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# INFLUENZA - DEALING WITH A CONTINUALLY EMERGING DISEASE

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## Abstract

There are two major types of influenza virus, types A and B, which are responsible for disease in man. Both types of virus display a progressive antigenic change known as antigenic drift while influenza A occasionally undergoes a more dramatic change known as antigenic shift. Antigenic shifts are typically associated with pandemic spread and severe disease and it is now believed that they occur when a new virus strain evolves by genetic reassortment between avian and human influenza viruses. In recent years influenza surveillance has provided for a good antigenic match between vaccines and the circulating epidemic viruses and the development of safe vaccines with good protective efficacy have been possible. However, in a pandemic situation the spread of the new virus is much more rapid and may well outstrip the capacity to produce vaccines.

## Influenza surveillance - origins of the global network

Influenza is one of the few diseases for which there is a truly worldwide surveillance network, the World Health Organization (WHO) global influenza program, which had its beginnings at the Fourth International Congress on Microbiology in Copenhagen in July 1947. At that time, a group of virologists proposed to the Interim Commission of the World Health Organization that a global program was required to study the epidemiology of influenza. They were prompted by a marked antigenic change in the circulating influenza A viruses in 1946-47 which brought associated vaccine failures. There were underlying concerns that future antigenic changes might give rise to a new pandemic strain with similar properties to the 1918-19 pandemic virus which claimed an estimated 21 million human lives, many of them in the 20-40 year-old age group.

The two major objectives of the WHO program at its outset were to[1]

- study the origins of epidemic and pandemic influenza strains, and
- provide new virus strains quickly for the production of vaccines in the event of outbreaks.

Since then the WHO influenza surveillance network has grown to involve approximately 110 National Influenza Centres in 80 countries and three WHO Collaborating Centres for Influenza Reference and Research located in London, Atlanta and Melbourne.

## Antigenic variation in influenza

Largely as a result of the WHO program, we now have a good understanding of the origins of new influenza strains through two forms of antigenic change which are often referred to as antigenic drift'and antigenic shift. Two major serotypes of influenza, types A and B, are responsible for human disease, both of which display antigenic drift. As the term implies, this is a continual and usually gradual antigenic change in the two viral surface antigens, the haemagglutinin and neuraminidase, which are embedded in the lipid membrane surface of the virus. Both antigens are glycosylated proteins; the haemagglutinin, which is more abundant, is also the more important antigen in the production of protective immunity. Only influenza A undergoes the more dramatic form of variation, or antigenic shift, in which a virus of a new subtype with completely different haemagglutinin, and often a different neuraminidase, suddenly appears in the human population. Antigenic shifts are typically associated with rapid pandemic spread, severe disease and a high level of mortality, and they occur at quite unpredictable intervals.

The degree of antigenic change in influenza is much greater than that observed for many other micro-organisms. This is largely due to the unique structure of the viral genome which exists as eight segments of negative sense, single-stranded RNA. Antigenic drift is now known to be a consequence of the very high mutation rate resulting from the uncorrected errors which occur when the single-stranded RNA is replicated. This has been estimated to occur at a frequency of $1.5 \times 10^{-5}$ per nucleotide per replicative cycle, a rate around ten times greater than that for the polio virus genome[2]. In addition, the surface structure of the virus is rather more accommodating to changes in the surface proteins than more highly constrained structures such as the icosahedral capsid of the polio virus.

## Antigenic change by genetic reassortment

The influenza A haemagglutinin has been recognised in 15 serologically distinct forms or subtypes and the neuraminidase in nine types[3,4]. All of these are to be found in aquatic birds, particularly ducks, which are now considered to be the primary host of influenza A viruses. A small number of the subtypes have become adapted to certain mammalian hosts, including humans, horses and the domestic pig. From time to time there is evidence of transmission of an avian influenza virus to a different species, such as the outbreak that occurred in harbour seals in the north-eastern United States of America in 1979[3].

Antigenic shifts accompanied by pandemic influenza in humans have been experienced only three times this century, in 1918-19 (Spanish Influenza), 1957 (Asian Influenza) and 1969 (Hong Kong Influenza). A further antigenic shift occurred in 1977, which affected primarily younger members of the population, in what might be termed a pseudopandemic. There is now overwhelming circumstantial evidence that antigenic shift can occur by a process of genetic reassortment between avian influenza and human influenza viruses. It has long been known that reassortment of the RNA gene segments from two influenza A viruses could be readily achieved in the laboratory by dual infection of cell cultures or embryonated eggs. This is a method regularly used for producing high-yielding strains of new influenza A variants for vaccine production[5].

Sequence analysis of RNA from the 1957 and 1969 pandemic viruses indicates that they almost certainly arose by genetic reassortment between the previous circulating human influenza strains and an avian virus such that the reassortant virus in both cases received the haemagglutinin and one non-structural gene of the avian virus[3]. In the case of the 1957 virus, the reassorted virus also received the avian neuraminidase. The fact that the Hong Kong virus shared a common neuraminidase with the preceding subtype may account for the slightly lesser impact that this virus had compared with the 1957 virus.

## The domestic pig as a genetic mixing vessel

While there is considerable evidence that avian viruses can cross the species barrier to horses, pigs and sea mammals, and ample evidence of antibody activity to a number of avian subtypes in people living in China[6], it is thought that the avian viruses will not readily adapt to growth in humans. Laboratory evidence suggests that the internal nucleoprotein of influenza viruses may play a role in restricting the species specificity of human and avian influenza[7]. This is why it is now widely believed that the domestic pig, which is susceptible to viruses with either form of the nucleoprotein, may play a role in genetic reassortment, acting as a genetic mixing vessel for generating new human pandemic strains.

To date, only two of the three human subtypes have been found in pigs. Sequence analysis of the RNA from $H_1N_1$ descendants of the 1918 virus which were isolated during the 1930s indicate that this virus may not have been formed by reassortment but rather by adaption of an avian virus to humans[3]. It has been suggested, however, that this is most likely to have occurred after adaption in the pig as an intermediate host. This is consistent with the apparently different geographic origins of the 1918 virus. The virus is thought to have originated in either France or the United States of America, while most new pandemic viruses appear to have their origins in China, where the agricultural practices would be conducive to the involvement of the domestic pig in genetic reassortment. There is no doubt that influenza viruses from pigs can infect humans and cause disease. This was demonstrated at Fort Dix in the United States of America in 1976 with the outbreak of A/New Jersey/76 virus, which was clearly derived from a swine influenza virus[8]. This occurred again recently in Europe where there are two recorded instances of transmission of human-avian reassortant viruses from pigs to humans[9].

Based on virological studies since the 1930s and retrospective serology for the preceding half-century, it seems fairly certain that during the last 110 years humans have been host to only three of the 15 influenza A haemagglutinin subtypes, and that each of these has occurred on two separate occasions. In the case of the $H_2$ subtype in 1957 and $H_3$ subtype in 1969, the virus recurred after an absence of about 70 years when there would have been little residual immunity in the human population. On both occasions, the new subtype completely replaced the previously circulating influenza A subtype. The recurrence of the $H_1$ subtype in 1977 was quite different, occurring after an absence of only 20 years. It did not replace the circulating $H_3$ subtype.

The short absence from the population and close genetic similarity between the 1977 virus and strains circulating in 1956 have resulted in speculation regarding the origin of the virus, including the possibility that it may have been inadvertently released from a laboratory[10]. Despite the advances in our knowledge of pandemic influenza, many unanswered questions remain, not least among them is whether there is some inherent restriction in the subtypes which can infect humans or whether all 15 may have this potential - a rather frightening possibility.

## The impact of influenza

Historically, the impact of pandemic influenza has raised the greatest public health concerns, while the pandemic impact of the disease is frequently underestimated. The annual death rate due to influenza in the United States of America has been estimated as 10-40,000 per annum. There are a number of studies which indicate that the total excess mortality associated with influenza is, in fact, many times higher than that recorded as influenza and pneumonia deaths[11]. The majority of the additional deaths are attributed on death certificates as due to cardiovascular disease and other causes[12,13].

While mortality is an obvious outcome of epidemic influenza, it reflects only a minor portion of the total impact of disease. The full economic impact, including cost to the individual, cost to the health care system and cost of lost productivity are extremely difficult to estimate. One published estimate put the annual cost to the United States of America as $3-5 billion per annum in 1986 dollars[14]. The cost to the Australian Medicare was estimated at $96 million during a moderate epidemic in 1985[15].

## Safety and efficacy of vaccines

It was demonstrated with the earliest inactivated influenza virus vaccines during the 1940s that these could

confer significant protection against infection. It quickly became clear that the level of protection was dependent on the closeness of the antigenic match between the vaccine strains and the circulating epidemic viruses. In addition, the level of absolute protection against infection in the elderly was considerably lower than that achieved in younger adults[16]. There have been a number of reports of serious vaccine failures when vaccines were not updated sufficiently quickly to cater for antigenic drift or shift changes in the virus.

Early influenza vaccines tended to be unduly reactive and the development of more acceptable vaccines took place over many years. This was achieved by progressive improvements in the methods used to purify the vaccine virus and, eventually, the finding that it was necessary to remove an apparent intrinsic toxicity associated with highly purified inactivated whole virus by chemical disruption or splitting[17]. Although one may occasionally hear anecdotal reports to the contrary, controlled placebo trials show unequivocally that today's split product and purified subunit vaccines are essentially devoid of systemic reactions[18], except in very young children[17]. These same trials did indicate, however, that the vaccines may produce mild transient local reactions in up to 20% of recipients.

Although inactivated vaccines fall well short of absolute protection against infection in the major target group - the older adult - there are now many studies which demonstrate a high level of protection against the severe consequences of infection. The studies also showed that excellent benefits can be achieved by annual vaccination in this group and others who are at increased risk of post-influenzal complications. A recent meta-analysis of 20 studies in elderly recipients showed that vaccination reduced total respiratory illness by an overall 56%, pneumonia by 53%, hospitalisation by 50% and death by a surprising 68%[19].

These figures are even more impressive when it is considered that agents other than influenza contribute significantly to these outcomes. Importantly, as shown in a recent study by Nichol[20], these benefits could be demonstrated even in a non-epidemic year, demonstrating that influenza is associated with an annual background of increased hospitalisations and mortality which can be significantly reduced by vaccination. Consistent with the studies on mortality, the Nichol study also showed that vaccination produced a reduction in hospitalisation for cardiac failure during a year when influenza A was epidemic. A number of economic analyses indicate that there is a substantial cost-benefit from influenza vaccination in the defined risk groups[20,21,22]. It is probably among the most cost-effective medical interventions possible in the older adult population. A recent study also showed that vaccination can be cost effective in a healthy young adult working population[23].

## Annual vaccine formulation

While the origins of new influenza strains are now much better understood, global surveillance remains as important as ever to provide the data and virus strains required for regularly updating vaccines. In February each year, the World Health Organization convenes its annual consultation on influenza vaccine formulation and reviews the accumulated surveillance data provided through the National Influenza Centres, the strain analysis data and serological results from the three collaborating centres. It might seem remarkable that much of the information considered by this meeting is generated by essentially the same methods that were used when the WHO network was first proposed almost 50 years ago. The haemagglutination-inhibition test which employs reference antigens grown in embryonated eggs and antisera prepared by infecting ferrets, is still the preferred method for antigenic analysis of virus isolates but is now supplemented by the use of monoclonal antibody panels and by sequence analysis of the viral haemagglutinin antigen.

To ensure the best possible match between the vaccine and circulating viruses, the Australian Influenza Vaccine Committee meets in late September each year to review the WHO formulation decision and to update it if necessary. Since 1977, when the re-emergence of the $H_1N_1$ subtype necessitated a trivalent influenza vaccine formulation, the committee has further updated the WHO formulation by, on average, one of the three vaccine component strains each year. The selection of the correct vaccine formulation is not always straightforward, as there are often multiple variants of a virus type or subtype present at any one time. It is a reflection of the success of the WHO surveillance network that both the WHO and Australian formulations, over recent years, have demonstrated a high degree of success in matching vaccine strains to the circulating epidemic viruses.

## Coping with the next pandemic

It is generally agreed among virologists that the question is *when* the next pandemic will occur, rather than *if* one will occur. Our ability to cope with a pandemic has gone essentially untested since 1969. While the evolution of new epidemic drift variants typically takes place over a period of 12-18 months, the new pandemic viruses in 1957 and 1969 spread essentially worldwide within six months of detection. The rate of spread outstripped the production of vaccine, although in some countries vaccines were available ahead of a second, more severe wave of infection which often occurs. Influenza surveillance has recently been improved in China and detection of the next pandemic virus may occur more quickly and closer to its source than previously. It would seem likely that the increasing extent and speed of human travel will spread the new virus even more rapidly than before, offsetting any advantage that might have been gained.

Many countries are now formulating detailed pandemic plans to determine how they might minimise the impact and social disruption which could occur if a virulent virus such as that experienced in 1918 should appear.

## Now is the time for influenza vaccinations

### National Health and Medical Research Council recommendations on influenza immunisation[1]

Influenza vaccine should be given routinely on an annual basis to:

- Individuals over 65 years of age: the risk to the elderly is greatest if they also have chronic cardiac or lung disease, and is increased for residents of nursing homes and other chronic care facilities;
- Aboriginal and Torres Strait Islander adults over 50 years of age, because of the greatly increased risk of premature death from respiratory disease.

Annual vaccination should be considered for individuals who are in the following groups:

- Adults with chronic debilitating diseases (especially those with chronic cardiac, pulmonary, renal and metabolic disorders);
- Children with cyanotic congenital heart disease;
- Adults and children receiving immunosuppressive therapy;
- Staff who care for immunocompromised patients (patients with immune deficiency or malignancy, bone marrow transplant recipients and liver transport recipients are at high risk from influenza infection, but have an attenuated immune response to influenza vaccine);
- Residents of nursing homes and other chronic care facilities;
- Staff of nursing homes and other chronic care facilities (in an attempt to protect the patients).

1. Based on: National Health and Medical Research Council *The Australian immunisation procedures handbook*, fifth edition. Canberra: Australian Government Publishing Service, 1995.

## Progress in the control of influenza

A number of developments currently in progress may influence our ability to deal with epidemic and pandemic influenza in the future. There is renewed interest in the possibility of commercialising live attenuated influenza vaccines which have been trialed over many years[24], particularly in children, but which have yet to gain registration in Western countries. New and improved vaccines using a variety of adjuvants, recombinant antigens and the exciting prospect of DNA vaccination hold promises such as improved protection[25], broader and longer-lasting immunity and simplified or more rapid vaccine production. There is also the prospect that a new specific antiviral compound will be available within the next few years[26].

None of these, however, is likely to obviate the need for an effective global surveillance program and detailed knowledge of the circulating virus strains. Nor are they likely to have any significant impact on the ongoing antigenic variation of the virus. It is worth reflecting on how much might be achieved by simply improving the use of today's effective influenza vaccines compared with the promises offered by the products of the future.

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# IMMUNISATION COVERAGE OF TWO YEAR OLD CHILDREN IN CHILD-CARE CENTRES IN THE ILLAWARRA AND SHOALHAVEN REGIONS OF NEW SOUTH WALES

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## Abstract

A survey of immunisation records was conducted in registered child-care centres in the Illawarra and Shoalhaven regions of New South Wales in 1995. The study showed that amongst two year old children attending child-care centres in those regions, less than 66% were fully immunised, excluding Hib vaccine. Of these, less than 25% had had their immunisations at the due time or within 30 days of when they were due. The survey highlighted the need to address our immunisation programs.

## Background

Under section 42c of the New South Wales Public Health (Amendment) Act 1992, directors of child-care centres are required to maintain a register of the immunisation status of children enrolled in their facility. In 1995, the New South Wales Department of Health introduced the State-wide Sentinel Immunisation Surveillance Scheme (SSISS). This scheme measures the age-appropriate vaccination of two year old children attending child-care in New South Wales by reporting the immunisation status of a sample of children. The scheme surveys a portion of children between 24 and 36 months of age enrolled in registered child-care centres in that State.

New South Wales is served by 14 public health units, an important role of which is the prevention and control of infectious diseases. Public health units were directed to supply unit record data for their area or district for a specified reporting period, usually one month. For the Illawarra region, the sample represented 34 records, randomly selected, taken in September 1995. The Illawarra Public Health Unit however surveyed children in all registered child-care facilities in the Illawarra and the Shoalhaven regions. The survey offered the unit a means of increasing communication with the local centres and providing better links with children and their families.

## Methods

The Illawarra Public Health Unit surveyed every licensed child-care facility, preschool and kindergarten in the Illawarra and Shoalhaven regions. The addresses of the centres were obtained from the New South Wales Department of Community Services regional offices, as all child-care centres must be licensed in order to operate in New South Wales. The directors of child-care centres were reminded that, under provisions of the New South Wales *Public Health (Amendment) Act 1992*, they are required to maintain a register of the immunisation status of children attending the facility. The letter that was sent to schools also contained a sample enrolment form which had provision for recording immunisation data for each enrolled child.

Following the initial notification, all facilities were asked to complete survey forms regarding the immunisation status of children born between 15 September 1992 and 14 September 1993. Information was requested about whether and at what date the children had received two-, four- and six-month diphtheria-tetanus-pertussis vaccines (DTP), oral polio vaccine (OPV), measles-mumps-rubella vaccine (MMR), 18-month DTP, and *Haemophilus influenzae* type B vaccine (Hib) for two, four, six, twelve and eighteen months, where appropriate.

Centres were sent two follow-up letters four weeks apart and several were telephoned.

Fully immunised was defined as having had all eight immunisations by the age of two years. Age appropriate was defined as receiving all eight immunisations at the due time or within 30 days of when they were due. While data were collected for Hib immunisations, they were not included in the analyses.

Data were analysed using the Epi Info version 5 program. Immunisation status was categorised as

On time: those immunised either on the date due or within 30 days of that date.

Late: those who had had the immunisation but the immunisation was given more than 30 days late.

Partial: those who had been only partly immunised.

No: those who had not supplied any records and who may or may not have been immunised.

Unknown: those who were recorded as being immunised but the dates were not available.

The New South Wales Midwives Data Collection for the period September 1992 - September 1993 provided the number of births recorded for the Illawarra and Shoalhaven hospitals as 4,679[1]. This figure serves as an estimate of the total number of two to three year old children living in the Illawarra and Shoalhaven regions.

## Results

The list of child-care centres provided by the department contained the addresses of 169 facilities, of which

**Table 1. Fully immunised status[1] for 1,109 children in child-care in the Illawarra and Shoalhaven regions**

| Immunised | Number | % |
|---|---|---|
| On time | 267 | 24.1 |
| Late | 466 | 42.0 |
| Partial[2] | 193 | 17.4 |
| No | 109 | 9.8 |
| Unknown | 74 | 6.7 |
| Total | 1109 | 100 |

1. Totals of two-, four- and six-month DTP/OPV, twelve-month MMR and eighteen-month DTP.
2. Children who had received only part of the recommended immunisation schedule, excluding Hib.

eight were no longer operating. Two were excluded from the survey; one was an occasional child-care centre and one was a mobile service operating only two hours per day at various centres. Of the 159 remaining centres 85 catered for children under three years of age. Of these, five did not respond in time for the data to be included in the survey, leaving 80 centres which were included in the survey.

The immunisation records of 1,109 two to three year old children were included in the survey and represented 23.7% of the estimated total population of 4,679 children of the relevant age in the region.

Immunisation coverage rates are summarised in the tables. Of the 1,109 two year old children surveyed, 66.1% were fully immunised but only 24.1% had been age appropriately immunised (Table 1). Table 2 shows coverage for individual immunisations.

On completion of the data analysis, a brief report of the findings was distributed to all the child-care centres. The report contained the basic results of the data analysis and stressed the need for continuing surveillance of childhood immunisation rates. The report was designed to be reproduced in child-care centre parents newsletters prior to Christmas 1995. A shorter statement was released to the local media. An article about the findings appeared in *Illawarra Mercury* on 20 December 1995.

## Discussion

The results of this survey are only applicable to children in child-care. The survey, however, did not cover informal child-care arrangements such as nannies or those children cared for in private homes. It is not known whether children in formal child-care facilities are more likely to be immunised than those not in child-care or in informal care. Our survey showed however that immunisation levels are unacceptably low in formal child-care centres; coverage in other children may be even lower. In addition the risk of transmission of infectious diseases in child-care centres is high because of the close contact with other children. Finally while centres were given sufficient notice to update their records prior to the survey, there was no verification of the records, and some may have been incomplete.

This survey showed that immunisation coverage decreased with age and a child is more likely to be age-appropriately immunised for the initial immunisations than for the later ones. The survey also showed the urgent need not only to continue with immunisation programs for young children, but to target older children who are not fully immunised. Child-care centres represent an ideal forum for targetted immunisation programs.

The findings of this survey substantiate similar findings from a nationwide sample survey conducted by the Australian Bureau of Statistics. That survey showed 54% of two year old children in New South Wales had been fully immunised in April 1995 (excluding Hib)[2].

This survey has been based on the documentation of immunisation status of children attending child-care as required by public health legislation. The results provide a baseline against which progress can be monitored and indicate the need for further efforts to improve immunisation coverage.

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**Table 2. Individual vaccine coverage for 1,109 chidren in child-care in the Illawarra and Shoalhaven regions**

| Immunised | 2 month DTP/OPV % | 4 month DTP/OPV % | 6 month DTP/OPV % | 12 month MMR % | 18 month DTP % |
|---|---|---|---|---|---|
| On time | 75.2 | 66.1 | 54.7 | 44.9 | 44.3 |
| Late | 7.4 | 15.6 | 25.1 | 32.6 | 25.6 |
| Partial[1] | 1.0 | 1.9 | 2.5 | N/A | N/A |
| No | 10.5 | 10.7 | 11.8 | 16.2 | 23.8 |
| Unknown | 6.0 | 5.7 | 5.9 | 6.2 | 6.3 |
| Total | 100[2] | 100 | 100 | 100[2] | 100 |

1. Received only DTP or OPV
2. Figures rounded to the nearest whole number.

# EDITORIAL: MEASURING IMMUNISATION COVERAGE

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Measurement of immunisation status is not as straightforward as it might seem. A child may have received several vaccines but despite this he or she may *not* be adequately protected against disease. Protection depends on the nature of the vaccines, the ages at which doses were given, the intervals between doses, the age at which courses were started, and the size and sometimes site of the doses. Surveys of immunisation coverage need to consider at least some of these issues.

Depending on the focus of interest, there are several possible definitions of immunisation status. The following is one scheme:

- *immunised up to date*. This means that a child has received an immunising course of a vaccine (and therefore should be protected against disease);
- *fully immunised*. This means that a child is immunised up-to-date for all recommended vaccines for which the child is eligible;
- *age-appropriately immunised*. This means that a child has received a full course of a vaccine at the recommended ages. An age-appropriately immunised child would also, by definition, be immunised up to date.

The primary objective of an immunisation program is to prevent disease, therefore our principal measurement of coverage should indicate our success or otherwise in reaching this goal. In my view, the best summary of immunisation coverage *in a single figure* is the proportion of children fully immunised at the second birthday. At the age of two years all children should have completed the major part of the primary schedule and will have had six months to receive their fourth doses of Hib (*Haemophilus influenzae* type b) and DTP (diphtheria-tetanus-pertussis) vaccines.

There are further complications. Should there be a minimum starting age to count an immunisation series as valid? Should there be a minimum interval between doses? Courses started too young, or doses given too close together, may be ineffective. Although a child may be up to date (and therefore protected), not all doses may have been received at the appropriate ages. Children usually do not receive their immunisations on the exact day they become due.

A reasonable, although demanding, method of assessing up to date immunisation status would be to count as *invalid*

- doses of Hib, DTP and OPV (oral polio vaccine) given at ages younger than six weeks;
- doses of these vaccines given less than six weeks after prior doses; and
- doses of MMR (measles-mumps-rubella) vaccine given at ages younger than 9 months.

In the measurement of age-appropriate immunisation status, count as *on time* doses given within one month of the recommended age, provided prior doses have also been given on time.

Perhaps more important than absolute epidemiological validity is uniformity. Depending on the methods adopted, it is possible to derive very different coverage levels for the same population. It is also evident that we need several indices of immunisation coverage to serve different purposes. In my view, the most important is the age-specific proportion of fully immunised children, as this determines how well vaccine-preventable diseases will be controlled. In order to evaluate the effectiveness and acceptability of the immunisation service, we also need to know the age- and antigen-specific proportions of children immunised up to date and age-appropriately immunised. In an attempt to clarify some of these issues, Heath, Bin Jalaludin and colleagues at the Western Sector Public Health Unit in Sydney are conducting a survey of immunisation experts.

Lloyd's study (in this issue of *CDI* page 217) shows that the Australian immunisation program serves children well until they reach the age of 18 months, when there is a large decline in the percentage of children receiving their Hib and DTP booster doses. As a result there is a fall in the proportion of children immunised up to date. This has become a serious problem in several countries. Britain (from 1993) and New Zealand (from February 1996) have implemented accelerated immunisation schedules to increase coverage.

The rationale underlying these changes is that more children complete the schedule when vaccines are given at younger ages. In both Britain and New Zealand the recommended immunisation schedules have been redesigned to reduce the number of immunisation visits, particularly in the second year of life. In Britain the accelerated schedules for Hib and DTP vaccines have been subjected to clinical trial, and immunisation authorities in both countries consider higher levels of coverage protect children far more than extra doses of vaccine. Increasing immunisation uptake and declining disease incidence figures in Britain would indicate that they are right. Perhaps Australia should also consider an accelerated schedule.

# ADULT TETANUS VACCINATION LEVEL SURVEY 1996, SOUTH AUSTRALIA

*From CDC Bulletin South Australia, March 1996*

Tetanus still occurs across Australia despite a safe and effective vaccine being available since 1953. In South Australia, no cases of tetanus were notified in 1995. However, in 1994, six cases and two deaths were recorded. Tetanus costs about $100,000 per case to treat.

The Health Omnibus Survey conducted in the spring of 1995 by Harrison Health Research included the question for persons over the age of 15 years, 'Have you had a tetanus booster in the last 10 years?' The survey format was by personal interviews at home, based on Australian Bureau of Statistics collector districts and was a multi-stage, clustered, self-weighting, systematic area sample.

Overall, two-thirds (67.1%) of the respondents said they had had a booster in the last ten years (61.5% in 1990). Of the remainder, 28.7% stated that they had not had a booster and 4.1% did not know (Table 1).

Older people were less likely to have had a booster for tetanus. In the 15-24 year age group, 82.5% said yes while only 47.4% of those over 65 reported having had a booster.

An ongoing (minor) tetanus vaccination promotion commenced in 1991.

Between 1980 and 1995, 42 cases of tetanus were notified in South Australia.

## Comments

Although the tetanus vaccination cover has increased from 20% to 53.9% in five years (1990-95) in the over-50s age group, more rapid improvement is needed to prevent this disease (Table 2).

Tetanus vaccination levels should be increased in all age groups, especially adults and particularly those over 55 years of age, and Year 10 school students.

### NHMRC recommendations for tetanus vaccination

**Primary immunisation**

Tetanus immunisation is part of the standard childhood vaccination schedule. Doses of diphtheria-tetanus-pertussis vaccine (DTP) are given at two, four, six and 18 months, and at four-five years, with a dose of adult tetanus vaccine (ADT) at 15-19 years. Primary immunisation for children who have not reached their eighth birthday is achieved with three doses of DTP (or CDT if there is a genuine contraindication to pertussis vaccine) at two monthly intervals. Primary immunisation of children who have passed their eighth birthday can be achieved with three doses of ADT at two monthly intervals.

**Booster doses**

Although immunity following complete vaccination is long lasting, maintenance of immunity with booster doses at ten-year intervals is recommended. Booster doses can be given either as tetanus toxoid vaccine or as ADT; the latter is preferred.

In the event of a tetanus-prone injury, a booster dose of tetanus vaccine (ADT or tetanus toxoid) should be given if five or more years have elapsed since the previous dose.

**Table 2. Tetanus vaccination cover in South Australia, 1990 and 1995 for adults >15 years and >50 years**

| Year | Age group | Percentage cover (%) |
|---|---|---|
| 1990 | 15+ | 61.5 |
| | 50+ | 20 |
| 1995 | 15+ | 67.1 |
| | 50+ | 53.9 |

**Table 1. Percentage of survey respondents who had received a tetanus booster in the past 10 years, by age group**

| | Age group in years | | | | | | |
|---|---|---|---|---|---|---|---|
| | 15-24 | 25-34 | 35-44 | 45-54 | 55-64 | 65+ | Total |
| Sample size | 418 | 571 | 562 | 450 | 366 | 649 | 3,016 |
| Yes | 82.5 | 72.9 | 70.3 | 67.0 | 56.6 | 47.4 | 67.1 |
| No | 13.7 | 23.3 | 26.5 | 27.1 | 37.6 | 49.2 | 28.7 |
| Don't know | 3.7 | 3.9 | 3.2 | 5.9 | 5.8 | 3.4 | 4.1 |

Note: all results reported as a percentage.

# OVERSEAS BRIEFS

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

## Ebola-like Reston virus in monkeys

Reston virus, a filovirus closely related to Ebola virus, has been isolated from two non-human primates (*Cynomolgus*) held at a quarantine facility in Texas, United States of America. One animal was dead and the other sick when sampled. The monkeys had been imported from the Philippines. At present there is no indication of associated human disease. A team from the Centers for Disease Control and Prevention is conducting further investigations on site.

Reston virus was first discovered in 1989 in monkeys imported from the Philippines which had died in a holding facility in Reston, Virginia, just outside Washington, DC, United States of America. While monkeys suffer a severe disease often leading to death, the limited information available indicates that humans may not become clinically ill. This is based on the isolation of Reston virus from one asymptomatically infected animal handler identified during the original outbreak and a few seroconversions that were not associated with clinical disease.

A formal quarantine procedure for imported monkeys was developed following the original Reston episode. It is this system that apparently identified the current cases.

## Cholera

### Latin America

Cholera continues to spread in Latin America, with over 1.3 million cases and 11,339 deaths reported since the epidemic began in Peru in January 1991.

In 1995, 85,802 cases of cholera and 8,471 deaths were reported from 14 countries.

Cholera surveillance figures represent only a small fraction of the actual number of people infected. In addition, year-to-year trends may be distorted by changes in cholera case definitions or reporting policies.

Countries reporting cholera cases in 1995 included:

| | |
|---|---|
| Peru, | 22,397 cases and 171 deaths; |
| Mexico, | 16,430 cases and 137 deaths; |
| Brazil, | 15,915 cases and 85 deaths; |
| Nicaragua, | 8,825 cases and 164 deaths; |
| Guatemala, | 7,970 cases and 85 deaths; |
| Honduras, | 4,717 cases and 77 deaths; |
| El Salvador, | 2,923 cases and 5 deaths; |
| Bolivia, | 2,293 cases and 54 deaths; |
| Ecuador, | 2,160 cases and 23 deaths; |
| Colombia, | 1,922 cases and 35 deaths; |
| Argentina, | 188 cases and 1 death; |
| Costa Rica, | 24 cases and no deaths; |
| Belize, | 19 cases and no deaths; |
| USA, | 19 cases and no deaths. |

Chile, Guyana, Panama, Paraguay, Suriname, and Venezuela reported no cases.

Improved cholera surveillance will help enhance understanding of the disease and contribute to controlling its spread.

### Nigeria

The outbreak is continuing with particularly high numbers reported in Kano and Kebbi states. The total number of cases since 1 January is 14,161 with 785 deaths.

Other countries reporting cholera in the last week are Mali, Niger, Bolivia, Brazil and Mexico.

# COMMUNICABLE DISEASES SURVEILLANCE

## National Notifiable Diseases Surveillance System, 31 March to 13 April 1996

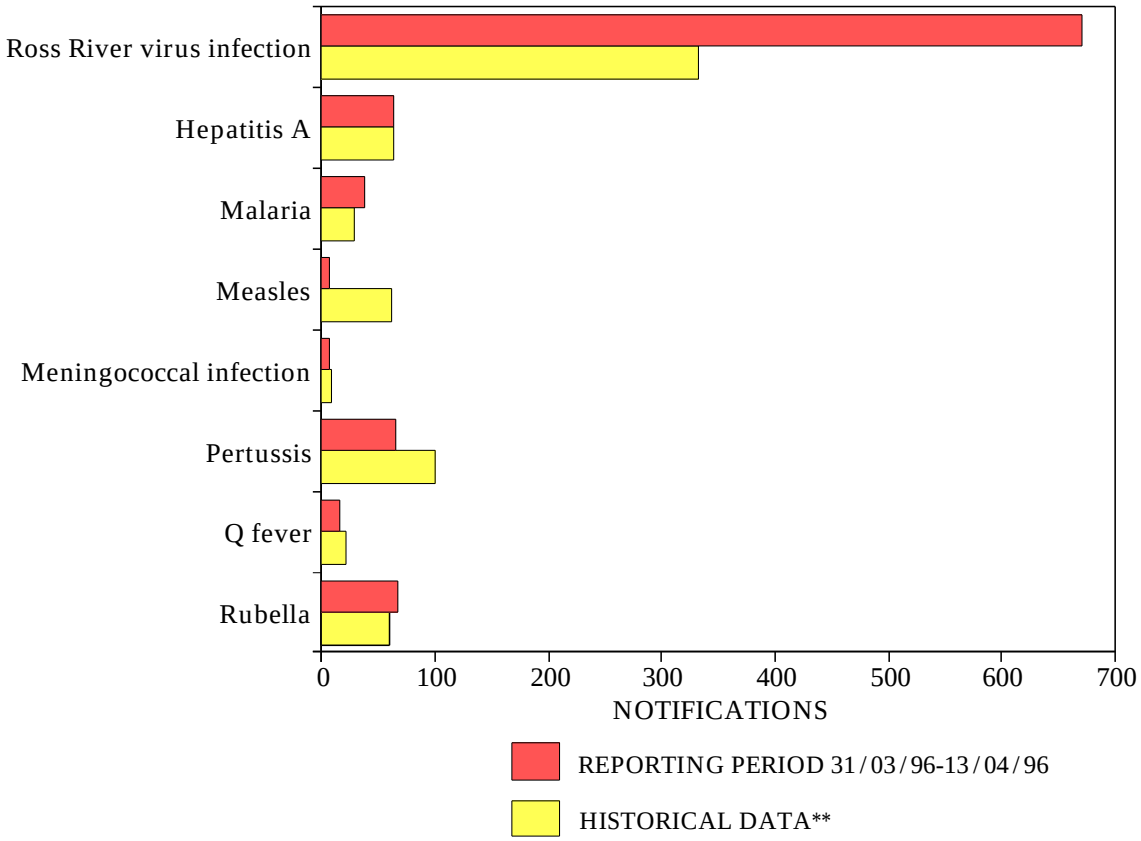
There were 2,208 notifications received for this two week period (Tables 1, 2 and 3, and Figure 1).

- There were 672 notifications of **Ross River virus infection**, half the number of cases reported for the previous fortnight. The male:female ratio was 1.0:1. All age groups were affected, although 61% of cases were aged between 30 and 54 years. The greatest numbers of cases continue to be reported from Queensland (especially the Southern and Central Coastal Statistical Divisions, and Darling Downs). In New South Wales the Northern and Richmond-Tweed Statistical Divisions are the most affected. In Western Australia the greatest numbers and rates are reported from the South West Statistical Division.
- Thirty-eight cases of **Barmah Forest virus infection** were reported from Queensland and one case from South Australia; 51% of the cases were in the age range 30-54 years.
- One case of **dengue** was reported from New South Wales in a male in the 35-39 years age group.
- Notifications of **campylobacteriosis** numbered 315, only 70% of the number reported for the previous fortnight. The male:female ratio was 1.2:1; all age groups were affected, with 23% of cases being aged less than five years.
- There were 192 notifications of **chlamydial infection** received. The male:female ratio was 1:2.3; 80% of the cases were aged between 15 and 29 years.
- There were 118 notifications of **gonococcal infection** received; 77 cases were male and 41 cases were female; 76% of the cases were aged between 15 and 29 years. One case was reported in a male child aged less than one year.
- One case of ***Haemophilus influenzae* type b infection** was reported, a five year old female from Victoria.
- There were 64 cases of **hepatitis A** reported, 80% of them in males. The cases were from all of the five-year age groups from 0-4 years to 65-69 years, with one case in an older male; 66% of the cases (42) were in males aged from 20 to 44 years. The majority of the cases were reported from the Metropolitan Statistical Divisions of Sydney (20 cases) and Melbourne (18 cases).
- Four cases of **hepatitis B (incident)** were reported; all were males, from three age groups between 15-19 years and 35-39 years.
- Five cases of **legionellosis** were reported. Three cases were in males and two in females. They were from age groups ranging from 50-54 years to 85-89 years. The cases were reported from the Metropolitan Statistical Divisions of Sydney, Adelaide and Perth.
- Seven cases of **leptospirosis** were reported. All were males: they were from several age groups between 25-29 years and 60-64 years. The cases were reported from five separate Statistical Divisions in New South Wales and Queensland.
- Two cases of **listeriosis** were reported; both were females over 60 years of age. The cases were reported from the Statistical Divisions of Brisbane and Adelaide.
- Thirty-eight notifications of **malaria** were received; 22 were males and 16 were female. Their ages ranged from four years to 74 years. The cases were reported from 13 separate Statistical Divisions in five States and Territories. The reported months of onset were January (11 cases), February (11), March (10) and April (6 cases).
- Seven cases of **measles** were reported; one case was male and six cases were female. Two cases were under five years of age; the remainder were from three age groups between 20-24 years and 50-54 years.
- There were eight cases of **meningococcal infection** reported from six separate Statistical Divisions in four States. All but one were males; their ages ranged from two years to 72 years. There was one apparent cluster of two cases reported from the same postcode area in New South Wales during the current and previous two-week reporting periods.
- There were 65 notifications of **pertussis**; 28 cases were male and 37 cases were female. All age groups but one from 0-4 years to 65-69 years were represented; one case was reported in an older male. Three cases were aged less than one year, and a further five cases were less than five years of age. Seven apparent clusters of two cases each were reported from the same postcode area during the reporting period; the apparent clusters occurred in four separate States and Territories.
- Sixteen notifications of **Q fever** were received; nine cases were male and seven cases were female. Fifteen cases were reported from eight separate rural Statistical Divisions in New South Wales, Queensland and Victoria. One case was reported from the Metropolitan Statistical Division of Melbourne. All age groups between 20-24 years and 55-59 years were represented. One case was reported in a female from the age group 10-14 years.
- There were 68 cases of **rubella** reported; 37 cases were male and 30 cases were female, the sex of the remaining case being not reported. The recorded ages of cases were from all five-year age groups up

to 50-54 years; 38% of the cases (26 cases) were reported in males 15-24 years of age, and 24% (16 cases) in women aged 15 to 44 years.

- There were 179 cases of **salmonellosis** reported; 98 cases were male and 76 cases were female; the sex of the remaining five cases was not reported; 43% of the cases were aged less than five years.
- Forty-five cases of **syphilis** were reported; 26 cases were male and 17 cases were female; the sex of the remaining two cases was not reported. All age groups but one from 15-19 years to 75-79 years were represented. There was one case reported in a female child aged less than one year.
- There were 28 cases of **tuberculosis** reported; 13 cases were male and 13 were female, the sex of the remaining cases being not reported. All age groups but one between 10-14 years and 80-84 years were represented. There was one case reported in a male child under five years of age.
- Three cases of **typhoid** were reported; all were male. The cases were reported from three separate Statistical Divisions in three States.
- Four cases of **yersiniosis** were reported. All cases were male. One case was reported in a child under five years of age, the remainder of the cases being aged between 20 and 34 years.

**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.

## 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 31 March to 13 April 1996

| DISEASES | ACT | NSW | NT | Qld | SA | Tas | Vic | 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 | 0 |
| *Haemophilus influenzae* b infection | 0 | 0 | 0 | 0 | 0 | 0 | 1 | 0 | 1 | 3 | 19 | 29 |
| Measles | 0 | 0 | 0 | 3 | 0 | 0 | 4 | 0 | 7 | 39 | 144 | 631 |
| Mumps | 1 | 0 | 0 | NN | 2 | 0 | 1 | 1 | 5 | 4 | 41 | 36 |
| Pertussis | 0 | 18 | 8 | 11 | 18 | 0 | 9 | 1 | 65 | 132 | 878 | 1455 |
| Poliomyelitis | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 |
| Rubella | 5 | 17 | 0 | 20 | 1 | 0 | 19 | 6 | 68 | 80 | 974 | 847 |
| Tetanus | 0 | 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 31 March to 13 April 1996

| DISEASES | ACT | NSW | NT | Qld | SA | Tas | Vic | WA | TOTALS FOR AUSTRALIA[2] This period 1996 | This period 1995 | Year to date 1996 | Year to date 1995 |
|---|---|---|---|---|---|---|---|---|---|---|---|---|
| Arbovirus infection | | | | | | | | | | | | |
| Ross River virus infection | 0 | 108 | 8 | 461 | 7 | - | 8 | 80 | 672 | 201 | 5081 | 848 |
| Dengue | 0 | 1 | 0 | 0 | 0 | - | 0 | 0 | 1 | 1 | 15 | 7 |
| Barmah Forest virus infection | 0 | 0 | - | 38 | 1 | 0 | - | - | 39 | 21 | 288 | 142 |
| NEC[3,4] | 0 | 24 | 2 | 0 | 0 | 0 | 4 | 2 | 32 | 57 | 162 | 166 |
| Campylobacteriosis[5] | 7 | - | 16 | 55 | 94 | 6 | 95 | 42 | 315 | 383 | 3395 | 3162 |
| Chlamydial infection (NEC)[6] | 5 | NN | 12 | 75 | 0 | 14 | 60 | 26 | 192 | 240 | 1978 | 1851 |
| Donovanosis | 0 | NN | 0 | 0 | NN | 0 | 0 | 0 | 0 | 2 | 17 | 27 |
| Gonococcal infection[7] | 1 | 15 | 24 | 37 | 0 | 0 | 9 | 32 | 118 | 154 | 1025 | 913 |
| Hepatitis A | 1 | 26 | 2 | 5 | 0 | 5 | 20 | 5 | 64 | 45 | 772 | 545 |
| Hepatitis B | 0 | 0 | 0 | 1 | 0 | 0 | 3 | 0 | 4 | 18 | 76 | 109 |
| Hepatitis C incident | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 11 | 5 | 26 |
| Hepatitis C unspecified | 14 | 0 | 19 | 59 | 0 | 14 | 87 | 34 | 227 | 292 | 2635 | 2427 |
| Hepatitis (NEC) | 0 | 0 | 0 | 0 | 0 | 0 | 2 | NN | 2 | 0 | 7 | 8 |
| Legionellosis | 0 | 2 | 0 | 0 | 2 | 0 | 0 | 1 | 5 | 6 | 52 | 73 |
| Leptospirosis | 0 | 2 | 0 | 5 | 0 | 0 | 0 | 0 | 7 | 5 | 71 | 40 |
| Listeriosis | 0 | 0 | 0 | 1 | 1 | 0 | 0 | 0 | 2 | 5 | 18 | 32 |
| Malaria | 0 | 10 | 0 | 22 | 1 | 0 | 4 | 1 | 38 | 41 | 225 | 186 |
| Meningococcal infection | 0 | 1 | 0 | 2 | 1 | 0 | 4 | 0 | 8 | 6 | 72 | 86 |
| Ornithosis | 0 | NN | 0 | 2 | 0 | 0 | 4 | 0 | 6 | 3 | 33 | 48 |
| Q fever | 0 | 10 | 0 | 4 | 0 | 0 | 2 | 0 | 16 | 15 | 138 | 136 |
| Salmonellosis (NEC) | 3 | 29 | 31 | 52 | 15 | 13 | 24 | 12 | 179 | 308 | 2118 | 2602 |
| Shigellosis[5] | 0 | - | 2 | 6 | 1 | 0 | 1 | 3 | 13 | 34 | 201 | 293 |
| Syphilis | 0 | 25 | 7 | 10 | 0 | 0 | 0 | 3 | 45 | 81 | 421 | 554 |
| Tuberculosis | 0 | 7 | 0 | 5 | 0 | 0 | 14 | 2 | 28 | 50 | 317 | 349 |
| Typhoid[8] | 0 | 0 | 0 | 1 | 1 | 0 | 1 | 0 | 3 | 1 | 33 | 27 |
| Yersiniosis (NEC)[5] | 0 | - | 0 | 2 | 1 | 0 | 0 | 1 | 4 | 12 | 86 | 134 |

1. For HIV and AIDS, see Tables 5 and 6. 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.

NEC Not Elsewhere Classified.

- Elsewhere Classified.

**Table 3. Notifications of rare[1] diseases received by State and Territory health authorities in the period 31 March to 13 April 1996**

| DISEASES | Total this period | Reporting States or Territories | Year to date 1996 |
|---|---|---|---|
| Botulism | 0 | | 0 |
| Brucellosis | 0 | | 9 |
| Chancroid | 0 | | 1 |
| Cholera | 0 | | 3 |
| Hydatid infection | 0 | | 11 |
| Leprosy | 0 | | 2 |
| 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.

## Australian Sentinel Practice Research Network

Data for weeks 13 and 14 ending 31 March and 7 April respectively are included in this issue of *CDI* (Table 4). The rate of reporting of influenza-like illness rose to 10.3 per 1000 consultations for week 14, the highest rate recorded by the scheme this year. The rate of reporting of pertussis has fallen in recent weeks, whilst that for gastroenteritis has risen.

## HIV and AIDS Surveillance

### Methodological note

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.

**Table 4. Australian Sentinel Practice Research Network, weeks 13 and 14, 1996**

| | Week 13, to 31 March 1996 | | Week 14, to 7 April 1996 | |
|---|---|---|---|---|
| Condition | Reports | Rate per 1000 encounters | Reports | Rate per 1000 encounters |
| Influenza | 61 | 6.3 | 114 | 10.3 |
| Rubella | 0 | 0 | 7 | 0.6 |
| Measles | 1 | 0.1 | 3 | 0.3 |
| Chickenpox | 14 | 1.5 | 19 | 1.7 |
| Pertussis | 3 | 0.3 | 4 | 0.4 |
| Gastroenteritis | 127 | 13.2 | 222 | 20.1 |

**Table 5. New diagnoses of HIV infection, new diagnoses of AIDS and deaths following AIDS occurring in the period 1 to 31 October 1995, by sex and State or Territory of diagnosis**

| | | ACT | NSW | NT | Qld | SA | Tas | Vic | WA | TOTALS FOR AUSTRALIA | | | |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| | | | | | | | | | | This period 1995 | This period 1994 | Year to date 1995 | Year to date 1994 |
| HIV diagnoses | Female | 0 | 1 | 0 | 0 | 0 | 0 | 0 | 0 | 1 | 7 | 64 | 65 |
| | Male | 2 | 29 | 0 | 16 | 1 | 0 | 17 | 1 | 66 | 78 | 648 | 724 |
| | Sex not reported | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 8 | 9 |
| | Total[1] | 2 | 30 | 0 | 16 | 1 | 0 | 17 | 1 | 67 | 85 | 722 | 798 |
| AIDS diagnoses | Female | 0 | 1 | 0 | 1 | 0 | 0 | 0 | 0 | 2 | 4 | 26 | 33 |
| | Male | 0 | 30 | 0 | 11 | 4 | 0 | 9 | 0 | 54 | 95 | 551 | 735 |
| | Total[1] | 0 | 31 | 0 | 12 | 4 | 0 | 9 | 0 | 56 | 99 | 578 | 772 |
| AIDS deaths | Female | 0 | 2 | 0 | 1 | 0 | 0 | 3 | 0 | 6 | 4 | 34 | 33 |
| | Male | 0 | 24 | 1 | 5 | 1 | 0 | 8 | 2 | 41 | 46 | 474 | 562 |
| | Total[1] | 0 | 26 | 1 | 6 | 1 | 0 | 11 | 2 | 47 | 51 | 509 | 599 |

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

**Table 6. Cumulative diagnoses of HIV infection, AIDS and deaths following AIDS since the introduction of HIV antibody testing to 31 October 1995, by sex and State or Territory**

| | | ACT | NSW | NT | Qld | SA | Tas | Vic | WA | AUSTRALIA |
|---|---|---|---|---|---|---|---|---|---|---|
| HIV diagnoses | Female | 15 | 543 | 3 | 94 | 44 | 4 | 163 | 69 | 935 |
| | Male | 165 | 9849 | 79 | 1541 | 554 | 70 | 3300 | 743 | 16301 |
| | Sex not reported | 0 | 2047 | 0 | 0 | 0 | 0 | 42 | 0 | 2089 |
| | Total[1] | 180 | 12446 | 82 | 1640 | 598 | 74 | 3513 | 814 | 19347 |
| AIDS diagnoses | Female | 5 | 130 | 0 | 27 | 18 | 2 | 47 | 17 | 246 |
| | Male | 71 | 3656 | 25 | 622 | 260 | 32 | 1290 | 263 | 6219 |
| | Total[1] | 76 | 3796 | 25 | 651 | 278 | 34 | 1344 | 281 | 6485 |
| AIDS deaths | Female | 2 | 96 | 0 | 20 | 13 | 2 | 31 | 9 | 173 |
| | Male | 49 | 2582 | 19 | 414 | 171 | 21 | 995 | 197 | 4448 |
| | Total[1] | 51 | 2684 | 19 | 436 | 184 | 23 | 1032 | 207 | 4636 |

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

HIV and AIDS diagnoses and deaths following AIDS reported for October 1995, as reported to 31 January 1996, are included in this issue of *CDI* (Tables 5 and 6).

## Surveillance of Serious Adverse Events Following Vaccination

The Serious Adverse Events Following Vaccination Surveillance Scheme is a national surveillance scheme which monitors the serious adverse events which occur rarely following vaccination. More details on 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 17 March to 13 April 1996**

There were eight reports of serious adverse events following vaccination for this reporting period. Reports were received from Queensland (2), the Northern Territory (2) and Western Australia (4).

Of the eight reports one was a case of persistent screaming, four of hypotonic/hyporesponsive episodes and three were other events temporally associated with vaccination (Table 7). The 'other' events included a severe local reaction following MMR vaccination, fever, vomiting and diarrhoea following DTP/Hib vaccination and dizziness and hypertension following ADT vaccination.

Events associated with DTP vaccine alone or DTP in combination with other vaccines were associated with the first (4), fourth (1) and fifth (1) doses. Two children were hospitalised. All children had recovered at the time the initial report was sent in.

**Table 7. Adverse events following vaccination for the period 17 March to 13 April 1996**

| Event | Vaccines | | | | | Reporting States or Territories | Total reports for this period |
|---|---|---|---|---|---|---|---|
| | DTP | DTP / Hib | DTP / OPV / Hib | ADT | MMR | | |
| Persistent screaming | 1 | | | | | Qld | 1 |
| Hypotonic/hyporesponsive episode | 3 | | 1 | | | Qld, WA | 4 |
| Other | | 1 | | 1 | 1 | NT, WA | 3 |
| Total | 4 | 1 | 1 | 1 | 1 | | 8 |

## Sterile Sites Surveillance (LabDOSS)

Data for this four-weekly period have been provided by seven laboratories. There were 237 reports of significant sepsis:

**New South Wales:** Royal North Shore Hospital 39.
**Tasmania:** Royal Hobart Hospital 21; Northern Tasmania Pathology Service 23.
**Queensland:** Sullivan and Nicholaides Partners 58; Ipswich General Hospital 30.
**Western Australia:** Princess Margaret Hospital for Children 20.
**South Australia:** Institute of Medical and Veterinary Science 46.

Organisms reported five or more times from blood are detailed in Table 8. Other blood isolates not included in Table 8 were:

**Gram-positive:** 1 *Bacillus cereus*, 1 *Corynebacterium jeikeium*, 1 *Corynebacterium* species, 4 *Enterococcus faecalis*, 1 *Enterococcus faecium*, 1 *Micrococcus* species, 3 *Streptococcus* Group B, 1 *Streptococcus* 'milleri', 2 *Streptococcus sanguis*, 2 *Streptococcus viridans* and 1 *Streptococcus* species.

**Gram-negative:** 4 *Acinetobacter* species, 1 *Aeromonas* species, 1 *Campylobacter jejuni*, 1 *Citrobacter freundii*, 3 *Enterobacter cloacae*, 2 *Enterobacter* species, 1 *Klebsiella oxytoca*, 1 *Klebsiella* species, 1 *Morganella morganii*, 4 *Proteus mirabilis*, 1 *Proteus* species, 1 *Psuedomonas pseudomallei*, 2 *Pseudomonas* species, 1 *Salmonella* species and 2 *Xanthomonas maltophilia*.

**Anaerobes:** 3 *Bacteroides fragiIis*, 1 *Bacteroides* species and 1 *Clostridium perfringens*.

**Fungi:** 1 *Candida* species.

There were 109 (49% of total) blood isolates reported for patients over the age of 65 years (Figure 2).

### Hospital acquired blood isolates

A total of 54 isolates were reported as being hospital acquired. The most commonly reported organisms were *Staphylococcus aureus* (16, including 4 MRSA) and *Staphylococcus epidermidis* (6).

**Table 8. 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 | Neonatal | |
| *Staphylococcus aureus* | 6 | 1 | | 3 | | 14 | 5 | 12 | 10 | 2 | 45[2] |
| *Staphylococcus* coagulase negative | | 2 | 1 | 1 | | 2 | 1 | 2 | 4 | 2 | 33 |
| *Staphylococcus epidermidis* | | | | 1 | 1 | 5 | 2 | 6 | 3 | 3 | 23 |
| *Streptococcus pneumoniae* | | 1 | 1 | | | | | | | | 5 |
| *Escherichia coli* | 1 | 1 | 1 | 7 | 13 | | 4 | 5 | | 1 | 38 |
| *Klebsiella pneumoniae* | | | | 1 | 4 | 1 | 3 | 2 | | 1 | 10 |
| *Pseudomonas aeruginosa* | | | 1 | | | 1 | 4 | 2 | 1 | | 8 |
| *Serratia marcescens* | | | | 1 | | | | 1 | | | 5 |
| *Candida albicans* | | | | | | 1 | 1 | 1 | 1 | | 5 |

1. Only organisms with 5 or more reports are included in this table.
2. MRSA 6.

**Meningitis and/or CSF isolate reports**

There were four reports of meningitis and / or CSF isolates. Included were 1 *Neisseria meningitidis* (seven month old female),1 *Staphylococcus aureus* (52 year old male with hepatic encephalopathy) and 2 *Streptococcus pneumoniae* (both female aged 58 years and 12 months).

**Isolates from sites other than blood or CSF**

**Joint fluid:** Two reports of *Staphylococcus aureus* were received in this period including 1 MRSA.

**Peritoneal dialysate:** Two reports were received in this period. Included was 1 *Staphylococcus aureus* and 1 *Streptococcus* species.

**Other:** A total of 7 reports was received. Included was 1 *Bacteroides* species, 1 *Candida* species, 1 *Enterococcus* species, 1 *Fusobacterium* species, 1 *Staphylococcus aureus* and 1 *Streptococcus* species.

## Virology and Serology Reporting Scheme

There were 1961 reports received in the *CDI* Virology and Serology Reporting Scheme this period (Tables 9, 10 and 11).

- **Ross River virus** was reported for 413 patients this fortnight. Diagnosis was by IgM detection (378), single high titre (29), fourfold change in titre (5) and virus isolation (one). Reports for 1996 to date are high compared to previous years (Figure 3).
- Eighteen reports of **Parainfluenza virus type 1** were received this period. Diagnosis was by antigen detection (12), virus isolation (5) and single high titre (one). Reports have increased in recent weeks (Figure 4).
- **Respiratory syncytial virus** was reported for 88 patients this period. Diagnosis was by antigen detection (59) and virus isolation (29). Ninety three percent (82) of the patients were between the ages of one month and 4 years old. Reports have been increasing over recent months.
- Nine reports of **Coxiella burnetti** (Q fever) were received this fortnight all patients being male. Diagnosis was by IgM detection (7), fourfold change in titre (one) and single high titre (one).
- ***Bordetella pertussis*** was reported for 35 patients this fortnight. Diagnosis was by IgA detction (28), single high titre (5) and antigen detection (2). Included were 23 females and 12 males. Eighty six percent of the cases were from Victoria with 14% from Western Australia.

**Figure 2. LabDOSS reports of blood isolates, by age group**

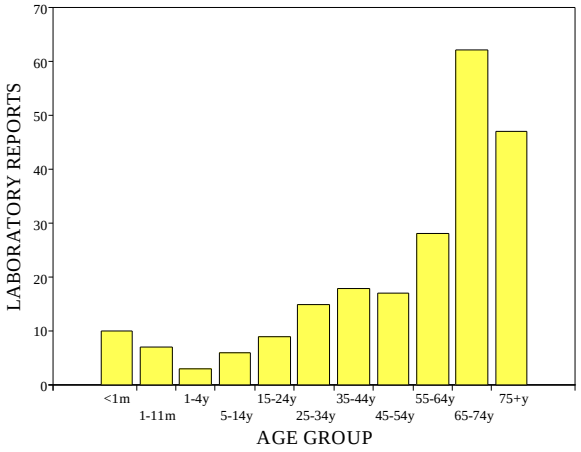



**Figure 3. Ross River virus laboratory reports, 1990 to 1995 average and 1996, by month of specimen collection**

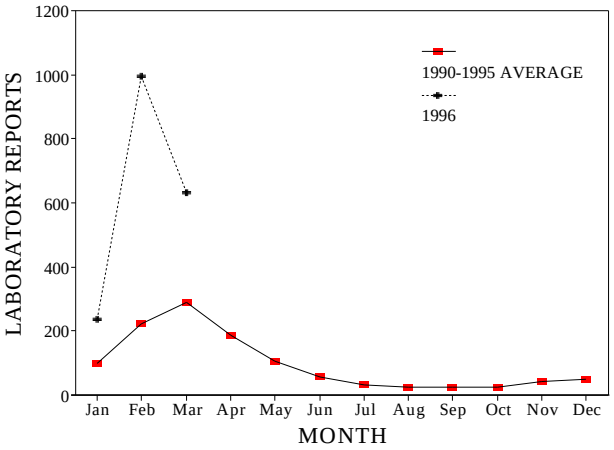
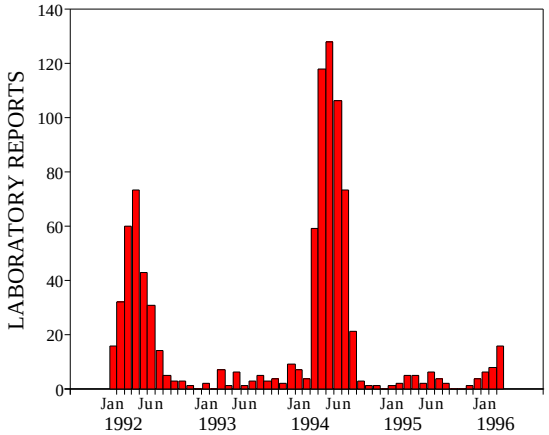



**Figure 4. Parainfluenza virus type 1 laboratory reports, 1992 to 1996, by month of specimen collection**



- Seven reports of **flavivirus** were reported this fortnight all from New South Wales. Diagnosis was byIgM detection in all cases. Included were 5 males and 2 females.
- **Rubella** was reported for 11 patients this period. Diagnosis was by IgM detection (10) and virus isolation (one). Included were 6 males and 5 females (2 childbearing age). Reports have decreased in recent months (Figure 5).

**Figure 5. Rubella laboratory reports, 1995 to 1996, by month of specimen collection**

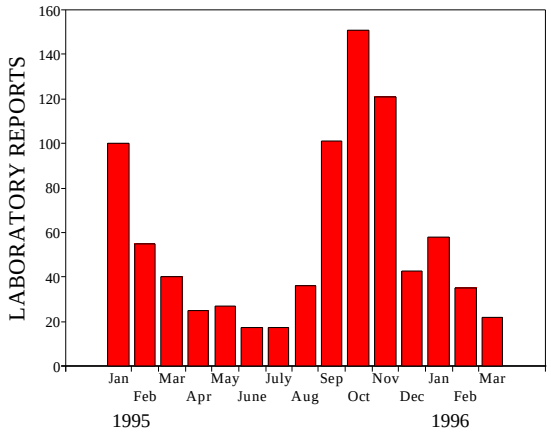



**Table 9. Virology and serology laboratory reports by State or Territory[1] for the reporting period 4 to 17 April 1996, historical data[2], and total reports for the year**

| BATCH 234, FROM 04/04/96 | TO 17/04/96 | | | | | | | | | | |
|---|---|---|---|---|---|---|---|---|---|---|---|
| | | | | | | | | | | | |
| . | 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 | | | | | | | | | | | |
| Measles virus | | | | | | | 1 | 1 | 2 | 39.5 | 24 |
| Mumps virus | | | | 2 | | | | 3 | 5 | 3.0 | 17 |
| Rubella virus | | 6 | | | | | 2 | 3 | 11 | 13.8 | 221 |
| HEPATITIS VIRUSES | | | | | | | | | | | |
| Hepatitis A virus | | 4 | 2 | | | | 1 | 9 | 16 | 12.7 | 190 |
| Hepatitis B virus | | | | | | | 7 | 1 | 8 | 81.7 | 440 |
| Hepatitis D virus | | 2 | | | | | | | 2 | .3 | 7 |
| ARBOVIRUSES | | | | | | | | | | | |

**Table 9. Virology and serology laboratory reports by State or Territory[1] for the reporting period 4 to 17 April 1996, historical data[2], and total reports for the year, continued**

| | | | | | | | | | | | |
|---|---|---|---|---|---|---|---|---|---|---|---|
| Ross River virus | | 31 | 3 | 147 | | | 4 | 228 | 413 | 95.3 | 2,066 |
| Barmah Forest virus | | | | 3 | | | | 1 | 4 | 11.3 | 94 |
| Dengue type 3 | | | | 1 | | | | | 1 | .2 | 3 |
| | | | | | | | | | | | |
| Dengue not typed | | | | | | | | 2 | 2 | 1.5 | 6 |
| Kunjin virus | | | 1 | | | | | 1 | 2 | .0 | 3 |
| Flavivirus (unspecified) | | 6 | | | 1 | | | | 7 | 2.0 | 16 |
| | | | | | | | | | | | |
| ADENOVIRUSES | | | | | | | | | | | |
| | | | | | | | | | | | |
| Adenovirus type 1 | | | | | | | 1 | | 1 | 1.2 | 8 |
| Adenovirus type 3 | 1 | | | | | | 2 | | 3 | 3.2 | 49 |
| Adenovirus type 7 | | | | | | | 1 | | 1 | .8 | 15 |
| Adenovirus type 40 | | | | | | | 2 | | 2 | .0 | 4 |
| Adenovirus type 43 | | | | | | | 1 | | 1 | .0 | 1 |
| Adenovirus not typed/pending | | 21 | 1 | 4 | | | 25 | 10 | 61 | 39.7 | 509 |
| | | | | | | | | | | | |
| HERPES VIRUSES | | | | | | | | | | | |
| | | | | | | | | | | | |
| Herpes simplex virus type 1 | 1 | 65 | 12 | 5 | | 11 | 51 | 116 | 261 | 136.3 | 2,154 |

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 10. Virology and serology laboratory reports by clinical information for the reporting period 4 to 17 April 1996**

| | Encephalitis | Meningitis | Other CNS | Congenital | Respiratory | Gastrointestinal | Hepatic | Skin | Eye | Muscle/joint | Genital | Other | Total |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| MEASLES, MUMPS, RUBELLA | | | | | | | | | | | | | |
| Measles virus | | | | | | | | 1 | | | | 1 | 2 |
| Mumps virus | | | | | | | | | | | | 5 | 5 |
| Rubella virus | | | | | | | | | | | | 11 | 11 |
| HEPATITIS VIRUSES | | | | | | | | | | | | | |
| Hepatitis A virus | | | | | | | 12 | | | | | 4 | 16 |
| Hepatitis B virus | | | | | | | 1 | | | | | 7 | 8 |
| Hepatitis D virus | | | | | | | | | | | | 2 | 2 |
| ARBOVIRUSES | | | | | | | | | | | | | |
| Ross River virus | | | | | | | | 24 | | 114 | | 275 | 413 |
| Barmah Forest virus | | | | | | | | | | | | 4 | 4 |
| Dengue type 3 | | | | | | | | | | | | 1 | 1 |
| Dengue not typed | | | | | | | | | | | | 2 | 2 |
| Kunjin virus | | | | | | | | | | | | 2 | 2 |
| Flavivirus (unspecified) | | | | | | | | | | | | 7 | 7 |
| ADENOVIRUSES | | | | | | | | | | | | | |
| Adenovirus type 1 | | | | | 1 | | | | | | | | 1 |
| Adenovirus type 3 | | | | | | 1 | | | | | | 2 | 3 |
| Adenovirus type 7 | | | | | | | | | | | | 1 | 1 |
| Adenovirus type 40 | | | | | | 1 | | | | | | 1 | 2 |
| Adenovirus type 43 | | | | | | | | | | | 1 | | 1 |
| Adenovirus not typed / pending | | 1 | | | 14 | 19 | | | 5 | | | 22 | 61 |
| HERPES VIRUSES | | | | | | | | | | | | | |
| Herpes simplex virus type 1 | | | | | 2 | | | 165 | 3 | | 25 | 66 | 261 |
| Herpes simplex virus type 2 | | | | | | | | 121 | | | 77 | 68 | 266 |
| Herpes simplex not typed / pending | 1 | | | | 1 | | | 5 | | | 3 | 67 | 77 |
| Cytomegalovirus | | 1 | 1 | 1 | 14 | 2 | | | | | | 50 | 69 |
| Varicella-zoster virus | | | | | | | | 46 | | | | 15 | 61 |
| Epstein-Barr virus | | | | | 27 | | 2 | 1 | | | | 45 | 75 |
| Herpes virus group - not typed | | | | | | | | 9 | | | 2 | | 11 |
| OTHER DNA VIRUSES | | | | | | | | | | | | | |
| Parvovirus | | | | | | | | | | | | 3 | 3 |

**Table 10. Virology and serology laboratory reports by clinical information for the reporting period 4 to 17 April 1996, continued**

| | Encephalitis | Meningitis | Other CNS | Congenital | Respiratory | Gastrointestinal | Hepatic | Skin | Eye | Muscle/joint | Genital | Other | Total |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| PICORNA VIRUS FAMILY | | | | | | | | | | | | | |
| Coxsackievirus A9 | | 1 | | | | | | 1 | | | | | 2 |
| Echovirus type 7 | | | | | | | | | | | | 1 | 1 |
| Echovirus type 30 | | | | | | | | | | | | 1 | 1 |
| Echovirus not typed / pending | | 1 | | | | | | | | | | | 1 |
| Poliovirus type 1 (uncharacterised) | | | | | | | | | | | | 3 | 3 |
| Poliovirus type 2 (uncharacterised) | | | | | | | | | | | | 1 | 1 |
| Poliovirus type 2 (vaccine strain) | | | | | 1 | | | | | | | | 1 |
| Rhinovirus (all types) | | | | | 24 | | | 1 | | | | 4 | 29 |
| Enterovirus not typed / pending | | 2 | | | 4 | 9 | | | | | | 18 | 33 |
| ORTHO / PARAMYXOVIRUSES | | | | | | | | | | | | | |
| Influenza A virus | | | | | 2 | 1 | 1 | 1 | | | | 2 | 7 |
| Influenza A virus H3N2 | | | | | 1 | | | | | | | | 1 |
| Influenza B virus | | | | | 1 | | | | | | | | 1 |
| Parainfluenza virus type 1 | | | | | 14 | | | | | | | 4 | 18 |
| Parainfluenza virus type 2 | | | | | 7 | | | | | | | | 7 |
| Parainfluenza virus type 3 | | | | | 8 | | | | | | | 11 | 19 |
| Respiratory syncytial virus | | | | | 77 | | | | | | | 11 | 88 |
| Paramyxovirus (unspecified) | | | | | 1 | | | | | | | | 1 |
| OTHER RNA VIRUSES | | | | | | | | | | | | | |
| HIV-1 | | | | | | | | | | | | 3 | 3 |
| HTLV-1 | | | | | | | | | | | | 1 | 1 |
| Rotavirus | | | | | | 30 | | | | | | 2 | 32 |
| Calici virus | | | | | | 2 | | | | | | | 2 |
| Norwalk agent | | | | | | 1 | | | | | | | 1 |
| OTHER | | | | | | | | | | | | | |
| *Chlamydia trachomatis* not typed | | | | | | 1 | | 7 | 3 | | 145 | 41 | 197 |
| *Chlamydia psittaci* | | | | | 2 | | | | | | | 1 | 3 |
| *Chlamydia* species | | | | | 1 | | | | | | | 9 | 10 |
| *Mycoplasma pneumoniae* | | | | | 10 | | | | | | | 19 | 29 |
| *Coxiella burnetii* (Q fever) | | | | | | | | | | | | 9 | 9 |
| *Streptococcus* group A | | | | | | | | | | | | 1 | 1 |
| *Brucella* species | | | | | | | 1 | | | | | 1 | 2 |
| *Bordetella pertussis* | | | | | 29 | | | | | | | 6 | 35 |
| *Legionella pneumophila* | | | | | 2 | | | | | | | | 2 |
| *Legionella longbeachae* | | | | | | | | | | | | 1 | 1 |
| *Leptospira canicola* | | | | | | | | | | | | 1 | 1 |
| *Leptospira hardjo* | | | | | 1 | 1 | | | | 1 | | 2 | 5 |
| *Leptospira australis* | | | | | | 1 | | 2 | | | | | 3 |
| *Leptospira* species | | | | | | | 1 | | | 1 | | 4 | 6 |
| *Treponema pallidum* | | | | | | | | | | | | 19 | 19 |
| *Entamoeba histolytica* | | | | | | | | | | | | 1 | 1 |
| *Toxoplasma gondii* | | | | | | | | | | | | 3 | 3 |
| *Schistosoma* species | | | | | | 1 | | | | | | 14 | 15 |
| *Strongyloides stercoralis* | | | | | | | | | | | | 1 | 1 |
| TOTAL | 1 | 6 | 1 | 1 | 244 | 70 | 18 | 384 | 11 | 116 | 253 | 856 | 1961 |

**Table 11. Virology and serology laboratory reports by contributing laboratories for the reporting period 4 to 17 April 1996**

| STATE OR TERRITORY | LABORATORY | REPORTS |
|---|---|---|
| New South Wales | Institute of Clinical Pathology & Medical Research, Westmead | 326 |
| | Royal North Shore Hospital, St Leonards | 18 |
| | Royal Prince Alfred Hospital, Camperdown | 27 |
| | South West Area Pathology Service, Liverpool | 146 |
| Queensland | State Health Laboratory, Brisbane | 196 |
| Tasmania | Northern Tasmanian Pathology Service, Launceston | 7 |
| | Royal Hobart Hospital, Hobart | 24 |
| Victoria | Microbiological Diagnostic Unit, University of Melbourne | 5 |
| | Monash Medical Centre, Melbourne | 21 |
| | Royal Children's Hospital, Melbourne | 148 |
| | Unipath Laboratories | 56 |
| | Victorian Infectious Diseases Reference Laboratory, Fairfield Hospital | 104 |
| Western Australia | PathCentre Virology, Perth | 552 |
| | Princess Margaret Hospital, Perth | 22 |
| | Western Diagnostic Pathology | 309 |
| TOTAL | | 1961 |