information see CDI 1996;20:9-10.

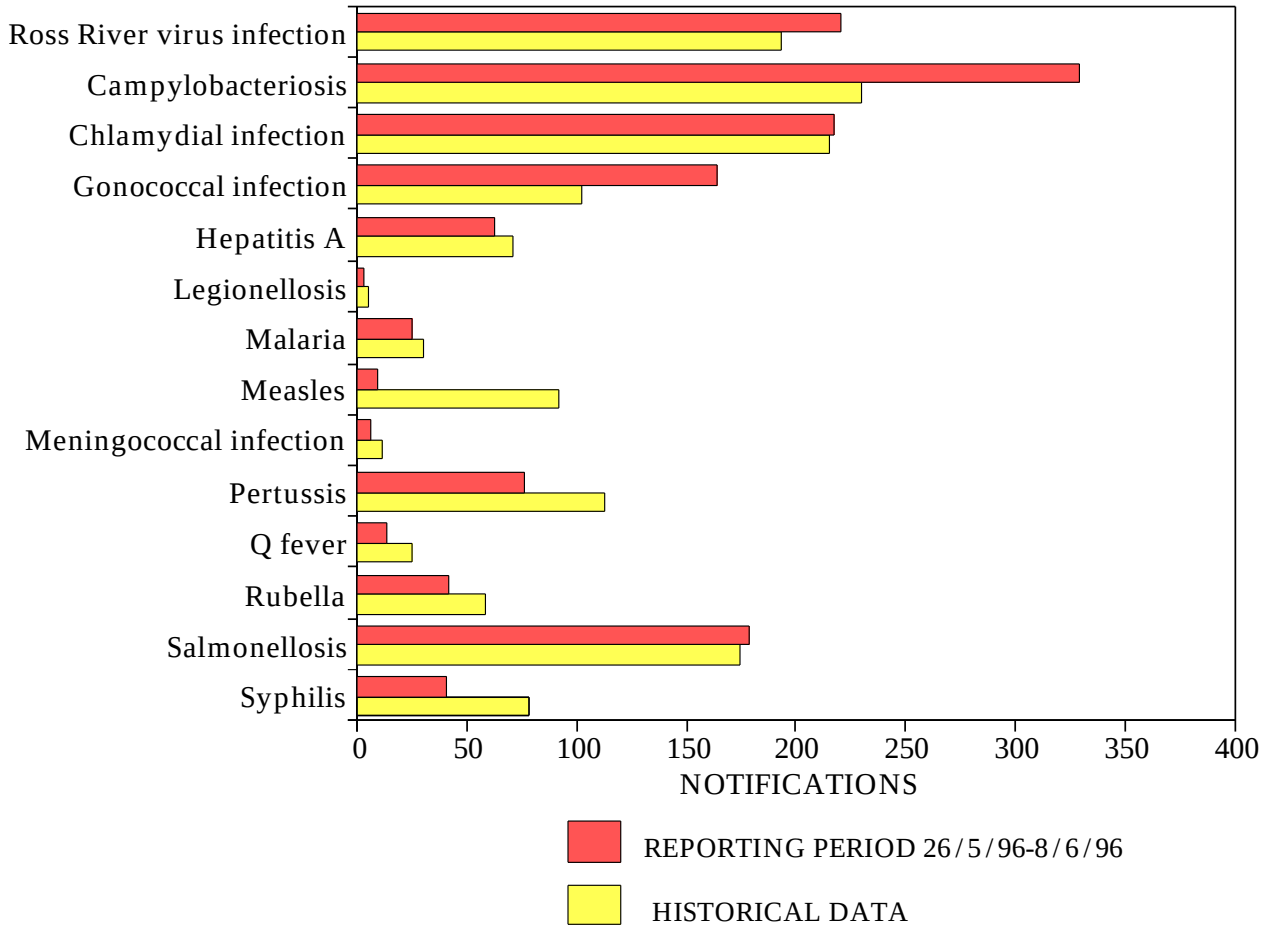
Notifications, 26 May to 8 June 1996

There were 1,687 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 previous years (Figure 1). As no reports were received from Victoria for the current period, Victorian data have been excluded from Figure 1.

The recent epidemic of **Ross River virus infection** has continued to abate, although some activity is continuing in the coastal regions of Queensland and northern New South Wales. There were 220 notifications received for the current period, the highest numbers of reports being received from the Queensland Statistical Divisions of Brisbane (61 cases) and Northern (42). The latter region has reported the highest rate.

Small numbers of cases of ***Haemophilus influenzae* type b** continue to be notified. Three reports were received for the current period. The total for this year is now 26, compared to 37 for the first five months of 1995, and 77 for the same period in 1994. Two of the current cases were aged less than one year; the other was one year old.

Figure 1. Selected National Notifiable Diseases Surveillance System reports, and historical data[1,2]



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.
2. No data from Victoria are included in this figure.

Figure 2. Meningococcal infection notifications 1993 to 1996, by month of onset

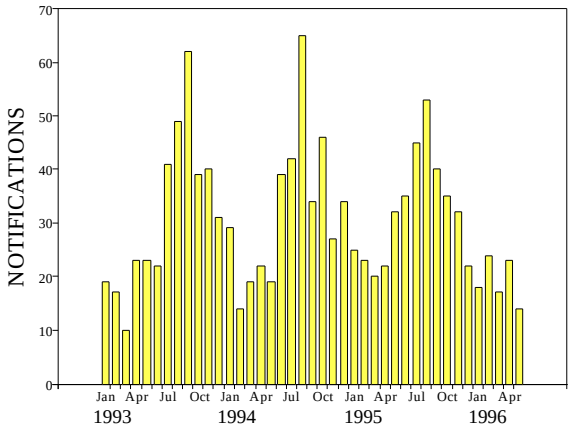
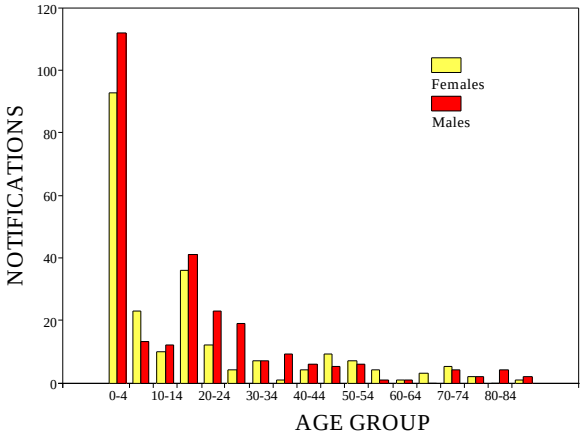


Figure 3. Meningococcal infection notifications 1995 and 1996, by age group and sex



Six cases of **meningococcal infection** were notified during the period from five Statistical Divisions in New South Wales and Queensland. A consistent seasonal distribution of cases has been observed over the last three years (Figure 2), with higher numbers of cases in the winter and spring months. Of 497 cases reported since January 1995, 205 (41%) have been in children under five years of age (Figure 3). Significant numbers of cases were also seen in the age group 15-19 years. The male:female ratio was 1.2:1. During the period, more than 75% of cases (387) were reported from three States: New South Wales (153 cases), Queensland (128) and Victoria (106). However, the highest rate is for the Northern Territory (11 cases, notification rate 4.4 per 100,000 per annum). The rate for Australia as a whole was 1.92 per 100,000 per annum.

There were 76 cases of **pertussis** reported during the period. Notifications this year have remained at a slightly lower level than in recent years (Table 1, Figure 1). Five cases were reported in children under one year, and a further six cases in children under five years. However, age was not recorded for 30 cases (40%).

There were 42 reports of **rubella** received for the period. Over the past three and a half years, a consistent seasonal pattern has been observed (Figure 4), without evidence of any marked change in the overall numbers of cases. The age distribution of cases reported since January 1995 reveals a large proportion of cases in young males between 10 and 29 years of age (Figure 5). The proportion of cases in males in this age group has remained in the range 44% to 52% for each of the years since 1991.

Figure 4. Rubella notifications 1993 to 1996, by month of onset

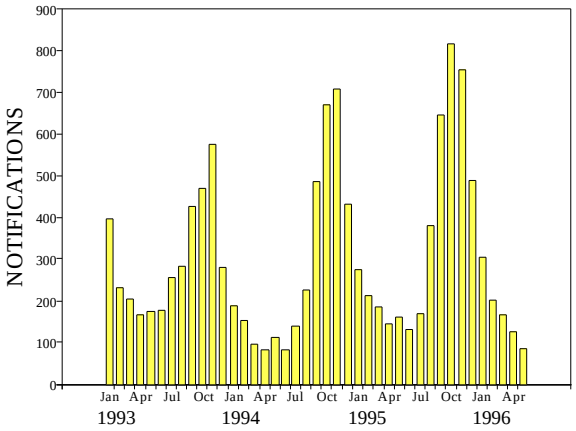


Figure 5. Rubella notifications 1995 and 1996, by age group and sex

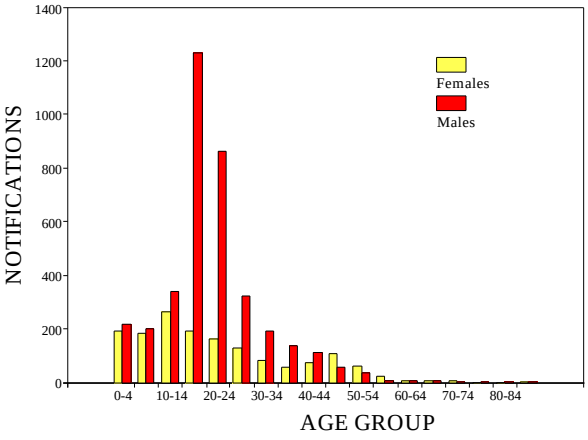


Table 1. Notifications of diseases preventable by vaccines recommended by the NHMRC for routine childhood immunisation, received by State and Territory health authorities in the period 26 May to 8 June 1996

DISEASE	ACT	NSW	NT	Qld	SA	Tas	WA	TOTALS FOR AUSTRALIA[1]			
								This period 1996	This period 1995	Year to date 1996	Year to date 1995
Diphtheria	0	0	0	0	0	0	0	0	0	0	0
Haemophilus influenzae B infection	0	0	0	3	0	0	0	3	2	26	37
Measles	1	4	0	3	1	0	1	10	59	200	804
Mumps	0	0	0	NN	1	0	0	1	9	50	61
Pertussis	0	21	0	23	30	0	2	76	159	1323	1941
Poliomyelitis	0	0	0	0	0	0	0	0	0	0	0
Rubella	1	5	0	31	2	1	2	42	73	1210	1108
Tetanus	0	0	0	0	0	0	0	0	0	1	2

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

NN Not Notifiable.

Table 2. Notifications of other diseases[1] received by State and Territory health authorities in the period 26 May to 8 June 1996

DISEASE	ACT	NSW	NT	Qld	SA	Tas	WA	TOTALS FOR AUSTRALIA[2]			
								This period 1996	This period 1995	Year to date 1996	Year to date 1995
Arbovirus Infection (NEC)[3,4]	0	0	0	0	0	0	1	1	21	104	323
Barmah Forest virus infection	0	8	-	38	0	0	-	46	38	522	228
Ross River virus infection	0	32	2	171	0	-	15	220	235	6823	1823
Dengue	0	0	0	0	0	-	0	0	0	22	13
Campylobacteriosis[5]	10	-	5	122	109	20	63	329	375	4827	4581
Chlamydial infection (NEC)[6]	10	NN	40	115	0	12	40	217	263	3101	2839
Donovanosis	0	NN	1	1	NN	0	0	2	2	25	39
Gonococcal infection[7]	1	17	55	55	0	1	35	164	138	1649	1347
Hepatitis A	3	34	3	22	0	0	0	62	77	1082	780
Hepatitis B incident	0	0	0	3	0	2	0	5	13	100	160
Hepatitis B unspecified	2	0	0	29	0	1	13	45	80	650	784
Hepatitis C incident	1	1	0	-	0	-	-	2	3	11	42
Hepatitis C unspecified	9	NN	0	80	NN	21	26	136	375	3463	3653
Hepatitis (NEC)	0	0	0	0	0	0	NN	0	3	10	14
Legionellosis	0	1	0	1	1	0	0	3	10	80	100
Leptospirosis	0	2	0	3	1	0	0	6	3	107	55
Listeriosis	0	1	0	0	0	0	0	1	2	24	36
Malaria	0	4	0	18	3	0	0	25	53	343	291
Meningococcal infection	0	2	0	4	0	0	0	6	24	106	146
Ornithosis	0	NN	0	2	0	0	1	3	1	39	64
Q fever	0	6	0	7	1	0	0	14	13	198	191
Salmonellosis (NEC)	0	28	9	108	17	2	15	179	233	3039	3486
Shigellosis[5]	0	-	3	13	2	0	4	22	33	296	392
Syphilis	1	22	6	7	0	1	4	41	92	651	884
Tuberculosis	0	6	1	5	1	1	3	17	28	458	495
Typhoid[8]	0	0	0	0	0	0	0	0	2	42	36
Yersiniosis (NEC)[5]	0	-	0	7	0	0	0	7	13	115	174

1. For HIV and AIDS, see *CDI* 1996;20:289. 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. WA, NT and Vic: 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.

Table 3. Notifications of rare[1] diseases received by State and Territory health authorities in the period 26 May to 8 June 1996

DISEASES	Total this period	Reporting States or Territories	Year to date 1996
Botulism	0		0
Brucellosis	0		13
Chancroid	0		1
Cholera	0		3
Hydatid infection	1	Qld	19
Leprosy	1	NT	7
Lymphogranuloma venereum	0		0
Plague	0		0
Rabies	0		0
Yellow fever	0		0
Other viral haemorrhagic fevers	0		0

1. Fewer than 60 cases of each of these diseases were notified each year during the period 1988 to 1994.

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 20 1996, pages 9-12.

The consultation rate for influenza-like illness recorded by ASPREN has continued to rise this fortnight, while that for the New South Wales scheme fell in early June (Figure 6). The absenteeism rate for a national employer remained stable (Figure 7).

The number of laboratory reports of influenza A diagnosed by a method other than single high titre (direct

Figure 6. Sentinel general practitioner influenza consultation rates per 1,000 encounters, 1996, by week

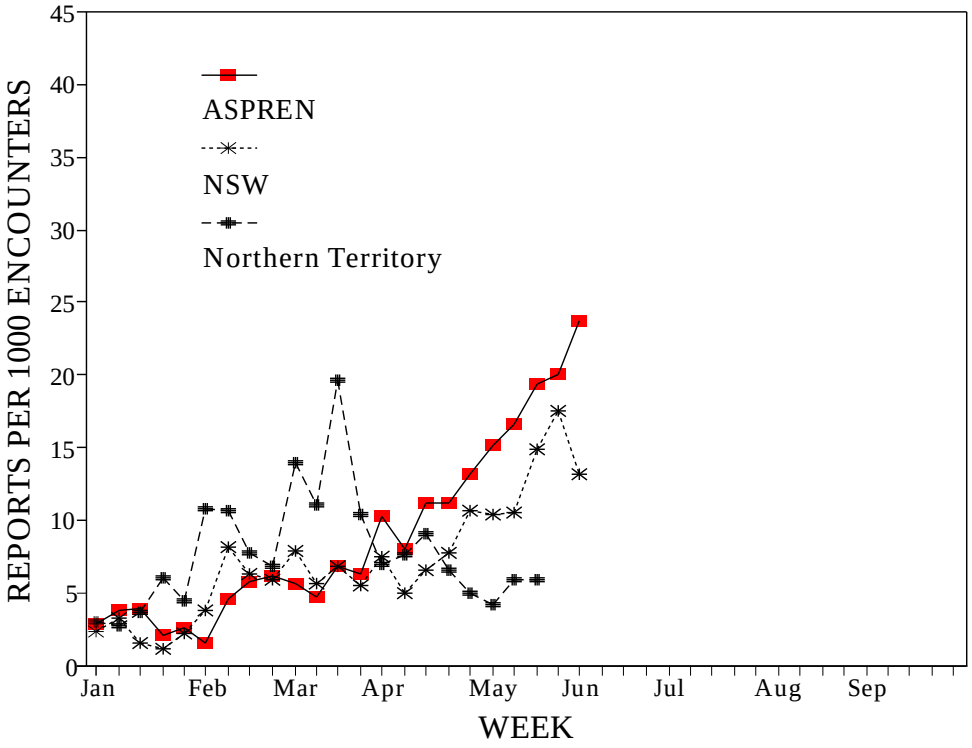
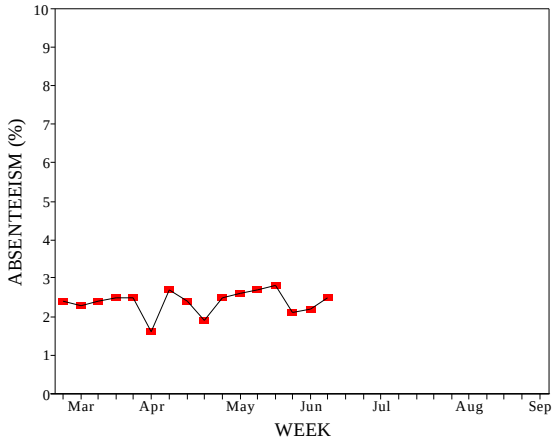


Figure 7. Australia Post absenteeism, 1996, by week



detection, virus isolation or fourfold rise in titre) rose in early June. However the total number of reports received for any week remained at similar levels to those recorded earlier in the year (Figure 8). A total of 55 reports of influenza A have been received so far this year. Of these 29% were for children under the age of 5 years and 13% were for adults over the age of 65 years.

Few reports of influenza B have been received so far this year (Figure 9).

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, chickenpox, pertussis and gastroenteritis. For further information including case definitions see CDI 1996;20:98-99.

Data for weeks 21, 22 and 23 ending 26 May and 2 and 9 June respectively are included in this issue of *CDI* (Table 4). The rate of reporting of influenza-like illness has risen each week, reaching 23.8 per 1,000 consultations for week 23. The rates of reporting of rubella, measles, chickenpox and pertussis continue at low levels. The reporting rate for gastroenteritis has also been low in recent weeks. This is consistent with previous observations for this time of the year (Figure 10).

Figure 8. Influenza A laboratory reports, 1996, by method of diagnosis and week of specimen collection

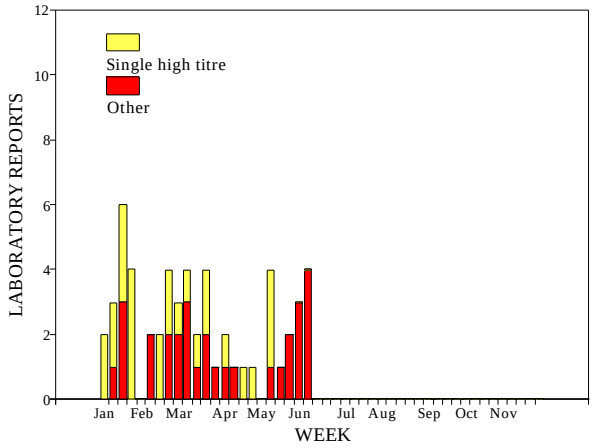


Figure 9. Influenza B laboratory reports, 1996, by method of diagnosis and week of specimen collection

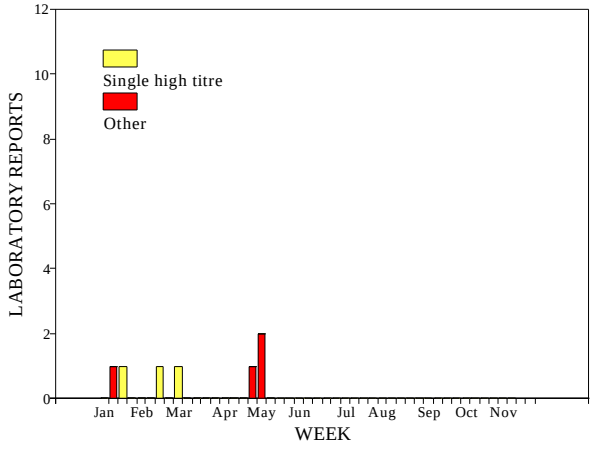


Figure 10. Consultation rates for gastroenteritis, 1994 to 1996, by month

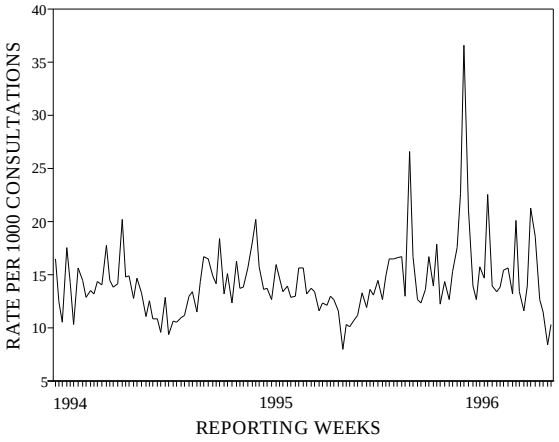


Table 4. Australian Sentinel Practice Research Network reports, weeks 21 to 23, 1996

Condition	Week 21, to 26 May 1996		Week 22, to 2 June 1996		Week 23, to 9 June 1996	
	Reports	Rate per 1000 encounters	Reports	Rate per 1000 encounters	Reports	Rate per 1000 encounters
Influenza	150	19.3	180	20.1	221	23.8
Rubella	0	0	2	0.2	3	0.3
Measles	0	0	1	0.1	0	0
Chickenpox	11	1.4	16	1.8	8	0.9
Pertussis	6	0.8	0	0	0	0
Gastroenteritis	89	11.5	75	8.4	96	10.3

Serious Adverse Events Following Vaccination Surveillance Scheme

The Serious Adverse Events Following Vaccination Surveillance Scheme is a national surveillance scheme which monitors the serious adverse events that occur rarely following vaccination. More details of the scheme were published in CDI 1995;19: 273-274.

Acceptance of a report does not imply a causal relationship between administration of the vaccine and the medical outcome, or that the report has been verified as to the accuracy of its contents.

It is estimated that 250,000 doses of vaccines are administered every month to Australian children under the age of six years.

Results for the reporting period 12 May to 8 June 1996

There were 11 reports of serious adverse events following vaccination for this reporting period. Reports were received from the Australian Capital Territory (4), New South Wales (4), The Northern Territory (1) and Queensland (2).

Of the 11 reports three were cases of persistent screaming, one of a hypotonic/hyporesponsive episode, one of convulsions, two of fever and four were other events temporally associated with vaccination (Table 5). The 'other' events included two episodes of rash, one of loss of consciousness and one of vomiting and pallor.

Events related to DTP vaccine alone or DTP in combination with other vaccines were associated with the first (3), second (2), fourth (1) and fifth (1) doses (one dose number was not reported). Three children were hospitalised. All children had recovered at the time the initial report was submitted.

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;20:9-12.

Data for this four weekly period have been provided by 8 laboratories. There were 368 reports of significant sepsis:

New South Wales: Prince of Wales Hospital 43.
Tasmania: Royal Hobart Hospital 27; Northern Tasmania Pathology Service 13.
Queensland: Sullivan and Nicholaides Partners 70; Toowoomba Pathology Laboratory 65.
Northern Territory: Alice Springs Hospital 75.
Western Australia: Princess Margaret Hospital for Children 29; Sir Charles Gairdner Hospital 46.

Table 5. Adverse events following vaccination for the period 12 May to 8 June 1996

Event	Vaccines					Reporting States or Territories	Total reports for this period
	DTP	DTP / OPV	DTP / OPV / Hib	CDT	Hep B		
Persistent screaming			3			ACT	3
Hypotonic/hyporesponsive episode					1	NSW	1
Temperature of 40.5° C or more	1		1			NSW, Qld	2
Convulsions				1		Qld	1
Other	1	1	1		1	ACT, NSW, NT	4
Total	2	1	5	1	2		11

Table 6. 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	
Enterococcus faecalis			1				1		1	1	6	6
Staphylococcus aureus	2	2		1	3	3	3	9	4	4	2	53[2]
Staphylococcus coagulase negative	1	4					3	16	4	2	5	62[3]
Streptococcus Group A						1						6
Streptococcus Group B		1				2		1			1	8
Streptococcus pneumoniae	1	8		3							1	20
Streptococcus viridans			1					1		1		5
Acinetobacter species							1	5	1	1		9
Enterobacter aerogenes									1	2		5
Enterobacter cloacae		1		1					1			7
Escherichia coli		4		3	13		3	3	1	3	2	48
Klebsiella pneumoniae		1			1			1				11
Klebsiella oxytoca		2						1	1	2		8
Pseudomonas aeruginosa								5	1	1		14
Salmonella species		1		1								7

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

Table 7. LabDOSS reports of meningitis and/or CSF isolates, by organism and age group

	1-11 months	1-4 years	25-34 years	35-44 years	55-64 years	75+ years	Total
Cryptococcus neoformans			1		1		2
Enterococcus faecalis						1	1
Haemophilus influenzae		1					1
Klebsiella pneumoniae				1			1
Neisseria meningitidis	1	1					2
Pseudomonas aeruginosa				2			2
Serratia marcescens					2		2
Serratia species	1						1
Staphylococcus epidermidis		1					1
Streptococcus pneumoniae	4					2	6
Streptococcus viridans					1		1
Streptococcus species						2	2

Blood isolates

Organisms reported five or more times from blood are detailed in Table 6. Other blood isolates not included in Table 6 were:

Gram-positive: 2 *Bacillus cereus*, 1 *Bacillus* species, 1 *Corynebacterium* species, 2 *Enterococcus faecium*, 1 *Enterococcus* species, 1 *Staphylococcus saprophyticus*, 1 *Stomatococcus mucilaginosis*, 1 *Streptococcus* Group G, 1 *Streptococcus* 'milleri' 1 *Streptococcus sanguis and* 4 *Streptococcus* species.

Gram-negative: 1 *Campylobacter jejuni*, 2 *Citrobacter freundii*, 1 *Citrobacter* species, 2 *Enterobacter* species, 1 *Gemella* species, 1 *Haemophilus influenzae*, 4 *Proteus mirabilis*, 1 *Salmonella paratyphi*, 3 *Serratia marcescens* and 2 *Xanthomonas maltophilia.*

Anaerobes: 2 *Bacteroides fragilis*, 1 *Bacteroides* species, 1 *Clostridium perfringens*, 1 *Peptostreptococcus* species, and 1 *Propionibacterium acnes.*

Fungi: 4 *Candida albicans* and 4 *Candida* species.

There were 205 (64% of total) blood isolates reported for patients over the age of 44 years (Figure 11).

Isolates from sites other than blood

Organisms reported in association with meningitis or isolated from CSF are detailed in Table 7.

Joint fluid: Three reports were received this period all involving *Staphylococcus aureus.*

Peritoneal dialysate: Fourteen reports were received this period. Included were 3 *Escherichia coli,* 1 *Klebsiella oxytoca,* 1 *Klebsiella* species, 3 *Proteus mirabilis,* 2 *Staphylococcus aureus,* 2 *Staphylococcus epidermidis,* 1 *Streptococcus* Group A and 1 *Xathomonas maltophilia.*

Pleural fluid

Two reports were received, both involving *Staphylococcus aureus* (1 MRSA).

Other: Four reports were received. Included was 1 *Clostridium* species, 1 *Escherichia coli,* 1 *Staphylococcus aureus* and 1 *Staphylococcus* coagulase negative.

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.

Following the evaluation of the *Communicable Disease Intelligence* Virus and Serology Laboratory Reporting Scheme, LabVISE, a number of changes have been made. Data on hepatitis B and C and herpes simplex virus will no longer be collected. A number of other agents have also been excluded from the scheme. In addition the table which listed agents by clinical diagnosis will be omitted from the fortnightly report in future. These modifications should reduce the number of reports recorded by the scheme while maintaining the collection of quality data.

There were 1,048 reports received in the *CDI* Virology and Serology Reporting Scheme this period (Tables 8 and 9).

The number of reports of **measles** and **rubella** remain at low levels.

The number of **hepatitis A** reports received has fallen after peaking in January (Figure 12).

Forty-five reports of **parainfluenza virus type 1** were received this period, 90% of which were for children under the age of five years. Epidemics of parainfluenza virus type 1 occur in Australia 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 lower than the last epidemic year of 1994 (Figure 13). In previous epidemic years reports have peaked in the months of April and May.

Figure 11. LabDOSS reports of blood isolates, by age group

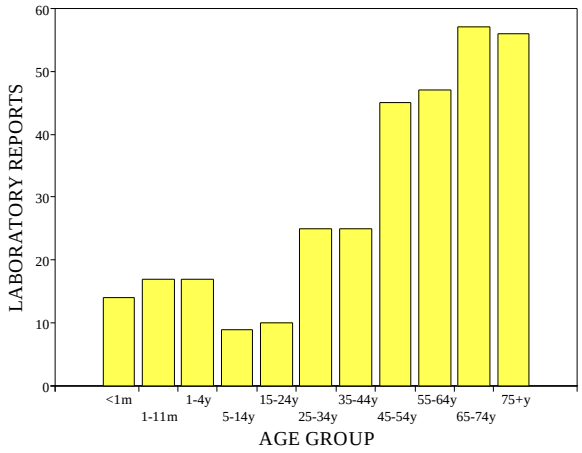


Figure 12. Hepatitis A laboratory reports, 1995 to 1996, by month of specimen collection

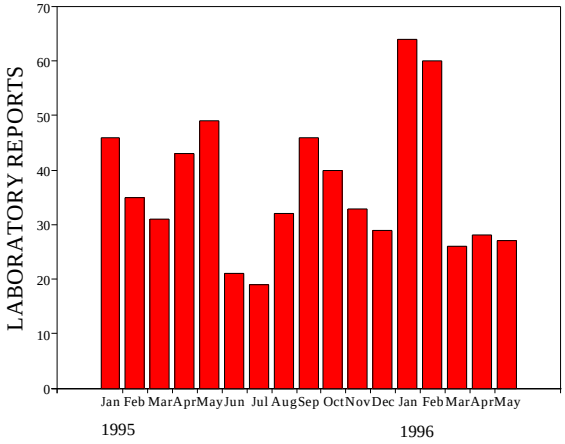
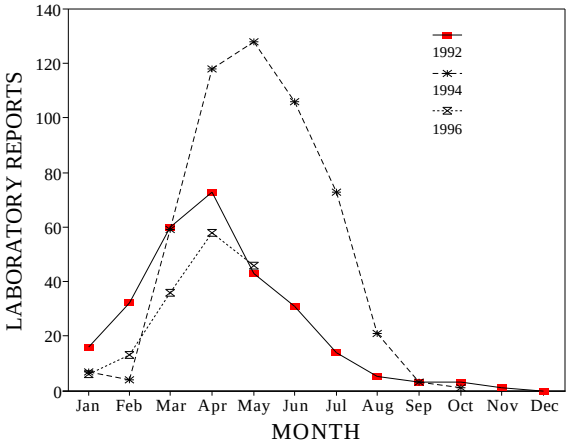


Figure 13. Parainfluenza virus type 1 laboratory reports, 1992, 1994 and 1996, by month of specimen collection



Respiratory syncytial virus was reported for 385 patients this period. Diagnosis was by antigen detection (219) and virus isolation (166). Ninety-six per cent of reports were for children under the age of five years and 73% under the age of one year. The number of reports has risen in recent months but remains average for the time of year (Figure 14)

Eighty-seven reports of **rotavirus** were received this fortnight. This is average for the time of year. The male:female ratio was 1:1 and 80 (92%) were for children under the age of five years, 26 of these (30%) being under one year of age. The peak month for rotavirus reports is August.

Figure 14. Repiratory syncytial virus laboratory reports, 1994 to 1996, by month of specimen collection

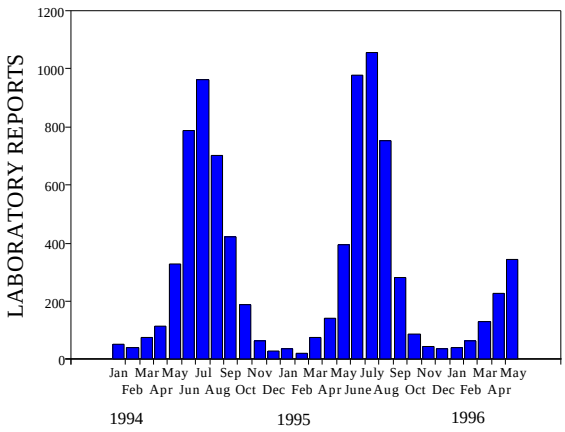


Table 8. Virology and serology laboratory reports by State or Territory[1] for the reporting period 30 May to 12 June 1996, historical data[2], and total reports for the year

BATCH 238, FROM 30/05/96	TO 12/06/96										
.	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											
Rubella virus		4		1			1		6	12.5	267
HEPATITIS VIRUSES											
Hepatitis A virus		2	1				3	1	7	21.2	248
ARBOVIRUSES											

Table 8. Virology and serology laboratory reports by State or Territory[1] for the reporting period 30 May to 12 June 1996, historical data[2], and total reports for the year, continued

Ross River virus		4	2			1		6	13	83.7	2,792
Barmah Forest virus			1						1	17.0	129
Flavivirus (unspecified)	1								1	1.8	21
ADENOVIRUSES											
Adenovirus type 11							1		1	.3	2
Adenovirus type 19							1		1	.0	4
Adenovirus type 37							2		2	.0	6

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 30 May to 12 June 1996

STATE OR TERRITORY	LABORATORY	REPORTS
New South Wales	Institute of Clinical Pathology & Medical Research, Westmead	80
	Prince Henry / Prince of Wales Hospitals, Sydney	34
	Royal Alexandra Hospital for Children, Westmead	60
	Royal Prince Alfred Hospital, Camperdown	28
	South West Area Pathology Service, Liverpool	62
Queensland	State Health Laboratory, Brisbane	299
Tasmania	Royal Hobart Hospital, Hobart	6
Victoria	Microbiological Diagnostic Unit, University of Melbourne	2
	Monash Medical Centre, Melbourne	39
	Royal Children's Hospital, Melbourne	127
	Unipath Laboratories	3
	Victorian Infectious Diseases Reference Laboratory, Fairfield Hospital	69
Western Australia	Princess Margaret Hospital, Perth	146
	Royal Perth Hospital	12
	Western Diagnostic Pathology	81
TOTAL		1049