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IDENTIFICATION OF LIKELY NATURAL HOSTS FOR EQUINE MORBILLIVIRUS

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Abstract

We describe the isolation of a paramyxovirus from three species of flying fox in Queensland. The species are *Pteropus alecto* (black flying fox), *Pteropus scapulatus* (little red flying fox) and *Pteropus poliocephalus* (grey headed flying fox). The virus appears to be identical to the equine morbillivirus which was associated with acute equine respiratory syndrome and two human deaths in two Queensland outbreaks. The isolation of this new virus suggests that flying foxes may be the natural host for the virus that causes acute equine respiratory syndrome. ***Comm Dis Intell* 1996;20:476.**

Background

In September 1994, there was an outbreak of acute equine respiratory syndrome (AERS) due to a previously undescribed virus, equine morbillivirus (EMV), in south-east Queensland[1]. Both humans and horses were infected. One of the two human cases died. A third human case was diagnosed in October 1995[2]. In this case transmission had apparently occurred 12 months previously when the patient was exposed to two infected horses that died following a severe, acute respiratory illness. This human case also died after developing a recurring encephalitis.

Following the first outbreak, an investigation was begun to determine the natural host of the virus. Since 1994 over 5,000 animals throughout Queensland, including over 30 wildlife species, have been tested for the presence of antibodies to EMV. Only flying foxes have been found to have antibodies to this virus[3]. All other testing has failed to show any evidence of EMV infection.

Testing carried out at the Animal Research Institute in Brisbane revealed that about 15% of the flying foxes (comprised of four species) present in Australia had antibodies to EMV. This antibody reactivity to EMV confirmed that the bats had previously been infected by a paramyxovirus similar to EMV. It also suggested that the bat virus is closely related to EMV. However it was not possible to establish the degree of relatedness between the bat paramyxovirus (BPV) and EMV on the basis of antibodies alone. To do this it was necessary to isolate BPV and compare it with EMV.

Isolation of a bat virus

In September 1996, we isolated a paramyxovirus from the uterine fluids of a flying fox found in the Brisbane area. The injured female grey headed flying fox, *Pteropus poliocephalus*, had miscarried twin foetuses. We have since isolated an apparently identical virus from three other flying foxes, one *Pteropus alecto* (black flying fox), one *Pteropus scapulatus* (little red flying fox) and a second *Pteropus poliocephalus* (grey headed flying fox).

Using an immunofluorescent staining technique, we were able to show that the virus isolated from the injured female reacts strongly with human and horse serum samples containing antibodies to EMV. Both BPV and EMV produce similar changes in cell culture. They also appear to be identical by electron microscopy. Preliminary studies have shown that a part of the genetic sequence of the newly isolated virus is identical to the corresponding sequence of the EMV isolates. These results indicate that BPV and EMV are likely to be the same virus. Thus flying foxes may be the natural host of the virus which has caused two serious outbreaks of disease in horses and humans.

Implications for human health

Flying foxes are native to Australia and are found widely throughout the country. As only two outbreaks of EMV have been recorded to date, it appears that spill-over of this virus to other species is a rare event. Current evidence indicates that flying foxes do not pose a significant risk to human health[4]. The three human cases all contracted the virus from horses.

Work on the natural history of this virus is continuing. The Animal Research Institute (funded by a new initiatives research program of the Animal and Plant Health Service) will investigate possible modes of transmission from bat to horse. We will also investigate whether the apparent seasonal occurrence of disease is related to breeding cycles in flying foxes or to pregnancy in the two mares which originally contracted the virus.

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SCREENING OF BAT CARERS FOR ANTIBODIES TO EQUINE MORBILLIVIRUS

Linda Selvey[1], *Roscoe Taylor*[2], *Antony Arklay*[3] *and John Gerrard*[4]

Abstract

In response to a recent finding of antibodies reactive with equine morbillivirus in four species of flying foxes, people who had close association with flying foxes were tested for antibodies to the virus. One hundred and twenty-eight bat carers were tested, none of whom had detectable antibodies. As people who had regular contact with flying foxes were targeted in this study, the majority of the subjects had considerable histories of contact with flying foxes, including scratches and bites. These findings suggest that neither prolonged close contact nor casual contact with flying foxes engenders a risk of equine morbillivirus infection in humans. ***Comm Dis Intell* 1996;20:477-478.**

Introduction

Two outbreaks of equine morbillivirus infection involving three human cases have occurred in Queensland[1,2,3,4]. One was in Mackay and the other was in Brisbane. Both outbreaks involved horses and the human cases were horse handlers. In a search for an animal reservoir for equine morbillivirus (EMV), Young *et al* detected in 9% of flying foxes antibodies reactive with equine morbillivirus in ELISA and in neutralisation assays[5,6]. Antibodies were detected in serum samples taken from all four species of flying fox that occur in Queensland. Antibodies have not been detected in any of the 46 other animal species tested so far[6].

This finding raised concerns among some people who care for flying foxes. First, some carers were concerned that they were at risk of equine morbillivirus infection because of close contact with flying foxes. Secondly, many carers were concerned that some people may use the potential risk of infection to humans as a reason to shoot flying foxes. Many bat carer organisations rely on volunteers to care for orphaned flying foxes and on donations received for giving talks about flying foxes which involved showing live animals. Both these activities appeared to be under threat because of fears of equine morbillivirus infection. For these reasons, several bat carers requested testing for antibodies to equine morbillivirus infection.

Methods

Bat careers were contacted through bat carer organisations in Queensland and New South Wales. Serological testing was offered to bat carers who had prolonged, significant contact with either adult or juvenile flying foxes. Others who had lesser amounts of contact were tested because of concerns about occupational health and safety or because of their association with a bat carer organisation. All those who were tested were asked to complete a self-administered questionnaire in which their history of contact with flying foxes was documented. The sera were tested for antibodies to equine morbillivirus using an ELISA assay and any equivocal results were sent to the Australian Animal Health Laboratory for serum neutralisation assays.

Results

None of the 128 bat carers who were tested had detectable antibodies to equine morbillivirus. All carers were from Queensland except for six who were from New South Wales. The median duration of contact was 48 months, ranging from one month to 36 years. Of the carers, 74% reported daily contact with flying foxes. When the duration and frequency of contact were considered together, the median number of months in which contact with flying foxes was experienced was 25.5, ranging from less than one month to 36 years. One of the subjects had received several deep puncture wounds to a finger and web space of one hand from a bite while handling a bat now known to be EMV antibody-positive at the time of the bites. This person's serology was negative at both three days and eight weeks post-bite, and she remains well.

Of the carers, 74% reported having been bitten, 88% reported having been scratched, and 60% reported exposure to flying fox blood. The majority (72%) reported caring for sick and injured flying foxes. Carers had been exposed to all four species of flying fox, with 51% reporting exposure to grey flying foxes, 59% to little red flying foxes, 74% to black flying foxes and 41% to spectacled flying foxes.

Discussion

We tested 128 bat carers, most of whom had prolonged contact with flying foxes which included having been bitten, scratched and having exposure to blood. None of these carers tested positive for antibodies to EMV infec-

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tion. There are a number of possible reasons for this. First, the flying foxes may have been infected with a paramyxovirus related to equine morbillivirus but which does not infect humans. The fact that antibodies from flying foxes neutralise equine morbillivirus in vitro makes this explanation unlikely. Secondly, the infection in the flying foxes leading to the production of antibodies may be short lived, making it unlikely that a bat carer will have exposure to an infected flying fox. A further possible explanation is that equine morbillivirus infection is not readily transmitted from flying foxes to humans. As we did not test all bat carers our findings cannot totally exclude the possibility of transmission to humans. However if this has occurred it must be extremely rare.

Regardless of the explanation, our data suggest that neither prolonged close contact nor casual contact with flying foxes engenders a risk of equine morbillivirus infection in humans.

Acknowledgements

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A FIVE YEAR REVIEW OF *CAMPYLOBACTER* INFECTION IN QUEENSLAND

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Abstract

Campylobacter infection consistently has one of the highest annual notification rates of all communicable diseases. We reviewed the epidemiology of *Campylobacter* infection in Queensland by analysing notification data for a five year period (1991 to 1995). This included incidence, age and sex distribution, seasonality, geographic distribution, and socioeconomic status. The review found the highest notification rate in children aged 12 - 23 months. There was no distinct seasonal pattern of infection. *Campylobacter* infection was reported more frequently in urban areas and for persons residing in higher socioeconomic areas. It would appear that factors which influence notification rates in the general population do not necessarily have the same influence on the 0 - 4 years age group. ***Comm Dis Intell* 1996;20:478-482.**

Introduction

Campylobacter infection has emerged during the past decade as the most frequently notified cause of acute bacterial diarrhoea in Australia. *Campylobacter* enteritis has been a notifiable disease in Queensland since 1990. There are around 10,000 cases of *Campylobacter* infection notified in Australia each year, and the illness consistently has one of the highest annual rates of notification of all communicable diseases[1]. Cases are predominantly caused by two species, *Campylobacter jejuni* and *Campylobacter coli*, with *C. jejuni* responsible for up to 90% of the infections[2].

Previous studies have shown that the notification rate for *Campylobacter* is highest in the 0 - 4 years age group[3,4,5]. Other studies have identified a number of potential risk factors associated with *Campylobacter* infection, including inadequately cooked chicken, domestic pets such as cats and dogs, raw milk, untreated water, and poor food hygiene and handling practices[6,7,8,9,10,11,12]. Large outbreaks and person-to-person transmission appear to be uncommon[4]. We are currently conducting a matched case control study in the southern part of Brisbane and the Gold Coast areas to determine whether the above or additional risk factors are associated with *Campylobacter* infection in children less than two years old. To provide background information for this study, we reviewed the epidemiology of *Campylobacter* infection in this State for the five year period 1991 to 1995.

Methods

All *Campylobacter* notifications to Queensland Health with reported onset dates between 1 January 1991 and 31 December 1995 were collated and analysed using Epi Info

Table 1. *Campylobacter* notifications in Queensland and Australia, 1991 to 1995

Year	Queensland		Australia[1]	
	Number of cases	Age-adjusted notification rate per 100,000 population	Number of cases	Crude notification rate per 100,000 population
1991	2724	90.8	8672	75.8
1992	2968	97.1	9135	54.2
1993	2474	79.2	8111	69.6
1994	2131	66.6	10117	85.8
1995	1975	61.9	10,933	91.6

1. Refer reference 1.

(version 6.02). Crude and age-specific notification rates based on data collected over the total five year period were calculated using the 1991 census population as the denominator[13]. Yearly notification rates were calculated using the Australian Bureau of Statistics (ABS) mid-year estimated resident population figures as the denominator[14]. Queensland age-adjusted rates were calculated by direct standardisation using the 1991 Australian Standard Population[13]. Age-adjusted rates for socioeconomic status and geographic distribution were calculated by direct standardisation using the 1991 Queensland Census Population[15].

Statistically significant differences in notification rates between the same population over different periods or between populations from different geographic areas, socioeconomic groups, or age groups were determined by calculating 95% confidence intervals around the rates[16]. Confidence intervals around the rates were calculated using the following formula:

$$95\%\ CI = r \pm 61.981 \sqrt{r / n}$$

where r = rate per 1,000 and n = denominator upon which the rate is based. Two independent rates were considered to differ significantly at the 5% level if their 95% confidence intervals did not overlap.

The Queensland Index of the ABS Index of Relative Socio-Economic Disadvantage, at the level of statistical local area (SLA) was used as an indicator of the population's socioeconomic status[17]. This measure focuses on attributes such as low income, low educational attainment and high unemployment, and provides a general socioeconomic index for a defined geographic area. SLAs are categorised into one of five quintiles according to their index. Cases residing in SLAs belonging to quintile 1 were classified as persons from high socioeconomic areas, and cases residing in SLAs of quintile 5 were classified as persons from low socioeconomic areas. For the purposes of this review, cases grouped in quintiles 2 to 4 were considered as residing in average socioeconomic areas.

The Commonwealth Government Rural, Remote and Metropolitan Areas Classification was used as a method of classifying *Campylobacter* infection according to geographic location[18]. This classification categorises SLAs into metropolitan areas (capital city and other major urban areas), rural zones and remote zones. We further classified these categories into two main groups: urban and rural and remote areas. Aboriginal and non-English speaking background status were not examined because of incomplete notification data.

Results

Incidence

For the five year period of surveillance, 12,272 cases of *Campylobacter* infection were reported in Queensland, representing an age-adjusted average annual rate of 82.3 cases per 100,000 population (crude rate, 82.5 per 100,000 population). Since 1992, *Campylobacter* notification rates in Queensland have been declining, whereas for Australia they have been increasing (Table 1). Queensland rates were significantly higher than the overall Australian rates between 1991 and 1993. However, for 1994 and 1995, notification rates in Queensland were significantly lower than the national figures.

Age and sex distribution

The age distribution for the five year period was found to be bimodal, with the highest incidence in the 0 - 4 years age group (251.3 per100,000 population) and a secondary peak occurring in the 20 - 24 and 25 - 29 years age groups (123.7 per 100,000 population and 112.6 per 100,000 popu-

Figure 1. Average annual notification rate of *Campylobacter* infection in Queensland, 1991 to 1995, by age group

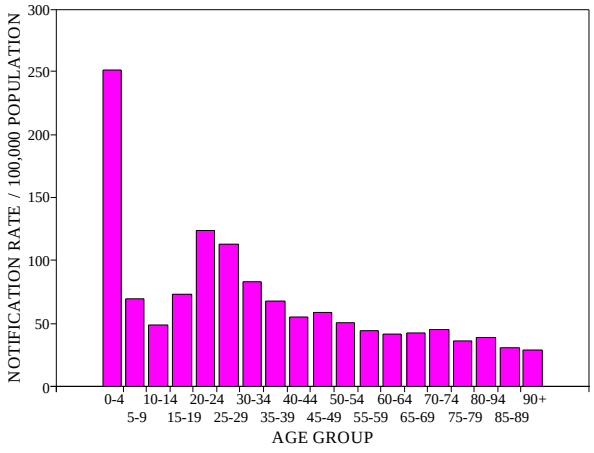


Table 2. Average annual notification rate of campylobacteriosis in Queensland, 1991 to 1995, for children aged 0 - 4 years

Age (months)	Total number of cases	Rate per 100,000 population
0-11	494	215.7
12-23	988	446.9
24-35	632	283.7
36-47	401	185.1
48-59	268	123.0

lation respectively) (Figure 1). The notification rate was higher in males (269.9 per 100,000 population) than females (225.1 per 100,000 population) in the 0 - 4 years age group, although this difference was not statistically significant. The rates for males and females in the 20 - 24 and 25 - 29 years age groups were very similar (118.5 versus 126.2, and 114.3 versus 108.9 respectively).

Seventy-one per cent of cases were found to occur in persons aged less than 35 years, and 23% of cases were in children under five years of age. For the 0 - 4 years age group, the highest incidence occurred in children aged 12 - 23 months (446.9 per 100,000 population), and this was significantly higher than the rate in each of the other 12-month age groups (Table 2). The annual *Campylobacter* notification rates in Queensland for the 0 - 4 years age group also declined, from 310.9 per 100,000 population in 1992 to 183.4 per 100,000 population in 1995. Fifty-two per cent of all notifications were male and 46% were female. For 2%, the gender was not available.

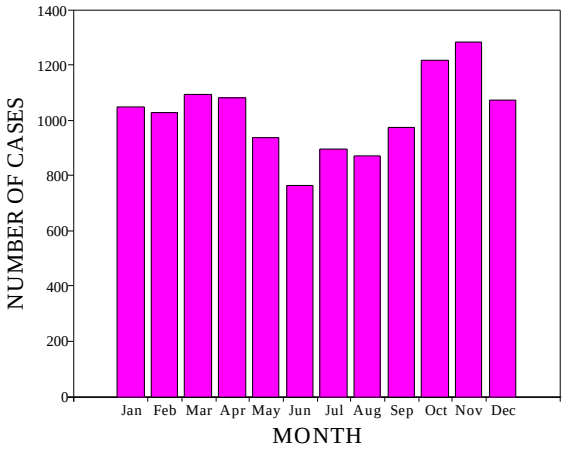
Seasonality

For the total five year period, a slight decline in incidence was seen during the winter months (Figure 2). The seasonal trend for *Campylobacter* notifications varied from year to year during this period. However, the numbers of cases were always elevated slightly during the warmer months, with peaks occurring either in spring or autumn.

Geographic distribution

Information on geographic distribution was available for 10,997 or 89.6% of the cases. The age-adjusted average annual notification rate was significantly higher in urban areas of Queensland compared with rural and remote areas (Table 3). Furthermore, the age-adjusted average annual notification rate was significantly higher in Brisbane and the surrounding metropolitan area compared with other major urban areas. This trend was seen in most age groups. For the 0 - 4 years age group, notification rates were similar in urban areas and in rural and remote areas.

Figure 2. Total number of notifications of *Campylobacter* infection in Queensland, 1991 to 1995, by month of disease



Socioeconomic status

Information on socioeconomic status was available for 10,533 or 85.8% of the cases. The highest age-adjusted average annual notification rate occurred in the higher socioeconomic areas of Queensland (Table 4). This was significantly greater than the notification rates found in both the average and lower socioeconomic areas. These differences were greatest among the 20 - 24 years age group (quintile 1, 192.8 per 100,000 population; quintile 5, 130.2 per 100,000 population). In contrast, there was a reverse trend in the 0 - 4 years age group, with the average annual notification rate being highest in the lower socioeconomic areas of Queensland. However, this rate was not significantly greater than the rates found in the average and higher socioeconomic areas.

Discussion

The notification rates for campylobacteriosis in Queensland and Australia have consistently been among the highest of all the notified communicable diseases. The reason for the decreasing trend in *Campylobacter* notification rates in Queensland since 1992 in contrast with the

Table 3. Average annual notification rates of campylobacteriosis, 1991 to 1995, by geographical area in Queensland

Area	Total Population		0 - 4 years age group	
	Total number of cases	Age-adjusted rate per 100,000	Total number of cases	Age-specific rate per 100,000
Brisbane metro	6091	102.1	1024	267.4
Other major urban	1838	85.5	358	248.7
Total urban	7929	97.9	1382	262.4
Rural and remote	3068	61.1	977	264.0

Table 4. Average annual notification rates of campylobacteriosis in Queensland, 1991 to 1995, by level of socioeconomic status

Quintile (socioeconomic status)	Total population		0 - 4 years age group	
	Total number of cases	Age-adjusted rate per 100,000	Total number of cases	Age-specific rate per 100,000
1 (high)	1909	109.7	285	250.7
2-4 (average)	6163	81.2	1459	261.9
5 (low)	2461	82.2	573	273.1

increasing trend for Australia is unknown. There are certain unquantifiable biases which affect notification data. These include the decision by the treating medical practitioner to collect a specimen for diagnostic testing (which is often dependent on the severity of the illness), the number of specimens collected, a patient's access to, and use of, health care services, and compliance with notification procedures by laboratories and medical practitioners. In addition, many infected patients with relatively mild symptoms do not seek medical care[19]. The actual incidence of *Campylobacter* infection in Australia is probably much greater than the currently reported rates. In England for example, the actual incidence has been conservatively estimated at about ten times the reported number[2].

The bimodal age distribution is characteristic of other developed countries, with the highest rate reported for the 0 - 4 years age group. In Queensland, the highest rate in this age group was for children aged 12 - 23 months, whereas in the United States of America the highest rate has been in children less than 12 months of age[4]. The reasons for the high notification rate in young children is unknown, but may be partly due to parents being more likely to seek medical care for these types of illnesses compared with adults. To account for this, Skirrow conducted a two year survey of *Campylobacter* isolates from five laboratories and calculated infection rates based on the number of faecal specimens tested[20]. When this denominator was used, the infection rate was highest in young adults (15 - 24 years old) and lowest in infants less than one year of age. This suggests that the higher notification rates found in our study for young children may be biased due to a higher sampling rate for this age group.

The high isolation rate in the 0 - 4 years age group followed by a large secondary peak in the 20 - 29 years age range is characteristic of *Campylobacter* infection. However, other bacterial gastrointestinal infections may have a similar age distribution but less pronounced peaks[2,4]. *Campylobacter* infections are a common cause of travellers' diarrhoea, and some investigators have suggested that the increased isolation rate seen in the 20 - 29 years age group is not unexpected, since young adults travel more than other age groups[3]. In Australia, recent data from the National Salmonella Surveillance Scheme would further support this argument, as notification rates for typhoid are highest in the 15 - 29 years age group and most infections are known to be acquired overseas[21].

It is generally accepted that in temperate zones, *Campylobacter* infection shows a seasonal distribution with a well defined summer peak[3,5,20]. In contrast, notifications in Queensland have a somewhat less marked seasonal trend, a feature which may be related to the State's mild winters. However, the seasonal distribution of notified cases for Australia overall shows a similar trend to Queensland, suggesting that factors other than climate may be involved[1]. It is possible that the observed seasonal trends overseas may be affected by other factors such as increased travel during summer holidays or increased consumption of poultry during the summer months, which may be less pronounced in Australia. Alternatively, the distinct seasonal trend seen in some European and North American countries may be related to other characteristics of these Northern Hemisphere countries. Imported cases of *Campylobacter* infection by travellers may be an important factor influencing seasonal trends in Australia.

The known risk factors associated with *Campylobacter* infection imply that the expected incidence of infection in rural communities would be higher than in residential areas, as has been reported in the United Kingdom[20] and New Zealand[5] (cattle, sheep, pigs and poultry are primary sources of human infection). Our results, however, show a significantly higher rate for urban areas compared with rural and remote areas. Similar results were also reported in a Norwegian study in which urban rates were more than twice those of rural areas[3]. However, this difference was attributed to a higher proportion of imported cases (from travel abroad) in urban areas. Overseas travel should therefore be considered as a contributing factor to the elevated rates in urban areas that were observed in our study. The recording of travel history data should be considered as part of the routine information collected on notifiable diseases. Another factor which is likely to contribute to this difference is that residents of urban areas, and in particular the capital city areas, are thought to have a higher sampling rate than residents in rural and remote areas. Furthermore, persons from non-metropolitan areas of Australia are, in general, significantly (10% to 20%) less likely to visit a medical practitioner than persons from metropolitan areas[22]. The similarity in geographic notification rates among the 0 - 4 years age group may be due to similar sampling rates or similar risk factors in this age group.

The data also showed that socioeconomic status may be related to the incidence of *Campylobacter* infection in Queensland. Reasons for the elevated notification rates

among persons from higher socioeconomic areas, with the exception of 0 - 4 year olds, is unknown. It is possible that elevated notification rates among higher socioeconomic groups are related to factors such as a higher rate of travel, greater use of medical services, or different food consumption patterns. Further investigations into the possible association between *Campylobacter* infection and socioeconomic status would be of interest.

In conclusion, this review of *Campylobacter* infection between 1991 and 1995 has shown that: the incidence in Queensland was highest in children aged 12 - 23 months; the distinct summer peak in incidence of the disease that is reported in other developed countries was not seen in Queensland; *Campylobacter* infection was reported more frequently from urban areas and from high socioeconomic areas of Queensland; and that factors which influence notification rates in the general population do not necessarily have the same influence on the 0 - 4 years age group. Case control studies of *Campylobacter* infection in young children are warranted to identify age-specific risk factors. These factors would enable the development of interventions to reduce the incidence of infection.

Acknowledgements

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OVERSEAS BRIEFS

Source: World Health Organization

Ebola haemorrhagic fever, Gabon

The government of Gabon and the World Health Organization (WHO) have confirmed that a virus of Ebola type is responsible for the outbreak of haemorrhagic fever in the Booué region of north-eastern Gabon. As at 19 October, the total number of cases was 19, of which 11 were fatal; 86 contacts remained under surveillance. According to the Ministry of Health of Gabon, the first case would have occurred on July 24 in a hunter who probably became infected in the forest and who later died.

An isolation ward has been prepared in Booué Hospital. Samples for laboratory investigation have been collected from patients and close contacts. The first analysis, done with locally available reagents, confirmed that an Ebola-like virus is responsible for the outbreak, but further tests on blood and tissue samples collected from patients are underway at the WHO Collaborating Centre for Haemorrhagic Fevers, the Centers for Disease Control and Prevention in Atlanta, United States of America. An education and information campaign for health workers has been undertaken in regions where the epidemic has occurred and in bordering areas where there have been no reports of suspected cases.

An International Committee for Technical and Scientific Coordination was established in Gabon with the collabo-

ration of the WHO. It includes experts from the Gabonese Ministry of Health, the International Medical Research Centre in Franceville, Gabon, Coopération Française and WHO.

The Ebola virus is one of the most pathogenic viral agents known to man, causing death in 80% or more of infected cases, but its natural host is still unknown. Scientific investigations have just begun in Côte d'Ivoire, in the Tai forest, to identify the natural reservoir of the virus. This research, which is coordinated by WHO, is being undertaken by scientific teams from Belgium, Canada, France and the United States of America, with the collaboration of scientists from the United Kingdom.

The primary mode of transmission of the virus is contact with contaminated blood and secretions of body fluids. Contaminated needles and syringes were the cause of transmission in previous cases in Zaire. The virus is not easily transmitted however, and requires intimate contact with an infected person, such as close nursing without protective clothing, or with contaminated injection equipment.

Last February, an outbreak of Ebola haemorrhagic fever killed 21 persons, from a total of 37 cases, in the same province of Gabon (Ogooué-Ivindo).

Cholera, Africa

Guinea-Bissau reported 143 cases with 7 deaths from 6 to 14 October in the districts of Bissau and Gabu. Senegal and Togo also reported cholera cases in the past week.

Dengue, India

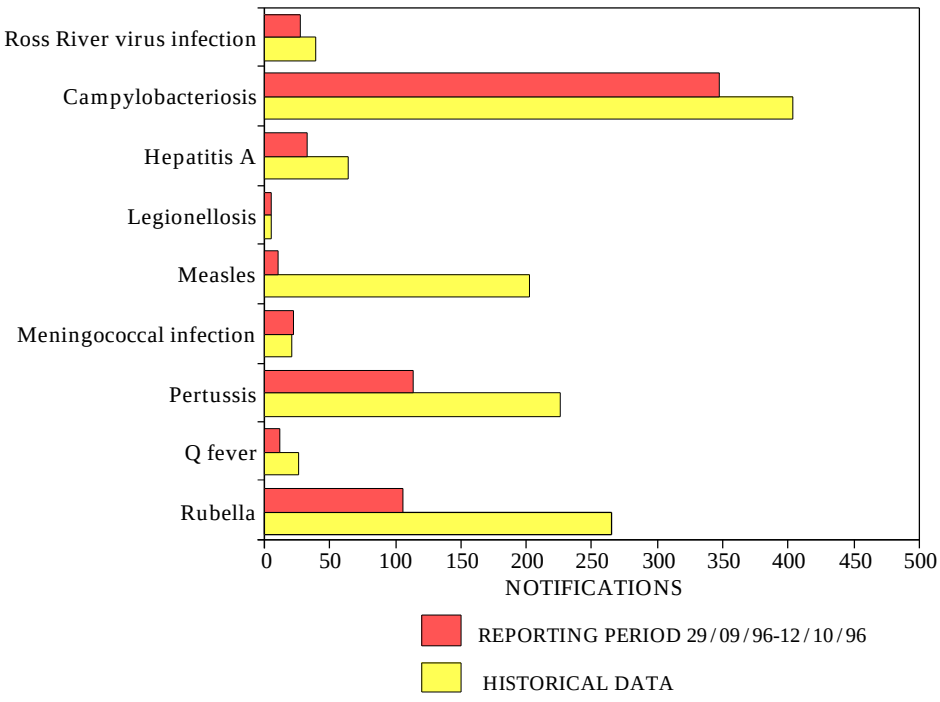
The National Institute of Communicable Diseases, Delhi reported 5,930 cases of dengue and dengue haemorrhagic fever up to 22 October 1996. There were 251 deaths. Reports came from hospitals in Delhi. Dengue virus type 2 has been isolated.

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 more than 40 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.

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.

Reporting period 29 September to 12 October 1996

There were 1,655 notifications received for this two-week period (Tables 1, 2 and 3). The numbers of reports for selected diseases have been compared with average data for this period in the previous three years (Figure 1).

Eleven notifications of **measles** were received in this reporting period. The number of cases remains low (Figure 2).

Pertussis was reported for 151 persons this period. Included were 7 apparent clusters of 3 or more cases in postcode regions of New South Wales, Victoria, Queensland and South Australia. While the number of cases has risen slightly in recent months, it remains below the level reported for the same period in the years 1993 to 1995 (Figure 3). For the year to date the male:female ratio was 1:1.4 and most reports (46%) were for those under the age of 15 years (Figure 4).

One hundred and six notifications of **rubella** were received this period. The number of cases has risen slightly in recent weeks but remains low for the time of year (Figure 5).

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

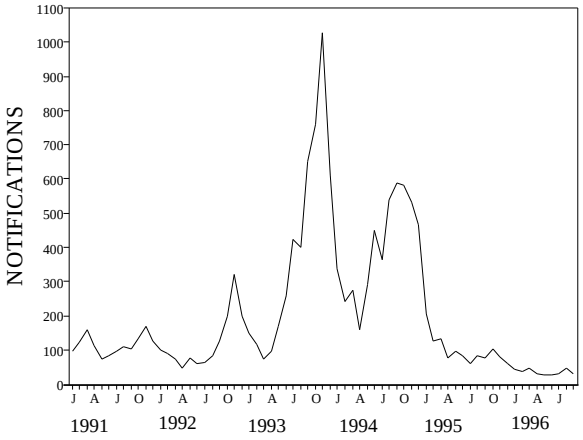
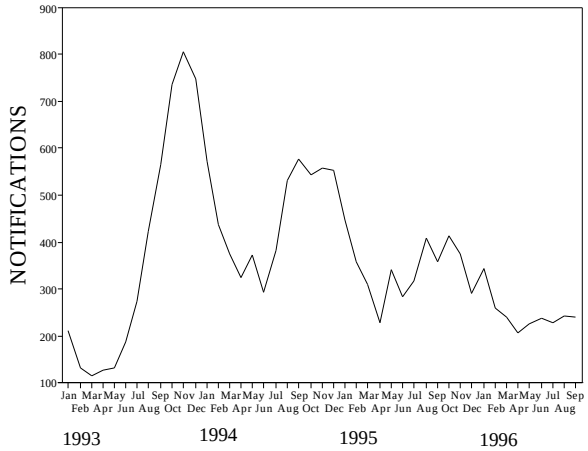


Figure 3. Pertussis notifications, 1993 to 1996, by month of onset



The number of notifications of **hepatitis A** continues to fall after peaking in January (Figure 6). For the year to date more males have been reported than females, the male:female ratio is 2.2:1.

Figure 4. Pertussis notifications, 1996, by age group and sex

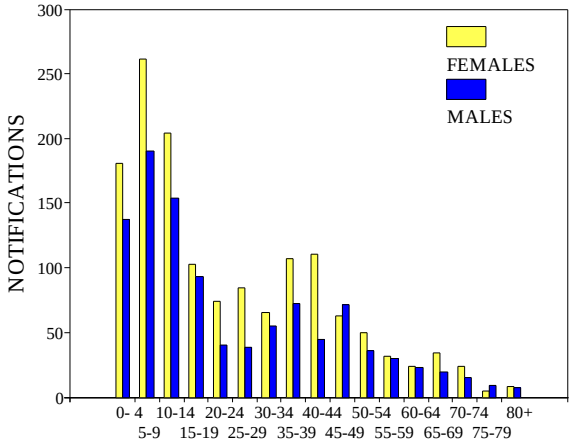


Figure 5. Rubella notifications, 1994 to 1996, by month of onset

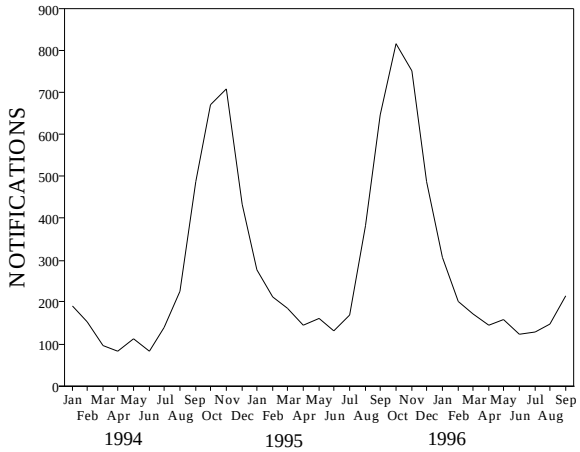


Figure 6. Hepatitis A notifications, 1994 to 1996, by month of onset

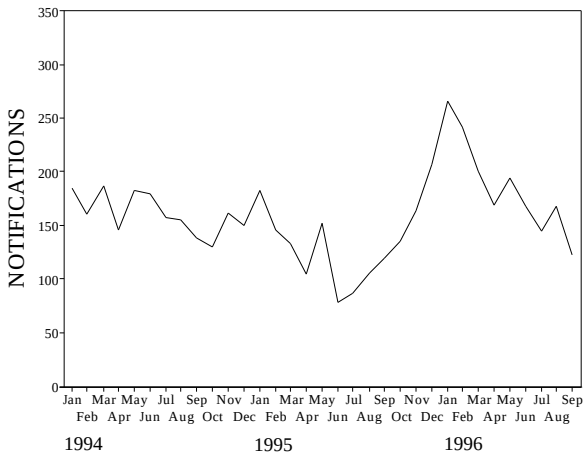


Table 1. Notifications of diseases preventable by vaccines recommended by the NHMRC for routine childhood immunisation, received by State and Territory health authorities in the period 29 September to 12 October 1996

DISEASE[1]	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
Diphtheria	0	0	0	0	0	0	0	0	0	0	0	0
Haemophilus influenzae b infection	0	0	0	1	0	0	0	0	1	2	46	55
Measles	1	3	0	2	0	1	4	0	11	42	363	1119
Mumps	0	1	0	NN	0	0	1	1	3	6	93	118
Pertussis	0	39	0	29	37	0	44	2	151	171	2490	3293
Rubella	1	15	0	53	16	0	11	10	106	385	1907	2600
Tetanus	0	0	0	0	0	0	0	0	0	0	1	3

NN Not Notifiable.

1. No notifications of poliomyelitis have been reported since 1986.
2. Totals comprise data from all States and Territories. Cumulative figures are subject to retrospective revision, so there may be discrepancies between the number of new notifications and the increment in the cumulative figure from the previous period.

Table 2. Notifications of other diseases received by State and Territory health authorities in the period 29 September to 12 October 1996

DISEASE[1]	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 (NEC)[3,4]	0	0	1	0	0	0	0	1	2	4	87	60
Barmah Forest virus infection	0	2	-	13	0	0	0	-	15	12	710	660
Ross River virus infection	0	3	4	18	0	0	1	1	27	25	7499	2416
Dengue	0	0	0	0	0	-	0	0	0	2	30	25
Campylobacteriosis[5]	8	-	5	95	99	9	75	56	347	423	9024	8122
Chlamydial infection (NEC)[6]	7	NN	2	114	0	8	53	30	214	281	5761	4864
Donovanosis	0	NN	0	0	NN	0	0	0	0	2	38	60
Gonococcal infection[7]	1	17	9	32	0	0	9	29	97	116	2986	2444
Hepatitis A	1	20	0	7	0	0	0	5	33	60	1791	1194
Hepatitis B incident	0	1	0	2	0	1	0	0	4	7	163	258
Hepatitis C incident	0	0	0	-	0	-	-	-	0	1	23	63
Hepatitis C unspecified	10	NN	0	78	NN	10	77	22	197	398	7168	7515
Hepatitis (NEC)	0	0	0	0	0	0	0	NN	0	0	17	10
Legionellosis	0	2	0	0	1	0	0	2	5	4	142	137
Leptospirosis	0	0	0	3	0	1	1	0	5	4	175	95
Listeriosis	0	3	0	0	0	0	1	1	5	1	55	49
Malaria	2	7	0	0	0	0	8	0	17	11	677	513
Meningococcal infection	0	9	3	6	0	1	3	0	22	15	330	307
Ornithosis	0	NN	0	0	0	0	1	0	1	7	54	96
Q fever	0	5	0	7	0	0	0	0	12	13	409	360
Salmonellosis (NEC)	2	24	6	42	4	5	18	18	119	152	4457	4777
Shigellosis[5]	0	-	2	7	3	0	0	3	15	9	521	606
Syphilis	1	30	2	9	0	0	0	1	43	78	1182	1499
Tuberculosis	0	15	0	2	3	0	15	0	35	45	843	799
Typhoid[8]	0	1	0	0	0	0	0	0	1	2	72	59
Yersiniosis (NEC)[5]	0	-	0	6	1	0	0	0	7	10	197	256

1. For HIV and AIDS, see Tables 4 and 5. For rarely notified diseases, see Table 3 .
2. Totals comprise data from all States and Territories. Cumulative figures are subject to retrospective revision so there may be discrepancies between the number of new notifications and the increment in the cumulative figure from the previous period.
3. Tas: includes Ross River virus and dengue.
4. NT, Vic and WA: includes Barmah Forest virus.
5. NSW: only as 'foodborne disease' or 'gastroenteritis in an institution'.
6. WA: genital only.
7. NT, Qld, SA and Vic: includes gonococcal neonatal ophthalmia.
8. NSW, Vic: includes paratyphoid.

NN Not Notifiable.

NEC Not Elsewhere Classified.

- Elsewhere Classified.

Table 3. Notifications of rare[1] diseases received by State and Territory health authorities in the period 29 September to 12 October 1996

DISEASES[2]	Total this period	Reporting States or Territories	Year to date 1996
Brucellosis	0		25
Chancroid	0		1
Cholera	0		4
Hydatid infection	1	Vic	32
Leprosy	1	WA	9

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

HIV and AIDS Surveillance

National surveillance for HIV disease is coordinated by the National Centre in HIV Epidemiology and Clinical Research (NCHECR), in collaboration with State and Territory health authorities and the Commonwealth of Australia. Cases of HIV infection are notified to the National HIV Database on the first occasion of diagnosis in Australia, by either the diagnosing laboratory (ACT, New South Wales, Tasmania, Victoria) or by a combination of laboratory and doctor sources (Northern Territory, Queensland, South Australia, Western Australia). Cases of AIDS are notified through the State and Territory health authorities to the National AIDS Registry. Diagnoses of both HIV infection and AIDS are notified with the person's date of birth and name code, to minimise duplicate notifications while maintaining confidentiality.

Tabulations of diagnoses of HIV infection and AIDS are based on data available three months after the end of the reporting interval

Table 4. New diagnoses of HIV infection, new diagnoses of AIDS and deaths following AIDS occurring in the period 1 to 31 May 1996, by sex and State or Territory of diagnosis

		ACT	NSW	NT	Qld	SA	Tas	Vic	WA	TOTALS FOR AUSTRALIA			
										This period 1996	This period 1995	Year to date 1996	Year to date 1995
HIV diagnoses	Female	0	2	0	0	0	0	0	0	2	14	35	42
	Male	2	33	1	17	3	0	12	0	68	71	310	346
	Sex not reported	0	0	0	1	0	0	0	0	1	1	4	7
	Total[1]	2	35	1	18	3	0	12	0	71	86	349	397
AIDS diagnoses	Female	0	1	0	0	0	0	0	0	1	3	3	16
	Male	0	12	0	6	2	0	1	2	23	69	161	314
	Total[1]	0	13	0	6	2	0	1	2	24	72	164	331
AIDS deaths	Female	0	1	0	1	0	0	1	0	3	4	9	19
	Male	0	7	0	3	3	0	4	1	18	51	146	272
	Total[1]	0	8	0	4	3	0	5	1	21	56	155	292

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

Table 5. Cumulative diagnoses of HIV infection, AIDS and deaths following AIDS since the introduction of HIV antibody testing to 31 May 1996, by sex and State or Territory

		ACT	NSW	NT	Qld	SA	Tas	Vic	WA	AUSTRALIA
HIV diagnoses	Female	16	565	4	98	44	4	167	73	971
	Male	170	10061	83	1604	571	70	3393	760	16712
	Sex not reported	0	2049	0	1	0	0	42	0	2092
	Total[1]	186	12682	87	1708	615	74	3611	835	19798
AIDS diagnoses	Female	5	134	0	28	18	2	47	17	251
	Male	75	3824	26	656	276	32	1341	292	6522
	Total[1]	80	3968	26	686	294	34	1395	311	6794
AIDS deaths	Female	2	101	0	23	13	2	37	11	189
	Male	50	2701	20	457	192	21	1063	215	4719
	Total[1]	52	2808	20	482	205	23	1106	227	4923

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

indicated, to allow for reporting delay and to incorporate newly available information. More detailed information on diagnoses of HIV infection and AIDS is published in the quarterly Australian HIV Surveillance Report, available from the National Centre in HIV Epidemiology and Clinical Research, 376 Victoria Street, Darlinghurst NSW 2010. Telephone: (02) 332 4648 Facsimile: (02) 332 1837.

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

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 40 and 41 ending 6 and 13 October respectively are included in this issue of *CDI* (Table 6). The consultation rate for influenza-like illness has declined to low levels from peak rates of 28 to 31 per 1,000 consultations in late June and July. There has been no appreciable change in the consultation rate for gastroenteritis over the last three months. Consultation rates for chickenpox remain higher than the rates for August and early September. Very few cases of rubella, measles and pertussis have been reported during the last six reporting weeks.

Table 6. Australian Sentinel Practice Research Network reports, weeks 40 and 41, 1996

Condition	Week 40, to 6 October 1996		Week 41, to 13 October 1996	
	Reports	Rate per 1,000 encounters	Reports	Rate per 1,000 encounters
Influenza	45	6.8	23	4.0
Rubella	2	0.3	1	0.2
Measles	0	0	0	0
Chickenpox	11	1.7	13	2.3
Pertussis	0	0	2	0.3
Gastroenteritis	101	15.3	105	18.3

Sentinel Chicken Surveillance Programme

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

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5. Department of Health and Community Services, Darwin, NT.

Sentinel chicken flocks are used to monitor flavivirus activity in Australia. The main viruses of concern are Murray Valley encephalitis (MVE) and Kunjin that cause the potentially fatal disease Australian encephalitis in humans. These viruses are enzootic in parts of the north-east Kimberley region of Western Australia and the Northern Territory but are epizootic in other areas of the Kimberley and in north Queensland. MVE virus is also responsible for occasional severe epidemics of Australian encephalitis in eastern Australia. The most recent was in 1974 when there were 13 fatalities and cases were reported from all mainland States. Since then, 48 cases have been reported and all but one of these were from the north of Australia.

Since 1974, a number of sentinel chicken flocks have been established in Australia to provide an early warning of increased MVE virus activity. These programs are supported by individual State health departments. Each State has a contingency plan which will be implemented if one or more chickens in a flock seroconverts to MVE virus.

Currently 22 flocks are maintained in the north of Western Australia, eight in the Northern Territory and ten in Victoria. The flocks in Western Australia and the Northern Territory are tested all year round but those in Victoria are tested only from November to March, during the main MVE risk season. New South Wales and Queensland have previously used these sentinel systems to monitor flavivirus activity but have not maintained flocks during 1996.

Results are coordinated by the Arbovirus Laboratory in Perth and reported bimonthly.

Sentinel chicken serology was carried out for 17 of the 22 flocks in Western Australia in July and August 1996. There were no seroconversions during this period.

There were no seroconversions during the 1995-96 wet season in the Kimberley region of Western Australia. This was the first wet season with no evidence of flavivirus activity in the north of Western Australia since surveillance began using sentinel chicken flocks in 1982.

Five flocks of sentinel chickens from the Northern Territory were also tested in July and August. There were no seroconversions to flaviviruses.

LabVISE

The Virology and Serology Reporting Scheme, LabVISE, is a sentinel reporting scheme. Twenty-one laboratories contribute data on the laboratory identification of viruses and other organisms. Data are collated and published in Communicable Diseases Intelligence each fortnight. These data should be interpreted with caution as the number and type of reports received is subject to a number of biases. For further information, see CDI 1996;20:9-12.

There were 680 reports received in the *CDI* Virology and Serology Reporting Scheme this period (Tables 7 and 8).

Laboratory reports of **parvovirus** in July and August have been the highest recorded since 1992 (Figure 7). In the last fortnight, 11 reports were received, with diagnosis by IgM detection (10) and nucleic acid detection (1).

There were 49 reports of ***Bordetella pertussis*** this period, all were from Victoria. Diagnosis was by IgA detection (47) and antigen detection (2). Eighteen reports were for children aged 5-14 years and one report was for a child aged under 12 months.

Reports of **parainfluenza virus type 3** increased in September but remain below the number reported for the same period in 1995 (Figure 8). In the last fortnight, 51 reports were received. Diagnosis was by antigen detection (23), virus isolation (22), single high titre (5) and four-fold rise in titre (1).

There were 34 reports of **influenza A** in the last fortnight, 22 of which were from Western Australia. Of the total, 53% (18) were for patients 65 years of age or older.

Figure 7. Parvovirus laboratory reports, 1992 to 1996, by month of specimen collection

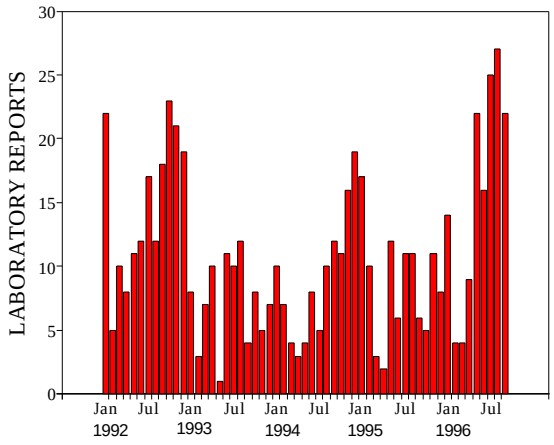

Figure 8. Parainfluenza virus type 3 laboratory reports, 1994, 1995 and 1996, by month of specimen collection

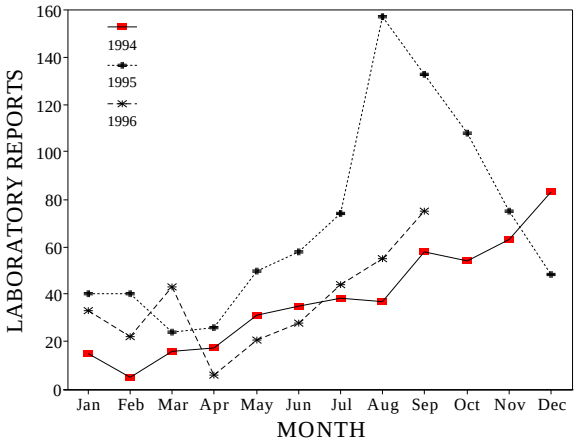

Table 7. Virology and serology laboratory reports by State or Territory[1] for the reporting period 3 to 16 October 1996, historical data[2], and total reports for the year

	State or Territory[1]							Total this fortnight	Historical data[2]	Total reported this year
	ACT	NSW	NT	Qld	SA	Vic	WA			
MEASLES, MUMPS, RUBELLA										
Measles virus						2		2	33.8	44
Mumps virus						1		1	2.8	33
Rubella virus							10	10	72.2	477
HEPATITIS VIRUSES										
Hepatitis A virus			3			2	3	8	14.7	352
ARBOVIRUSES										
Ross River virus			1				1	2	15.0	3,093
Barmah Forest virus			1				2	3	5.8	187
ADENOVIRUSES										
Adenovirus type 2						1		1	2.5	24
Adenovirus type 7						2		2	.7	23
Adenovirus type 8						1		1	.3	5
Adenovirus type 40							1	1	.0	29
Adenovirus not typed / pending		10		12		8	19	49	41.7	1,171
HERPES VIRUSES										
Cytomegalovirus		2		7		7	9	25	63.8	1,328
Varicella-zoster virus		1				14	9	24	37.7	996
Epstein-Barr virus		5	4			11	25	45	60.8	1,625
OTHER DNA VIRUSES										
Molluscum contagiosum							2	2	.2	5
Poxvirus group not typed						1		1	.2	3
Parvovirus	2	1				7	1	11	3.2	165
PICORNA VIRUS FAMILY										
Coxsackievirus A16						1		1	.0	5
Coxsackievirus B2		1				3		4	.2	7
Coxsackievirus B4						2		2	.3	5
Coxsackievirus B5						1		1	.0	8
Echovirus type 7						2		2	.0	14
Rhinovirus (all types)		3		8		8	8	27	33.0	618
Enterovirus not typed / pending		1		6		4	10	21	35.5	741
ORTHO / PARAMYXOVIRUSES										
Influenza A virus		2	3			7	22	34	21.0	1,446
Influenza B virus							5	5	9.0	51
Parainfluenza virus type 1		1		1			1	3	.3	300
Parainfluenza virus type 3		3		13		6	29	51	32.5	525
Parainfluenza virus typing pending							3	3	1.8	18
Respiratory syncytial virus	1	17		3		32	18	71	113.8	3,929
Paramyxovirus (unspecified)						2		2	.8	18
OTHER RNA VIRUSES										
HTLV-1							1	1	.2	8
Rotavirus		57			1	30	8	96	134.0	1,379
Small virus (like) particle						1		1	2.0	14
OTHER										
Chlamydia trachomatis not typed		3	18			6	34	61	86.0	3,079
Mycoplasma pneumoniae		10				21	16	47	26.2	634
Coxiella burnetii (Q fever)		3					1	4	7.8	155
Bordetella pertussis						49		49	33.0	505
Legionella longbeachae							1	1	.7	14
Cryptococcus species							2	2	.2	8
Schistosoma species			1			1	1	3	5.2	228
TOTAL	3	120	31	50	1	233	242	680	898.8	23,484

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 8. Virology and serology laboratory reports by contributing laboratories for the reporting period 3 to 16 October 1996

STATE OR TERRITORY	LABORATORY	REPORTS
New South Wales	Institute of Clinical Pathology & Medical Research, Westmead	26
	Royal Alexandra Hospital for Children, Camperdown	51
	South West Area Pathology Service, Liverpool	40
Queensland	State Health Laboratory, Brisbane	50
Victoria	Microbiological Diagnostic Unit, University of Melbourne	6
	Monash Medical Centre, Melbourne	13
	Royal Children's Hospital, Melbourne	153
	Victorian Infectious Diseases Reference Laboratory, Fairfield Hospital	67
Western Australia	PathCentre Virology, Perth	137
	Princess Margaret Hospital, Perth	61
	Royal Perth Hospital	11
	Western Diagnostic Pathology	65
TOTAL		680

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

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