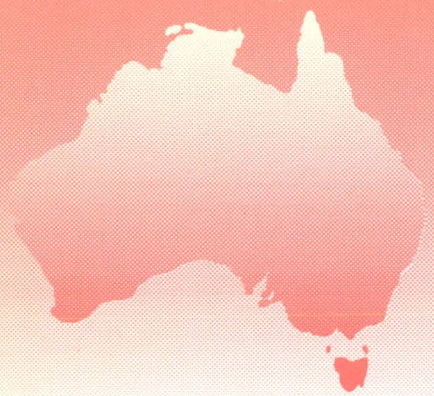

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COMMUNICABLE DISEASES NETWORK-AUSTRALIA
A National Network for Communicable Diseases Surveillance

HEPATITIS A OUTBREAK IN A PRESCHOOL IN EASTERN SYDNEY

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On 17 May 1993 the Eastern Sydney Public Health Unit was notified of a case of hepatitis A in a three year old girl. The child had earlier been admitted to hospital with gastrointestinal symptoms and had been discharged without a definitive diagnosis. Onset of jaundice the day following discharge prompted testing for hepatitis A virus (HAV) IgM antibody which was positive. No household contacts of the case were affected, but it was noted that the child attended a local preschool.

The next day, notification was received of a second case of serologically confirmed hepatitis A, in a seven year old child with onset of jaundice on 10 May. No illness was reported in the child's household contacts but a younger sibling attended the same preschool as the index case. We advised serological testing of the sibling, and this proved positive for HAV IgM.

The preschool director was then contacted by the Public Health Unit and informed that an outbreak of hepatitis A appeared to be centred on the preschool. Twenty-five children, including 16 Aboriginal children, were enrolled in the preschool in the care of five staff members. All children were toilet trained, although 'accidents' were not uncommon. Neither the staff nor the children attending had reported recent hepatitis or jaundice. Members of the Public Health Unit visited the preschool and provided information to staff regarding the importance of hygiene, particularly handwashing, in preventing the spread of hepatitis A. All staff were advised to have normal human immunoglobulin as soon as possible. An information letter and consent form for serological testing was distributed to parents, and blood was collected from the 16 children with parental consent on 25 May 1993.

It was found that one of the children absent from the preschool on the day of blood collection had just been admitted to The Prince of Wales Children's Hospital with unexplained fever, abdominal pain, vomiting and haematuria. Serology performed after discharge by the child's family doctor was reported as positive for HAV IgM. Altogether, 19 preschool children were tested, of whom six were HAV IgM positive, a further six were HAV IgG positive but HAV IgM negative and seven had no detectable antibodies to HAV. Thus, 63% had serological evidence of either recent or past HAV infection.

Two further cases came to light in the ensuring weeks and provided further evidence of the extent of the outbreak. On 25 June 1993 a nurse who worked in The Prince of Wales Children's Hospital and who remembered looking after the child who was absent from the preschool on the day of blood collection, developed jaundice and was found to be HAV IgM positive.

In mid-June the six year old asymptomatic sister of an HAV IgM negative, HAV IgG positive preschool child was reported by the family doctor to be HAV IgM positive. Both these children had been found to be HAV IgG negative in late March following contact with a child with hepatitis A unrelated to this outbreak and had been given immunoglobulin. It appears that the brother at the preschool had seroconverted to HAV IgG in April or May, and that HAV IgM had appeared and then become undetectable in this interval. We were uncertain whether these children were infected during this outbreak or as a result of the earlier contact.

The hepatitis A outbreak described involved 15 cases, including the two siblings just described and assuming that the brother and the other five HAV IgG positive, HAV IgM negative children had been infected during the outbreak in the two months prior to receipt of the first notification.

Four preschool children and one household contact had subclinical infection, two more preschool children had anicteric illness compatible with hepatitis and two contacts (one household and one nosocomial) had typical icteric hepatitis.

Outbreak management involved emphasis on hygiene and recommendations for use of normal immunoglobulin for staff, household contacts and susceptible or untested preschool children. Apart from the instance of nosocomial transmission, no further clinical cases were reported in association with the preschool.

Comment

In industrialised societies where rates of HAV infection are low, children are generally susceptible and constitute a large proportion of reported HAV infections. However, hepatitis A is often unrecognised in young children, as over 80% of infections in 1-2 year olds, and 50% of infections in 3-4 year olds are asymptomic[1]. Of the cases reported above in preschool attendees, all were anicteric and all but two were asymptomatic, whilst only older contacts presented with jaundice.

The significance of child care in the transmission of hepatitis A was suggested by a series of studies conducted in Phoenix, Arizona. When all reported cases in the community were followed up, it was found that 42% of them occurred in children attending child care, child-care staff or their household contacts[1]. Moreover, a campaign of immunoglobulin administration to all children and employees in a child-care centre when a single case was reported from that centre resulted in a 75% decline in the total number of reported cases in the community[2]. Clinical attack rates were higher in

child-care staff and adult household contacts than in children in child care[1].

Hepatitis A is readily spread from person to person by the faecal-oral route. For this reason, child-care centres with many children who are not yet toilet trained are most likely to be involved in outbreaks of hepatitis A[3]. Hanna recently reported a cluster of cases associated with a child-care centre in far north Queensland which catered for toddlers in nappies and older children[4]. Six children and nine contacts suffered symptomatic infections, whilst several more of the children in the centre probably developed subclinical infections (one was confirmed HAV IgM positive, and at least two more appeared to transmit the infection to family contacts). If serological testing had been performed on asymptomatic children, it is likely that further cases would have been identified. As in the present report, no cases were reported in carers, despite involvement of staff in nappy changing. In both outbreaks, mass use of immunoglobulin may have interrupted transmission, although it is probable that in the eastern Sydney outbreak most transmission occurred prior to its recognition.

References

1. Hadler SC, Webster HM, Erben JJ, Swanson JE, Maynard JE. Hepatitis A in day-care centers. *NEJM* 1980;**302**:1222-1227.
2. Hadler SC, Erben JJ, Matthews D, Starko K, Francis DP, Maynard JE. Effect of immunoglobulin on hepatitis A in day-care centers. *JAMA* 1983;**249**:48-53.
3. Hadler SC, Erben JJ, Francis DP, Webster HM, Maynard JE. Risk factors for hepatitis A in day-care centers. *J Infect Dis* 1982;**145**:255-261.
4. Hanna J. Hepatitis A in a child day-care centre. *Comm Dis Intell* 1993;**17**:73-74.

CDI editorial comment

Preventing spread of infectious diseases in child-care settings is the subject of a new book being published by the National Health and Medical Research Council and Commonwealth Department of Human Services and Health. *Staying Healthy in Child-care* will be available from Commonwealth Government Bookshops from the end of March 1994. The book includes information on how to minimize spread of disease such as hepatitis A as well as general information on individual diseases. It recommends hepatitis A immunisation for child-care workers who care for children under two years of age.

PERSISTING HEPATITIS A INFECTION IN A CENTRAL QUEENSLAND TOWN

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From April 1992 to May 1993, 43 serologically confirmed notifications of hepatitis A were received by Queensland Health from a small central Queensland town of population 3162. The first notification was of a single case in April 1992 and the initial series of notifications was sporadic, but from August 1992 the notifications constituted a more obvious propagated outbreak.

The Regional Health Authority became involved from October 1992 as a response to the notifications received and also because of mounting local concern and speculation as to the source of the outbreak. Local health workers began a public awareness campaign to increase both understanding of the mode of spread of hepatitis A, and awareness of the specific hygiene measures which could be used to contain the outbreak. Schoolchildren were particularly targeted and soap (previously not available) was placed in the school toilets. Throughout this period local practitioners administered gamma globulin to case contacts where this was considered appropriate.

Environmental Health Officers from the Regional Health Authority involved in this campaign became concerned when notifications continued to be received in increasing numbers. In November these Officers contacted the Communicable Diseases Branch of Queensland Health to request support. For various administrative reasons, investigation of the outbreak was not commenced until mid-January 1993, by which time 35 notifications had been received.

The investigation took the form of a case series study performed simultaneously with an investigation of the general ecology of the town and its surrounds. The case series investigation aimed to address three potential contributory factors: source infection in households with inadequate inter-personal hygiene measures, source infection in a food handler, and contamination of the environment related to ineffective human waste disposal systems. The socioeconomic disadvantage of the particular community was well recognised and any one or more of these factors could have been active. A case-control study, identification of cases that had not been notified, and a study of seroprevalence were considered unnecessary by the Regional Health Authority.

Using a prepared questionnaire which addressed the study factors the thirty-two cases who could be contacted were interviewed and asked about the number of persons in their household, the duration and nature

Figure 1. Hepatitis A notifications in a central Queensland town, April 1992 to October 1993, by month

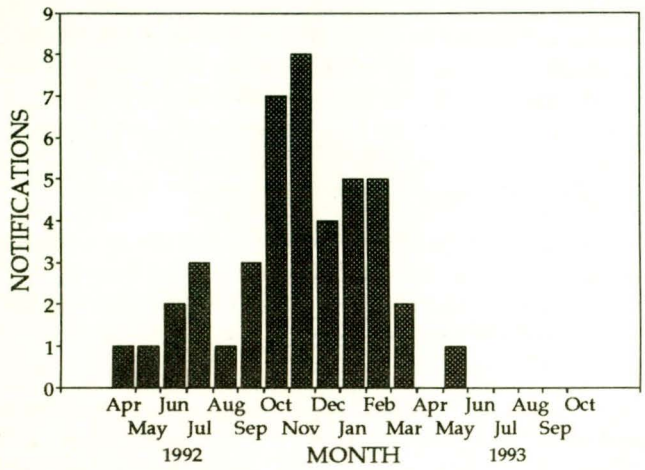
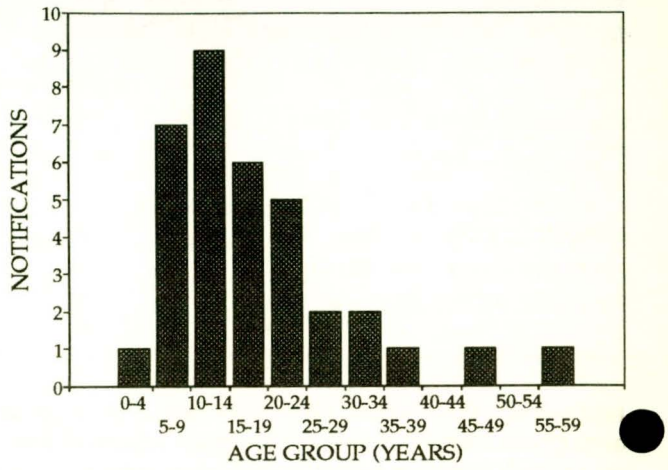


Figure 2. Hepatitis A notifications in a central Queensland town, by age group



of their illness, their occupation and potential risk-related exposures.

They were also questioned regarding their knowledge of the nature of hepatitis A, how it is spread and how its spread can be prevented.

The ecological investigation constituted an inspection of sanitation facilities and food handling practices in the town.

Investigation results

A total of 43 cases was documented in the outbreak, which peaked in the period October 1992 to February 1993 (Figure 1). These cases represented an annual crude attack rate of 12.6 per 1000 persons. The majority of those affected were schoolchildren and young adults in the age range 5 to 24 years (Figure 2).

The questionaires revealed that most cases were persons who lived in dwellings with two or more other persons; 16 cases were from households of six or more (Table 1). The mean duration of incapacity was 18 days and the median 14 days, but the range was from one to more than six weeks. Anorexia was reported by all cases, and abdominal discomfort, nausea, jaundice and fever by most (Table 2). One case was a chef.

Several possible risk-related exposures were identified in the interviews. Twenty-four had shared food or drink with a hepatitis A case (Table 3). Only 16 (50%) of the interviewed cases knew what hepatitis A was, 25 (78%) knew how it was spread and 16 (50%) knew how it was prevented, although all had recently had the disease, had consulted a doctor regarding their illness, and all children who had been attending school had been targeted by an awareness campaign in the schools prior to being questioned.

The inspection of sanitation facilities and food handling practices revealed a number of areas of concern: misuse of an unapproved parcel of land as a sanitary depot for disposal and burial of nightsoil, non-compliance with conditions of authorisation of the refuse tip, 25% of houses had poorly-sited, ill-repaired, or poorly-maintained septic tanks, 20% of house yards provided

Table 1. Hepatitis A cases in a central Queensland town, by size of household

Persons in household	Cases
1	1
3	5
4	5
5	5
6	11
7	2
10	2
15	1

Table 2. Hepatitis A cases in a central Queensland town, by reported symptoms

Sympton	Cases(%)
Anorexia	100
Abdominal discomfort	94
Nausea	94
Jaundice	84
Fever	81
Diarrhoea	7
Vomiting	4
Dark urine	4
Myalgia/arthralgia	4

Table 3. Hepatitis A cases in a central Queensland town, by potential risk-related exposures

Potential risk-related exposure	Yes	No
Shared food or drink with a hepatitis A case	24	8
Septic toilet	31	1
Septic pumped in the last 3 years	13	18
Swam in river	9	23
Household member attended day-care	4	28
Bought grocies locally	28	4
Bought take-aways locally	25	7
Household member used school tuckshop	15	17

harbourage for vermin, there was heavy fly-breeding at the refuse tip and the unapproved nightsoil depot, no formal drinking water sampling program existed, and there was poor understanding by food handlers of their responsibilities and obligations under the regulations.

Comment

The outbreak described above is typical of person to person propagated spread arising from an initial infected sufferer. Actual numbers affected will never be known but a symptomatic:asymptomatic ratio of 1:4 is considered conservative[1]. Our interviews suggested that the first patient notified acquired the infection from an itinerant out-of-town visitor who stayed with the family for a short period immediately before that person's own illness became apparent. Subsequently all but three of those notified could be traced as having close contact with another person who suffered with hepatitis A. In particular, a child's birthday party attended by one of the children who was known to be infectious at the time presented an opportunity for multiple transmissions. All early cases in school children were from the State primary school. The age pattern of those notified corresponds to that seen in communities where the population is largely vulnerable[2].

The frequency of symptoms and signs described is as would be expected in a predominantly childhood presentation except for dark urine and pale stools which were reported less commonly than experience would suggest[4].

A case series is useful for formulating a hypothesis as to causation but provides no information about statistical association[3]. Our study presents some interesting facts which cannot be subjected to analysis for the above reason.

Specific questioning of cases and the results of the ecologic investigation did not provide evidence for any conclusion with regard to the three potential contributory factors. The benefit of this aspect of the study lay in identification of areas of public health concern. We were presented with a community which had a number of important public health problems, any of which could precipitate a serious outbreak of disease. The challenge was to identify potential solutions. The issue was further compounded by the lack of resources facing the community and its inability to address the issues, an inability which will only grow as the rate base of the area is eroded by a continuing fall in income. At what stage should outside agencies become involved, and should disease notifications be part of the mechanism which triggers this involvement? Patterns of best-practice suggest that addressing these questions is a fundamental task of any properly-organised surveillance program[5]. The response of the health authorities was to communicate the results of our investigation to the Shire for their attention, and to offer an enlarged environmental health presence to assist with any interventions the Shire felt were appropriate.

The results of specific questioning of knowledge of hepatitis A also have public health implications. The 32 persons questioned had all suffered clinical disease, had all sought medical attention, and all children attending school had been targeted by an awareness program. In spite of this, half those questioned had no awareness of the cause of hepatitis A or of means for preventing the spread of the infection. This suggests that a continuing education program which stresses the importance of standard hygiene measures and is combined with provision of handwashing facilities with appropriate incentives for use is absolutely essential in containing or preventing an outbreak such as this[6]. Whether areas recognised to have a low socioeconomic status and with associated high rates of notified diseases should be targeted for extra education programs is debatable. Considerations of equity and distribution of wealth suggest this should be the case.

Finally, the issues of active and passive hepatitis A immunisation should be considered in the context of an outbreak such as this. General practitioners should be made aware of the presence of hepatitis A in their area as early as possible, to allow early confirmation of new cases and to encourage the detection of asymptomatic disease in young children. These measures are more important in areas of socioeconomic disadvantage where factors such as poor hygiene will encourage spread of the disease. Administration of immunoglobulin in an institutional setting has been shown to shorten the duration of an outbreak[7] but beyond vaccination of household contacts, attitudes vary with regard to classroom and work contacts[8,9] in a community outbreak. This advice must be taken in context and in a small country town, 'household contacts' may mean most classroom contacts and administration of immunoglobulin may need to be more liberal. The product literature of the inactivated hepatitis A vaccine suggests that a 'rapid immunisation' schedule may be used in conjunction with immunoglobulin in an outbreak situation[10]. Studies in non-human primates challenged with live virus show that those vaccinated do not excrete virus whereas those given immunoglobulin, although protected against disease, continue to excrete virus[11]. Perhaps children who have had

contact with a case of hepatitis A and who have been given immunoglobulin should be excluded from school. Active immunisation in the outbreak situation may stop virus circulation. These questions remain largely unanswered. Vaccination for those not closely associated with the outbreak may not be cost-effective. In the general community, public health history attests to the importance of clean water and good hygiene before vaccination and other measures in the prevention of disease.

References

1. Gust I. *Epidemiological patterns of hepatitis A in different parts of the world* [an address to the International Conference on Prospects for Eradication of Hepatitis B Virus]. Vienna, February 23-26, 1992.
2. Skinhoj P, Mikkelsen F, Hollinger B. Hepatitis A in Greenland: importance of specific antibody testing in epidemiologic surveillance. *Am J Epidemiol* 1977; **105**:140.
3. Hennekens C H, Buring JE. *Epidemiology In medicine.* Boston: Little, Brown & Co,1987.
4. Lemon SM. Type A viral hepatitis, new developments in an old disease. *New Engl J Med* 1985;**313**: 1059.
5. Proceedings of the 1992 International Symposium on Public Health Surveillance. *MMWR* 1992;**41(S)**:57.
6. Rajaratnam G, Patel M, Parry JV, Perry KR,Palmer SR. An outbreak of hepatitis A: school toilets as a source of transmission. *J Publ Health Med* 1992;**14**:72.
7. Reid J, Carter JM. Hepatitis A - investigation and control of outbreaks in two primary schools. *Public Health* 1986;**100**:69-75.
8. Benenson AS, editor. *Control of communicable diseases in man.* 15th ed. Washington: American Public Health Association,1990.
9. Immunisation Practices Advisory Committee (ACIP). Recommendations for protection against viral hepatitis. *MMWR* 1985;**34**:313-315.
10. *Havrix - hepatitis A vaccine.* SmithKline Beecham Biologicals, 1991.
11. Blaine Hollinger F, Lemon SM, Margolis H, editors. Viral hepatitis and liver disease. *Proceedings of the 1990 International Symposium on Viral Hepatitis and Liver Disease: contemporary issues and future prospects.* Baltimore: Williams and Wilkins, 1990.

REVIEW OF HEPATITIS A IN QUEENSLAND, 1983 TO 1993

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Introduction

Queensland experienced a major outbreak of hepatitis A in 1993. A review of notifications for the years 1983 to 1993 showed that numbers began to increase markedly in 1991, after the previous outbreak in 1985. A similar pattern was seen in some other Australian States and in some other countries. Hepatitis A is an important public health issue, and with a new inactivated vaccine now available a review of the epidemiology of hepatitis A is timely to ensure that this vaccine is used efficiently.

Hepatitis A is notifiable in Queensland following the demonstration of 'positive serology for hepatitis A virus'. This invariably means the demonstration of IgM antibodies against hepatitis A, and notification is predominantly laboratory-based. The laboratory-based notification system was introduced in Queensland from 1988, replacing practitioner notification. It resulted in an (approximate) 80% increase in notifications received. Contact tracing is only undertaken by health authorities in a localised outbreak.

Results

From 1 January 1984 to 31 October 1993, 2506 notifications of hepatitis A were received by Queensland Health. This represented a mean annual incidence o

Figure 1. Hepatitis A notifications, Queensland, 1984 to 1993, by year

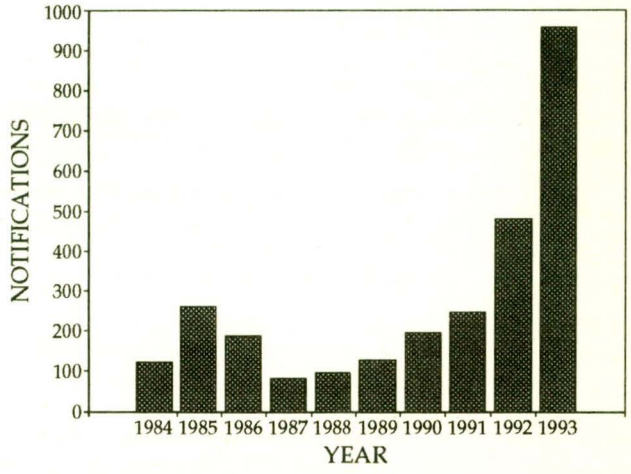


Table 1. Hepatitis A notifications, Queensland, 1984 to 1993, by Local Authority Area[1]

Local Authority Area	Notifications	Rate per 100,000 per year
Brisbane	523	8.1
Cairns	138	35.6
Mt Isa	95	47.0
Gold Coast	82	8.3
Rockhampton	72	12.8
Logan	57	4.4
Mackay	56	25.8
Maroochy	55	9.6
Mulgrave	54	6.6
Ipswich	47	3.3
Redcliffe	46	9.6
Townsville	46	3.2
Mt Morgan	45	223.0
Torres	44	67.4
Albert	36	4.1

1. Areas notifying the highest number of cases only.

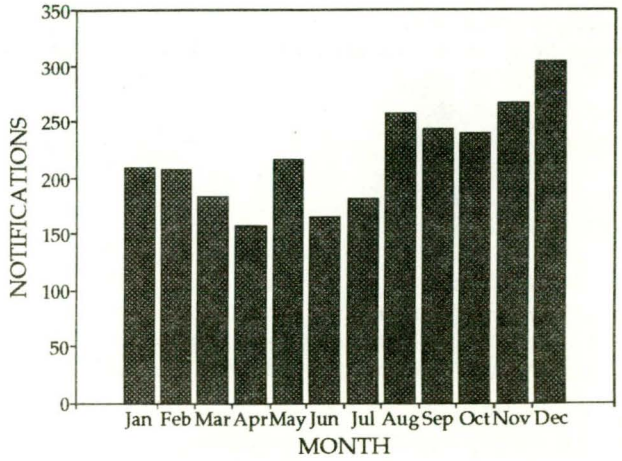
7.3 per 100,000 (4.7 per 100,000 for 1984 to 1987, and 9.9 per 100,000 for 1988 to 1993). Rates of notification did not appear to be constant throughout, but rather presented two relatively distinct peaks centred on 1985 and 1993 (Figure 1). To the end of 1993, monthly rates of notification did not appear to be subsiding. Whilst the monthly pattern of notifications (Figure 2), suggests a seasonal variation, this should be interpreted with caution due to the timing of the most recent outbreak.

Table 2. Hepatitis A notifications, Queensland, 1984 to 1993, for Local Authority Areas[1] and Aboriginal and Torres Strait Islander communities

Local Authority Area	Notifications	Rate per 100,00 per year
Hopevale	14	320
Mt Morgan	45	223
Wujal Wujal	3	196
Boulia	10	149
Lockart River	3	120
Woorabinda	7	106
Quilpie	7	87
Kowanyama	4	83
Pormpuraaw	2	79
Yarrabah	11	78
Torres	44	67
Weipa	12	59
Paroo	8	50
Aboriginal and Torres Strait Islander communities	70	56

1. Areas with the highest rates of notifications.

Figure 2. Hepatitis A notifications, Queensland, 1984 to 1993, by month



For the period 1983 to 1993 the highest raw numbers of notifications were received from the more populous Local Authority Areas, as would be expected (Table 1). However, the highest average annual incidence rates were generally from other areas (Table 2), some the sites of intense but limited duration epidemics. Notifications were assigned to 'Aboriginal and Torres Strait Islander communities' by place of residence only. This grouping allows a larger denominator population with more reliable results. For six of the ten years 1984 to 1993, the annual incidence rate for these communities has been greater than 50 per 100,000.

The rates for the period of laboratory notification (1988 to 1993), by Queensland Health Region were higher in the northern and south-western parts of the State (Figure 3).

The increase from background rates which first heralded the most recent outbreak began in 1991 in Brisbane, on the Gold Coast and in Cairns, and was followed by an increase on the Sunshine Coast. Subsequently, notifications were received from urban and rural Queensland. Local authority annual rates of disease notification exceeding 100 per 100,000 were not uncommon.

In the 10 year period 1984 to 1993, 59% of notifications of hepatitis A were for males. In the most recent outbreak the initial increase in notifications was for males aged 15 to 65 years (Figure 4), and stratification by five-year age group showed the increase to have occurred initially in the 20 to 49 year age group (Figure 5). Female notifications did not show the same pattern.

For the general community the mean age of those notified with hepatitis A was 26.8 years and the median age was 25.5 years. For the Aboriginal and Torres Strait Islander communities the mean age of those notified was 12.6 years and the median age was 6.6 years.

Figure 3. Average annual incidence rates per 100,000 population for hepatitis A notifications, Queensland, 1988 to 1993, by Queensland Health Region

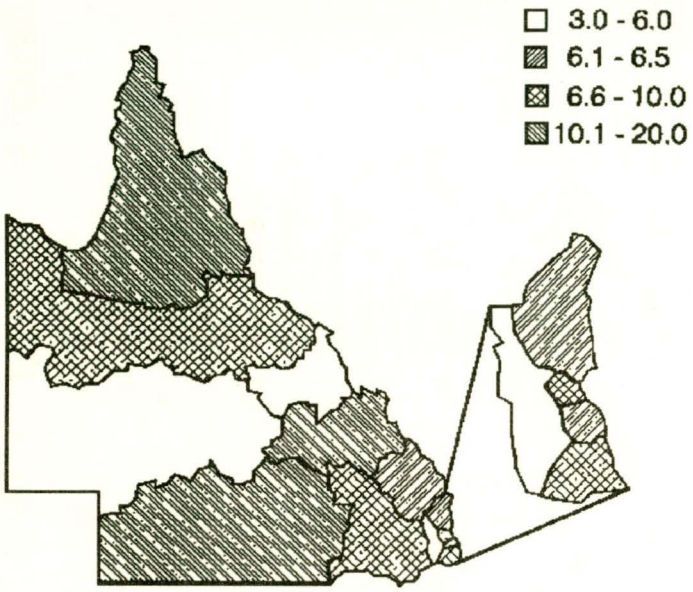
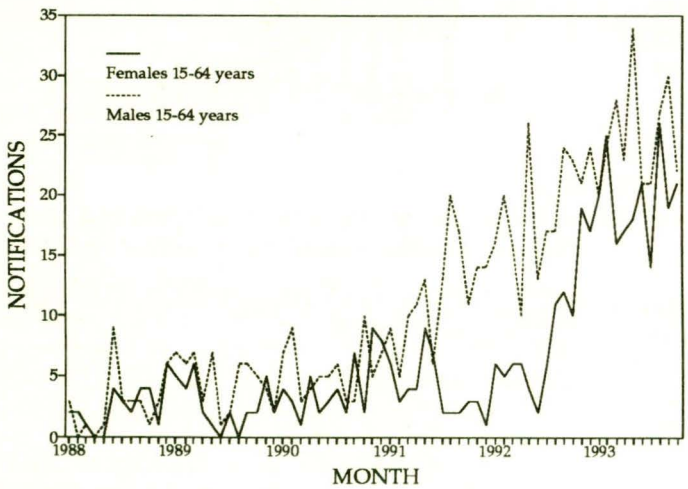


Figure 4. Hepatitis A notifications, Queensland, 1988 to 1993, by patient type and month



There were no reports of increases in the usual rates of notification in institutional settings such as jails or army barracks.

No information is available to indicate how many notifications were received for travellers returning from overseas.

Discussion

The data presented are based on notifications to Queensland Health. Even the best notification data are recognised as being an underestimate of disease incidence[1] due to mild or asymptomatic disease, inaccurate diagnosis, under-reporting, or failure to pursue a good active surveillance program - difficulties inherent in most disease surveillance programs. These limitations, which may result in significant bias and confounding, should not be forgotten when interpreting the data.

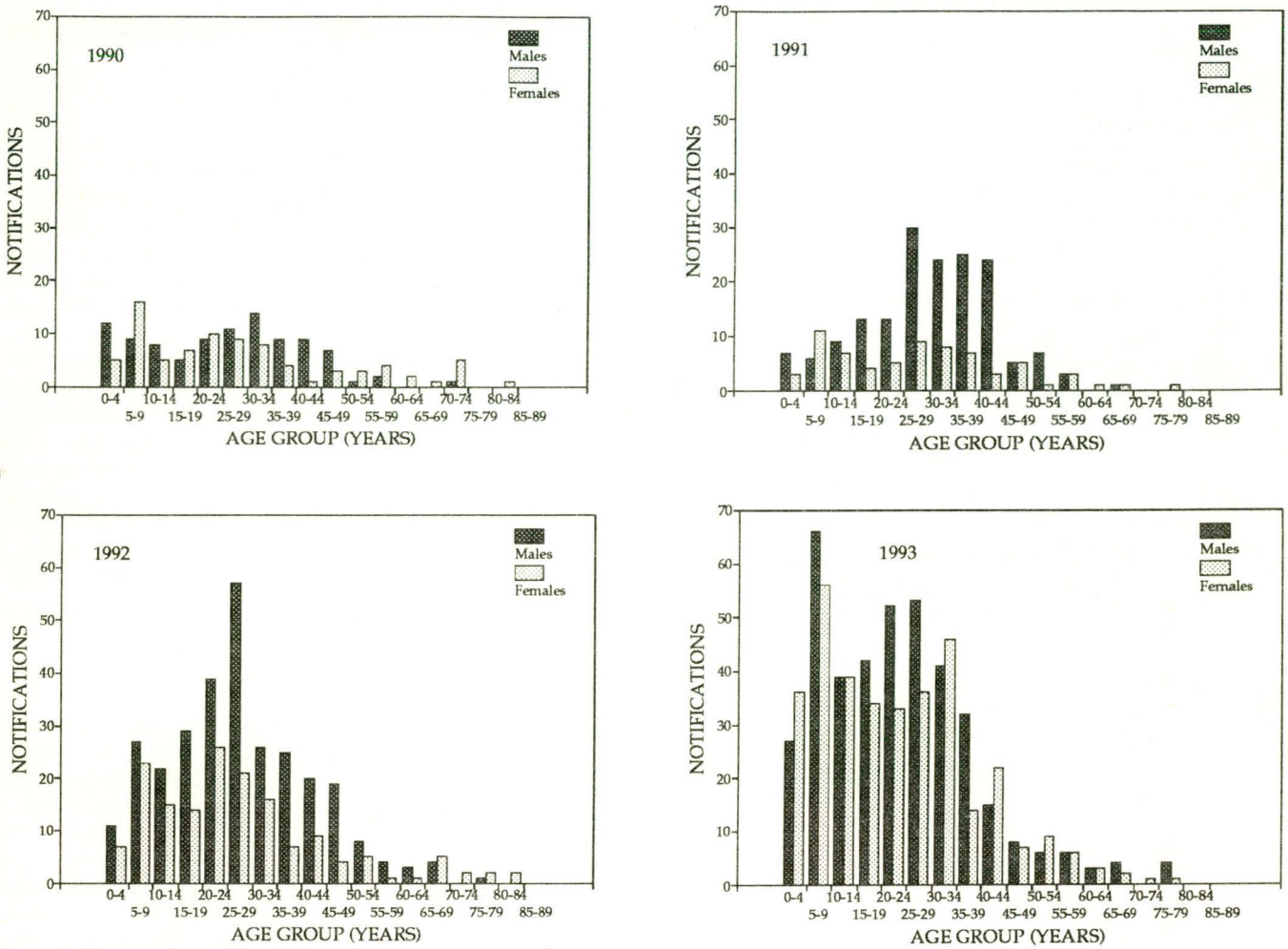
Worldwide, hepatitis A incidence has been shown to peak every five to 10 years[2], and to show a seasonal pattern which may be related to climate and other factors such as holiday breaks and festivals[3]. In Queensland the pattern is difficult to assess because in 1988 the system of notification changed from practitioner notification to laboratory notification. The hepatitides have also gained a higher profile recently with more information available to the public regarding hepatitis B and C, and consequently a greater concern with serological diagnosis. However, there appear to have been local outbreaks of hepatitis A centred on the years 1985 and 1993, confirming the experience with timing of epidemics seen elsewhere.

Notification numbers in Queensland began to rise in 1991, parallelling the pattern described in Victoria and New South Wales for that year. These results from the southern States have been well documented[4], and have been traced to outbreaks in homosexual males. In Queensland, 71% of the year's notifications were from men. Fifty per cent of the year's notifications were from the 20 to 40 years age group and of these, 76% were males. Similar patterns have been reported from the United States and Canada and in all instances higher rates of notification have been found in homosexual populations[4]. Reports of hepatitis A in homosexual men have been published from before 1980[5]. A program is being implemented in Queensland to ascertain seroprevalence in the homosexual community and to offer education regarding risk behaviours for hepatitis A.

In Queensland, 1992 and 1993 have seen an increase in hepatitis notifications for both sexes and in almost all age groups. We have no evidence to suggest that this increase was a consequence of the increase in the earlier notifications for males, which may have represented the 'leading edge' of a developing outbreak, made more apparent by viral amplification in a particular behavioural group. Education is vital for any group in which enhanced spread is possible.

The high rates of notification reported from the Aboriginal and Torres Strait Islander communities indicate a pattern of intermediate endemicity[6]; the mean and

Figure 5. Hepatitis A notifications, Queensland, 1990 to 1993, by year and age group and sex



median ages of notification are lower than for the general population. Rates of notification have shown no evidence of reduction during the period under review, being markedly higher than for the general population in most years. Rates of notification of salmonellosis and shigellosis, diseases associated with crowding and poor sanitation, are correspondingly higher in these communities than in the general population (Queensland Health notifiable diseases 1985-93, unpublished data).

There is a significant association between statistical local authority rates of notification and the index of relative socio-economic disadvantage developed by the Australian Bureau of Statistics (data not shown). Similar results have been reported overseas[7,8].

With the development of a new vaccine for active immunisation against hepatitis A the potential exists to alter the epidemiology of this disease by targeting specific groups seen to be at risk[9]. Whether the vaccine should be offered routinely to the population at large is controversial and should only be entertained after appropriate modelling and cost-benefit analysis[10,11]. This analysis suggests that the vaccine should be considered for homosexual men and for staff and health care workers dealing with Aboriginal and Torres Strait Islander peoples, at least in indigenous communities.

Other groups for whom hepatitis A vaccine has been considered are those travelling to endemic areas overseas, those at risk from occupational exposures (health care workers, plumbers, members of the armed forces), food handlers (who have a great potential to spread infection before the icteric phase), staff at day-care centres (who may handle asymptomatic children), and staff and patients in institutions for the intellectually disabled[12]. There may also be some benefit in using the vaccine in an outbreak situation. The vaccine should not be seen as a 'cure-all' whereby the considerable gains made in more than one hundred years of public health in the areas of public and personal hygiene are forgotten.

References

1. Gust I. *Epidemiological patterns of hepatitis A in different parts of the world* [an address to the International Conference on Prospects for Eradication of Hepatitis B Virus]. Vienna, February 23-26, 1992.

2. Polakoff S. Reports of clinical hepatitis A from public health and hospital microbiology laboratories to the PHLS Communicable Disease Surveillance Centre during the period 1980-1988. *J Infect* 1990;**21**:111-117.

3. Gust I, Feinstone SM, editors. *Hepatitis A.* Florida: CRC Press, 1988.

4. Centers for disease control. Hepatitis A among homosexual men - United States, Canada, and Australia. *MMWR* 1992;**41**:155-164.

5. Corey L, Holmes K. Sexual transmission of hepatitis A in homosexual men. *N Engl J Med* 1980;**302**:435-438.

6. Prevention of foodborne hepatitis A. *WHO Wkly Epidemiol Rec* 1993;**68**:25-32.

7. Szmuness W, Dienstag JL, Purcell RH, Harley EJ, Stevens CE, Wong DC. Distribution of antibody to hepatitis A antigen in urban adult populations. *N Engl J Med* 1976;**295**:755-759.

8. Majeed FA, Stuart JM, Cartwright KAV, Room R, Gilkes JR, Smith MC, Watson BE. An outbreak of hepatitis A in Gloucester, UK. *Epidemiol Infect* 1992;**109**:167-173.

9. Tilzey AJ, Banatvala JE. Hepatitis A - changing prevalence and possible vaccines [editorial]. *BMJ* 1991;**302**:1552-1553.

10. Hall AJ. [letter] *BMJ* 1991;**303**:1634-1635.

11. Zuckerman JN, Cockcroft A, Griffiths P. [letter] *BMJ* 1991;**303**:1635.

12. Update on hepatitis. Royal Australasian College of Physicians. *Fellowship Affairs*, 1993 Aug;9.

CDI editorial comment

In 1991 there was a marked increase in hepatitis A notifications for Australia overall. A total of 2195 was reported to the National Notifiable Diseases Surveillance System, after an average of 576 for the period 1987 to 1990. This higher number of notifications has continued since, with 2109 for 1992, and 1973 so far for 1993.

A 1993 date of onset was recorded for 1954 notifications, with no apparent seasonal trend. There were two peaks in the age distribution, one for the 5 to 9 years age group and the other for the 20 to 24 years age group. There were similar numbers of males and females notified (1026 males and 911 females; male:female ratio 1.13:1.00), in contrast with 1991 (1629 males and 529 females; ratio 3.08:1.00) and 1992 (1316 males and 733 females; ratio 1.80:1.00).

The National Health and Medical Research Council's 1991 recommendations for the control of hepatitis A include the use of normal immunoglobulin (human) for postexposure prophylaxis for some contacts of cases, and for pre-exposure prophylaxis for travellers to areas where the disease is highly endemic. Its Panel on Hepatitis A is currently considering recommendations for the use of the inactivated hepatitis A vaccine.

INCREASED INCIDENCE OF ANTIBIOTIC RESISTANT PNEUMOCOCCI IN A QUEENSLAND PATHOLOGY PRACTICE

Gavine Cooper, Mark Tandy and Peter Bielenberg, Laboratories of Drs JJ Sullivan, NJ Nicolaides & Partners, Brisbane and Bundaberg, Queensland

Introduction

In May 1993 we isolated from the sputum of a young, normally healthy Bundaberg man with community acquired acute bronchitis, a strain of *Streptococcus pneumoniae* (pneumococcus) that was multiply-antibiotic resistant. As well as intermediate penicillin resistance, the strain also showed chloramphenicol resistance, an event which had not been previously recorded in our practice. We were prompted by these matters to search for and report on antibiotic resistant pneumococci in our practice records.

Methods

Penicillin resistance was detected by the National Committee for Clinical Laboratory Standards (NCCLS) oxacillin disk method[1] and confirmed by the E-test[2].

Susceptibility tests for other antibiotics were done in our laboratory by the NCCLS method[1]. Chloramphenicol resistance was confirmed by a positive chloramphenicol acetyl transferase (CAT) test.

Results

Over a four year period, June 1989 to July 1993, thirteen strains of penicillin G resistant pneumococci were identified from 2092 isolates made during this period (Table 1).

All of the resistant strains showed intermediate penicillin resistance with a minimum inhibitory concentration (MIC) range of 0.125 - 1.0 mg/L.

Multiple resistance (resistance to penicillin and two other antibiotic classes) was found in six of our 13 strains (Table 2, isolates 2, 4, 6, 8, 10, 12). Two isolates were chloramphenicol resistant, including the one which prompted this analysis (isolate 10).

Overall, for all 328 pneumococcal isolates obtained during 1992-93, resistance incidence for cotrimoxazole was

37%, for tetracyline, 13%, and for macrolide (erythromycin), 8%.

Comment

Our increasing incidence of penicillin and/or multiply-resistant pneumococci mirrors national trends which are of increasing concern to infectious disease physicians. There are important therapeutic implications when such organisms cause serious disease[3].

As an addendum we found that in the second six months of 1993 there was a continuing trend of increased isolation of antibiotic resistant pneumococci in our laboratories.

Table 1. Incidence of penicillin resistant pneumococci, July 1989 to June 1993, by year

Year (July-June)	Total isolates	Pencillin resistant isolates	% penicillin resistant
1989-90	597	0	0
1990-91	575	3	0.5
1991-92	592	1	0.2
1992-93	328	9	2.7

Acknowledgment

We thank Jan Bell, Woden Valley Hospital, ACT for performing the CAT test on our isolates.

References

1. National Committee for Clinical Laboratory Standards. *Performance standards for antimicrobial disk susceptibility tests*, 4th ed. Villanova: NCCLS, 1990. NCCLS Document M2-A4.
2. Jacobs MR, Bajaksouzian S, Appelbaum PC, Bolmstrom A. Evaluation of the E-test for susceptibility testing of pneumococci. *Diagn Microbiol Infect Dis* 1992;15:473-478.
3. Collignon PJ. Penicillin-resistant pneumococci: will the recent Olympics bring back more to Australia than gold? *Med J Aust* 1992;157:655-657.

Table 2. Antibiotic resistance patterns and other details of penicillin resistant isolates, July 1989 to June 1993

Isolate	Age	Sex	Specimen	Antibiotic resistance patterns			
				Cotrimoxazole	Erythromycin	Tetracycline	Chloramphenicol
1	69 years	F	Sputum	R	S	S	S
2	47 years	F	Sputum	R	R	R	S
3	65 years	F	Sputum	S	S	S	S
4	66 years	M	Sputum	R	R	R	S
5	7 months	F	Blood	N/A	S	N/A	S
6	9 years	F	Eye	R	R	R	S
7	9 month	F	Eye	R	S	S	S
8	36 years	F	Eye	R	R	R	S
9	64 years	F	Antrum	R	S	S	S
10	26 years	M	Sputum	R	R	R	R
11	1 month	M	Nasopharynx	R	S	S	S
12	23 years	F	Nasopharynx	R	R	R	R
13	1 month	M	Ear	R	S	S	S

R resistant
S susceptable
N/A not available

NORWALK-LIKE VIRUS OUTBREAK IN A HOSTEL FOR THE AGED, VICTORIA

Kath Taylor, Public Health Officer, Anne Murphy, Public Health Nurse; Health and Community Services, Victoria

The Department of Health and Community Services, Victoria was notified on 25 January 1994 of an outbreak of diarrhoea and vomiting affecting a number of residents in a hostel for the aged. Two officers from Health and Community Services visited the hostel on the day of the notification to investigate the outbreak. An Environmental Health Officer from the local council also attended. Information on the outbreak was obtained through discussions with several members of staff and from residents' records. It was not possible to interview the residents because of their age.

A total of 43 males and females, average age 89 years, lived at the hostel. Of these, 39 lived in separate rooms located in four units, each resident sharing a shower and toilet with one other resident. Four residents lived in their own flats each with its own shower and toilet. Residents were served breakfast in their rooms by personal care assistants, and all 43 residents attended a common dining room for lunch and tea.

There were 28 staff members comprising the nursing director, assistant director, personal care assistants, two cooks, a kitchen hand, four contract cleaners and a gardener. One staff member only was rostered for night duty on a rotational basis. Visiting staff included 20 doctors, a dietitian and a social worker.

The first single case of vomiting and diarrhoea occurred on 22 January 1994. By 25 January there had been 12 residents with symptoms, most occurring on 23 January. At that time no staff members were ill.

The units and kitchen were inspected and food handling procedures discussed. Detailed instructions outlining safe food handling measures to prevent person to person transmission of infection were given to the director. These included the isolation of symptomatic patients and decontamination of surface areas with 500 ppm hypochlorite, using disposable gloves and gowns.

It was considered that a virus was the most likely cause of the outbreak. Staff were requested to collect faecal samples from new cases and notify the Department to enable early transport to the laboratories.

The outbreak continued, the last resident to be affected becoming ill on 29 January. Thirty residents, including one resident of a flat suffered gastroenteritis, vomiting being the most prominent symptom. All were seen by doctors, who prescribed maxolon or stemetil where needed. Gastrolyte was given to all affected residents to ensure adequate hydration.

A personal assistant member of staff reported that she had become ill on the afternoon of 25 January, three days after the onset of illness in a resident. Three kitchen staff were later infected. These staff had delivered food trays to the residents' rooms. On 27 January a kitchen aide vomited in the kitchen area, and although thorough cleaning of all surfaces was undertaken the two cooks became ill within the next two days, suggesting that aerosols produced by the vomiting had lead to contamination. In all, eight staff members were affected. They were excluded from work until 48 hours after recovery.

The duration of illness among residents and staff was 24 to 36 hours, the major symptoms of vomiting and diarrhoea occurring during the first six hours. No-one was seriously ill but one resident was transferred to hospital because he was an insulin-dependent diabetic.

Faeces specimens from five persons were sent to the Microbiological Diagnostic Unit, University of Melbourne for bacterial investigation and to the Victorian Infectious Diseases Reference Laboratory at Fairfield Hospital for electron microscopy. Three, which were fresh diarrhoeal specimens, revealed Norwalk-like virus detected by electron microscopy (reported in this issue of *CDI*, page 95).

The outbreak of the viral infection lasted for ten days. No secondary cases occurred in staff, family members or visitors to the hostel. Despite the high attack rate, 30 of 43 residents (70%), and 8 of 28 staff (29%), the infection was mild and the residents, although elderly, were not considered to be at risk.

The source of the infection was not determined. There was no evidence that it was foodborne since the food handling practices were considered satisfactory and only freshly cooked food is served. There were no salads, cold meats or sandwiches and no food was retained after meals or reheated. The kitchen staff did not become ill until late in the outbreak, suggesting that person to person transmission occurred from the residents to the staff who were delivering and collecting food trays.

Recovery was complete and all residents were again able to attend the dining room for their meals. Interestingly, the oldest resident, a female aged 101 years who lived in a unit room with shared facilities, continued to enjoy all her meals in her room and the dining room throughout the outbreak, remaining asymptomatic.

This outbreak of gastroenteritis illustrates the high infectivity of the virus despite good cleaning procedures. Vomiting, at times projectile, was the most prominent symptom and it was probable that aerosols contaminating surfaces were responsible for the spread of infection. Isolation of infectious patients and staff and environmental decontamination, using sodium hypochlorite, are essential control measures in outbreaks of this nature.

OVERSEAS BRIEFS

In the last two weeks, the following information has been supplied by the World Health Organization (WHO), the WHO Collaborating Centre for Influenza Reference and Research, Melbourne, the Department of Foreign Affairs and Trade and the Institut Pasteur, Paris.

Influenza in Papua New Guinea

There have been recent reports of a severe epidemic occurring in a remote region of the Madang Province of Papua New Guinea commencing in November 1993. The outbreak is estimated to have involved 3,000 to 4,000 cases over a three month period with up to 200 deaths, primarily among the very young and the elderly.

Although the outbreak has not yet been confirmed by laboratory diagnosis, an epidemiologist who has visited the area believes that the outbreak is consistent with influenza virus infection.

There is evidence of some continuing cases in the region and attempts are being made to collect specimens for study at the WHO Collaborating Centre, Melbourne.

Influenza in the Northern Hemisphere

In western Europe, influenza activity has almost disappeared. Reports from Austria, Belgium, Denmark, Finland, France, Germany, Iceland, Italy, Netherlands, Portugal, Sweden, Switzerland and the United Kingdom indicate no recent activity or a continuing decline in the number of isolations and activity as measured by other indicators. Activity is still increasing in a few eastern European countries, in particular Bulgaria, the Czech Republic, Hungary, Lithuania, Romania, the Russian Federation, Solvakia and Yugoslavia. Bulgaria has reported epidemic levels of activity in half its cities, and isolations of influenza A H_3N_2. Romania reported a sudden increase in morbidity and epidemic levels in the north and north-east and isolations of influenza A H_3N_2 in January, but declining activity in February. Increased activity rates were reported by the Russian Federation for many cities in January, and detections of influenza A H_3N_2 and influenza A H_1N_1.

In the United States, widespread or regional activity was reported from 26 States on 1 February, and pneumonia and influenza deaths were still above expected levels. A total of 98% of isolates have been influenza A H_3N_2. Influenza activity increased in January in Canada and was sporadic in some areas and widespread in others.

In Asia, influenza B has been the predominant type reported from China and Thailand. Japan has had a quiet influenza season. There have only been a few isolations, of influenza A H_3N_2, close to A/Beijing/32/92.

Cholera update

Lebanon has reported its first recent cases of cholera. Two hundred and sixteen cases and 12 deaths were reported for the period 30 August to 15 October 1993.

A suspected outbreak of cholera has been reported from East Timor, Indonesia. To the end of January, eight persons were reported to have died and 65 hospitalised in the Bobonaro Region. A number of posts had been set up to treat the sick and to prevent the illness from spreading further.

Cases of cholera have also been reported for November, December and January from Argentina, Belize, Bolivia, Brazil, China, Costa Rica, Djibouti, Ecuador, El Salvador, Guatemala, Iran, Hong Kong, Mexico, Nicaragua and Zaire.

CDI NOTICE TO READERS

HealthROM

Health ROM is an Australian CD-Rom reference source of publications and citations produced by the Information Resources Section of the Department of Human Services and Health in collaboration with the National Health and Medical Research Council, the Australian Institute of Health and Welfare, the Alcohol and Drug Council of Australia and the National Library of Australia.

It covers the broad field of public and environmental health, clinical medicine, HIV/AIDS and communicable diseases, alcohol and drug use, nutrition, therapeutic goods, family health research and sports medicine.

It embraces over 100 full-text publications produced by the participating organisations, including *Communicable Disease Intelligence*, as well as directories, reports, bibliographies and databases of citations from the professional literature.

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HealthROM (including three quarterly updates) costs $250 per annum for individuals ($350 for corporations

and libraries) and has versions for DOS (386 with 1.8 Meg RAM), WINDOWS (4 Meg RAM) and Macintosh. Enquires should be directed to John Holgate, telephone (06) 289 8488 or fax (06) 289 7102.

COMMUNICABLE DISEASES SURVEILLANCE

Virology and Serology Reporting Scheme

There were 1704 reports received in the *CDI* Virology and Serology Reporting Scheme this fortnight (Tables 6, 7 and 8).

- There were 53 reports of **measles** this period including 46 from Queensland. Two reports were of one year old males, one from the Australian Capital Territory and the other from Queensland. Fifty-one diagnoses were by measles specific IgM detection. The number of measles reports recently received by this Scheme remains high (Figure 1).
- **Rubella** was reported for 15 patients this fortnight (6 from Queensland, 3 each from Western Australia and New South Wales, 2 from South Australia and one from the Australian Capital Territory). Included were 4 females in the 15 to 44 years age group. Diagnosis was by viral IgM detection in 14 cases.
- Seventeen reports of **hepatitis A** were received this fortnight including 8 from New South Wales and 7 from Queensland.
- Positive **hepatitis B** serology was reported for 117 patients this period. Included was a 4 year old female from Victoria.
- The following patients were reported positive for **hepatitis C virus** by serology: a 6 year old haemophiliac, a 40 year old male with a history of blood transfusion, and 2 female injecting drug users, 22 and 37 years old, the latter reported as having been jaundiced for 10 years.
- **Ross River virus** infection was reported for 158 patients this period (specimen collection dates in 1994 apart from one which was December 1993). One hundred and one reports were from Queensland, 4 from New South Wales, and one each from the Northern Territory, Western Australia and Victoria. All were presumptive diagnoses. Fifty-nine patients reported muscle/joint disease, three skin disease and the clinical information was unknown for the remainder. More laboratory reports of Ross River virus have been received with collection dates in January than for any year since 1989 (Figure 2).
- There were 7 cases of **Barmah Forest virus** infection reported, 3 from Western Australia, 3 from Queensland and 1 from the Northern Territory. One case, a 50 year old male from the Gold Coast, Queensland, was confirmed (fourfold change in titre), and 6 were presumptive diagnoses.
- Fifty-nine reports of **adenoviruses** were received this fortnight. Fourteen patients reported respiratory symptoms, 19 gastrointestinal disease, 12 eye disease and one skin disease.
- **Herpes simplex virus type 1** was reported isolated from the eye of a 4 year old female with eye disease.
- **Herpes simplex virus type 2** was isolated from a postmortem kidney specimen from a 10 day old child, and from a 39.5 weeks pregnant female with a 6 day history of perineal ulceration .
- There were 31 reports of **cytomegalovirus (CMV)** infection this fortnight. Included was a 4 month old

Figure 1. Measles laboratory reports, 1992 to 1994, by month of specimen collection

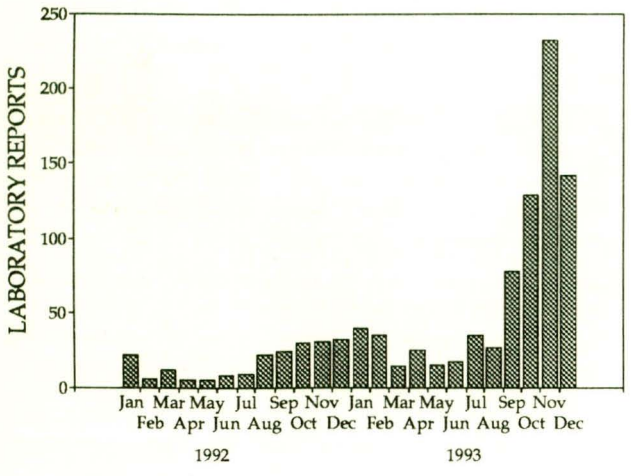
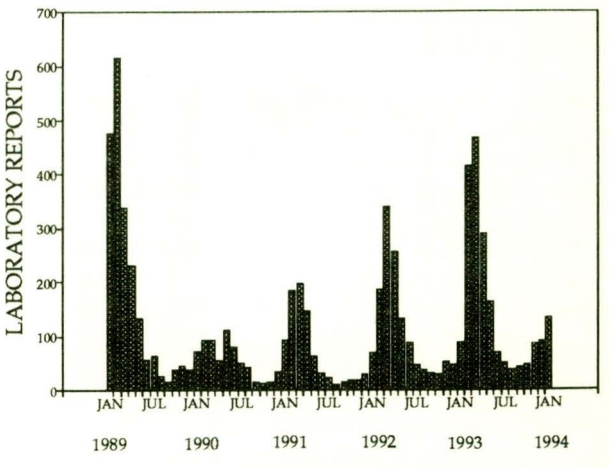


Figure 2. Ross River Virus reports, 1989 to 1994, by month of specimen collection



male who had suddenly died, a 23 year old pregnant female with abnormal liver function tests, and a 72 year old female. Eleven reports were of virus isolation and 20 of viral IgM detection.

- Forty-one reports of **varicella-zoster virus** were received this fortnight, 29 from Queensland, 5 from Victoria, 3 each from New South Wales and Western Australia and one from South Australia. Included was a 36 year old pregnant female (13 weeks gestation). Six cases were diagnosed by virus isolation, 20 by antigen detection, 14 by IgM detection and one by a fourfold change in titre.
- **Parvovirus** was reported for 5 patients this fortnight, 3 from Western Australia, one from Victoria and one from Queensland. Diagnosis was by virus specific IgM detection.
- Three reports were received of **coxsackievirus type B4,** including a 12 year old male with meningitis (virus isolated from a throat swab), and a virus isolate from the nasopharynx of a 7 month old male with pneumonia from whom cytomegalovirus was also isolated.
- Isolation of **coxsackievirus type B5** was reported from the CSF of a 25 year old female with a diagnosis of meningitis.
- The number of reports of **echovirus type 30** remains high, 20 isolates being reported this period, 17 from Victoria, 2 from Tasmania and one from Western Australia. Meningitis was reported for 17 patients, and respiratory symptoms for one.
- Isolation of **poliovirus type 2** was reported from a stomach biopsy from a 48 year old HIV positive patient.
- Sixteen reports of **influenza A** virus were received this fortnight, 15 of which were from South Australia. Five of the patients were over 65 years of age and all diagnoses were by single high titre. The number of influenza A laboratory reports remains high for the time of year (Figure 3).
- **Influenza B** was reported for 3 South Australian patients this period, all diagnosed by single high titre.
- **Parainfluenza virus type 3** was reported for 8 patients this period, 3 from New South Wales, 2 each from South Australia and Victoria and one from Western Australia. Six diagnoses were by virus isolation, one by single high titre and one by IgM detection.
- Twenty reports of **respiratory syncytial virus (RSV)** were received this fortnight, 11 from Western Australia, 5 from Victoria, 2 from the Northern Territory, and one each from New South Wales and South Australia. During 1992 and 1993 RSV has been reported more than ever before by this Scheme (Figure 4).
- Three cases of **Norwalk-like virus** were reported in 73, 89 and 93 year old patients as part of an outbreak of gastroenteritis in a nursing home in Victoria (see article, p 92 this of issue of *CDI*). Diagnosis was by electron microscopy.
- **Coronavirus** was diagnosed by electron microscopy for a 2 year old male with gastroenteritis.
- One hundred and fourteen cases of ***Chlamydia trachomatis***, including three cases of eye disease and 100 of genital infection (one an 18 year old pregnant female) were reported this fortnight. Sixty-three diagnoses were by isolation, 45 by antigen detection, 5 by nucleic acid detection and one by IgM detection.
- ***Mycoplasma pneumoniae*** was reported for 54 patients this period, one a 5 month old male who had been hospitalised for 28 days prior to onset. During 1993 more *Mycoplasma* reports were received than any year since 1983 (Figure 5).

Figure 3. Influenza A laboratory reports, 1989 to 1994, by month of specimen collection

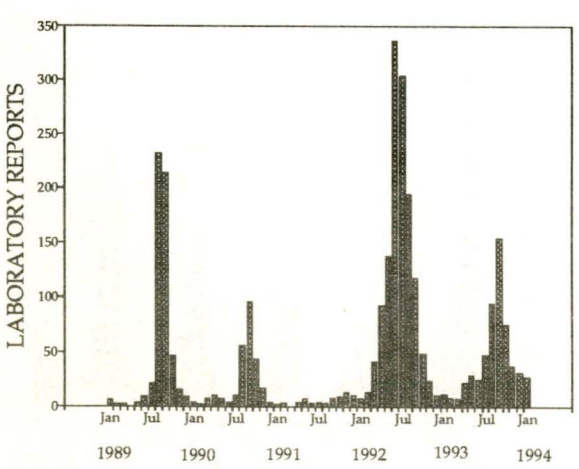


Figure 4. RSV laboratory reports, 1982 to 1994, by year of specimen collection

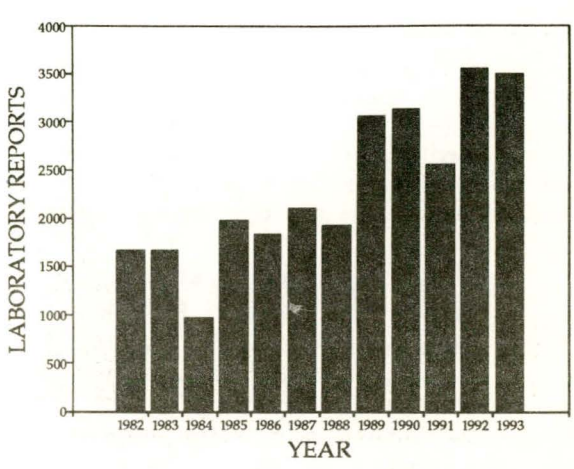
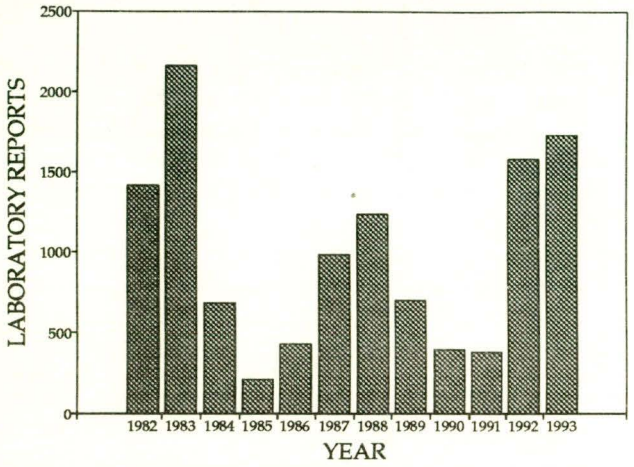


Figure 5. *Mycoplasma pneumoniae* laboratory reports, 1982 to 1993, by year of specimen collection



- Sixteen cases of **Q fever** were reported this fortnight, 8 from Queensland, 5 from New South Wales and 3 from Victoria. Included were a 39 year old dairy farmer and a 27 year old abattoir worker.
- There were 60 ***Bordetella*** reports this reporting period (15 *Bordetella pertussis* and 45 *Bordetella* species). The number of reports being received by this Scheme remains at a high level (Figure 6).
- Positive **syphilis** serology was reported for a 13 year old male from Queensland and a neonate from New South Wales whose mother had been treated for syphilis.
- A single case of ***Echinococcus granulosus*** was reported for a 73 year old female from Victoria with hydatid liver disease.

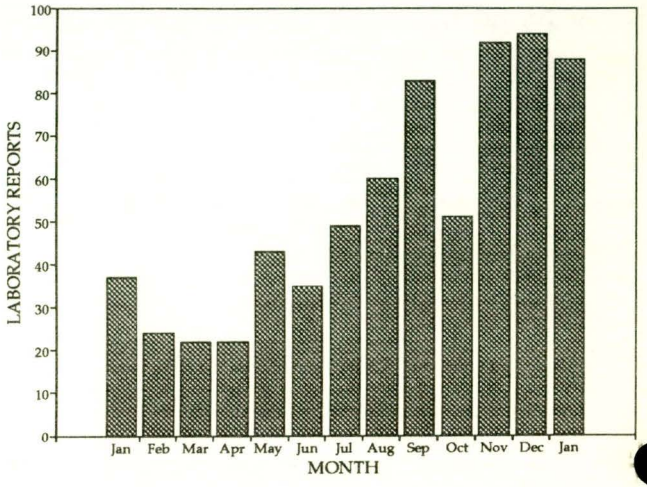
Sterile Sites Surveillance (LabDOSS)

There were 210 reports of recent sepsis provided by 12 laboratories. *CDI* welcomes Greenslopes Repatriation Hospital Queensland to the LabDOSS scheme.

The number of reports of sepsis this fortnight were: Greenslopes Repatriation Hospital Queensland 12, Nambour Hospital Queensland 6, Sullivan Nicolaides Queensland 3, Ipswich General Hospital Queensland 7, Sir Charles Gairdner Hospital Western Australia 14, IMVS South Australia 47, Liverpool Hospital New South Wales 20, ICPMR Westmead New South Wales 11, Royal North Shore Hospital New South Wales 37, Woden Valley Hospital Australian Capital Territory 31, Northern Tasmanian Pathology Service 5, Royal Hobart Hospital 17.

Ninety-eight reports of organisms from cases of sepsis in November and December have been merged with 1993 data. These included 3 cases of *Haemophilus influenzae* type b sepsis (a 3 year old female with epiglotittis, a 4 year old male and a 23 year old female), 2 cases of meningococcal meningitis (a one year old male in New South Wales with group C *Neiserria meningitidis* and an

Figure 6. *Bordetella* laboratory reports, 1993 to 1994, by month of specimen collection



84 year old female in Queensland, serogroup not provided), and one case of *Salmonella* Cerro septicaemia in a 2 year old male in New South Wales.

Organisms reported 5 or more times from blood are detailed in Table1. Other blood isolates not included in Table 1 were:

Gram positive: 1 *Listeria monocytogenes* (immunocompromised 59 year old male from South Australia), 3 *Enterococcus faecalis*, 1 *Enterococcus faecium*, 1 *Streptococcus* Group A, 2 *Streptococcus* Group G, 1 *Streptococcus sanguis*, 1 *Streptococcus mitis* 2 Streptococcus 'viridans', 1 *Streptococcus 'milleri'*.

Gram negative: 1 *Haemophilus influenzae* type b (73 year old male), 1 *Salmonella* species (a 4 year old female from Queensland), 2 *Acinetobacter* species (1 *A. calcoaceticus*, 1*A. lwoffi*), 1 *Aeromonas sobria*, 3 *Proteus mirabilis*, 2 *Serratia* species (1 S. *marcescens*, 1 S. *liquefaciens*).

Figure 7. LabDOSS reports of blood isolates, by age group

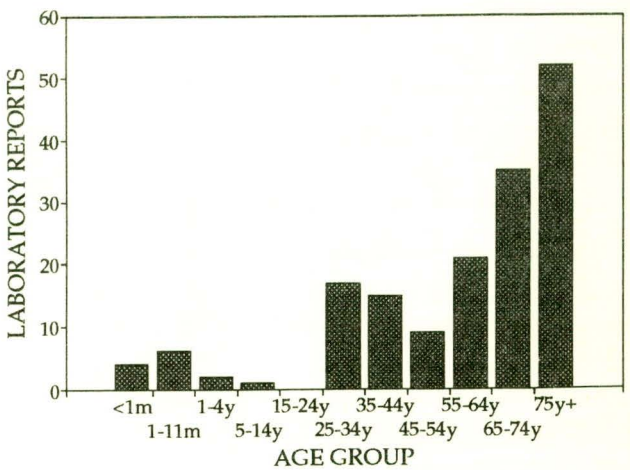


Table 1. LabDOSS reports of blood isolates, by organism and clinical information

Organism	Clinical Information						Risk Factors					Total[1]
	Bone/Joint	Lower respiratory	Endocarditis	Gastrointestinal	Urinary Tract	Skin	Surgery	Immunosuppressed	IV line	Hospital acquired	Neonatal	
Staphylococcus aureus	2	2	1	1		12	6	3	10	5		45[2]
Staphylococcus coagulase negative				1	1							24[3]
Group B *Streptococcus*	1				1	1	1	3				8
Streptococcus pneumoniae		6			1	1						8
Escherichia coli	1		5		13			4				29
Enterobacter species				3	1		1	2				7[4]
Klebsiella species		1		7	2			7	1	1		19[5]
Pseudomonas species								2	1	1		5[6]

1. Only organisms with 5 or more reports are included in this table.
2. MRSA 9.
3. *Staphylococcus epidermidis* 21, *S. warneri* 1, *S. capitis* 1.
4. *Enterobacter cloacae* 5, *E. aerogenes* 1.
5. *Klebsiella pneumeniae* 13, *K. oxytoca* 6.
6. *Pseudomonas aeruginosa* 4, *P.putida* 1.

Table 2. LabDOSS meningitis reports, by organism and age group

Organism	1-4 years	25-34 years	35-44 years	Total
Listeria monocytogenes[1]			1	1
Escherich ia coli		1		1
Pseudomonas aeuginosa		1		1
Staphylococcus coagulase negative	1			1

1. 41 year old female, ACT

Anaerobes: 5 *Bacteroides* species (4 *B fragilis*), 2 *Clostridium* species, 1 anaerobic *Streptococcus.*

Fungi: 2 *Candida albicans.*

Most reports were for patients over the age of 55 years (Figure 7).

CSF isolates andor meningitis reports.

There were four reports of CSF isolates and/or meningitis (Table 2).

Isolates from sites other than blood or CSF

Peritoneal dialysate: 1 *Neisseria mucosa.*

Joint fluid: 7 *Staphylococcus aureus,* 1 *Streptococcus sanguis,* 1 Group G *Streptococcus,* 1 *Staphylococcus lugdunensis.*

Other: 4 *Staphylococcus aureus,* 1 group G *Streptococcus,* 2 *Streptococcus* 'milleri', 1 *Streptococcus pneumoniae,* 5 *Escherichia coli,* 1 *Proteus* species, 1 *Pseudomonas aeruginosa,* 1 *Acinetobacter* species, 1 *Bacteroides fragilis.*

National Notifiable Diseases Surveillance System, 23 January 1994 to 5 February 1994

There were 1,770 reports for this period (Tables 6,7 and 8 and Figure 12). No reports were received from Victoria.

- There were 268 notifications of **Ross River virus infection** for this period; 112 males and 155 females. (Figure 8) The sex of one case was unrecorded. The cases ranged in age from the 0-4 to 85-89 years age groups, with 86% (230) of cases occurring between the 20-24 and 55-59 years age groups. The majority of cases occurred in Queensland, with highest number of cases being reported in the Statistical Divisions of Brisbane (74), Fitzroy (53), Moreton (28), and Wide Bay-Burnett (21). The

Northern Territory reported 43 cases but the Statistical Divisions were not recorded. The remaining cases came from other statistical divisions in Queensland, New South Wales, South Australia and Western Australia.The reported onset dates were December (236), January (11) and February (21).

- Two cases of **dengue** were notified; one case was reported for a female in the 50-54 years age group and one in a male in the 75-79 years age group. Both cases were resident in rural Queensland. Onset dates were recorded as December and January respectively.
- A single case of **diphtheria** was reported for an aboriginal male in the 45-49 years age group resident in the Northern Territory. The onset date was January.
- Sixty-one notifications of **gonococcal infection** were received in the period. There were 45 males and 16 females. Ages ranged between the 0-4 and 75-79 years age groups. Age was unrecorded in one case.
- A single case of ***Haemophilus influenzae* type b infection** was reported in a female in the 75-79 years age group who was resident in New South Wales (Figure 9). The onset date was in January.
- **There were 65 cases of hepatitis A** reported in the period. Forty-one cases were males and 23 were females. Sex was unrecorded for one case. Ages ranged from the 0-4 to the 90-94 years age groups. The age was unrecorded for one case.
- Forty-eight cases of **hepatitis B** were reported. There were seven cases from the States that report incident cases (representing new infections); New South Wales (one), Western Australia (5) and Northern Territory (one).
- Three cases of **legionellosis** were reported; one case was male and 2 were females. Ages ranged between the 20-24 and 80-84 years age groups. There were no apparent clusters.

Figure 8. Ross River virus infection, January 1992 to February 1994, by month of onset

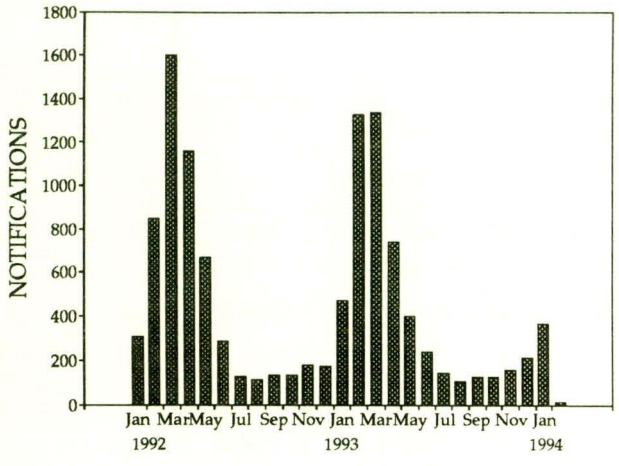
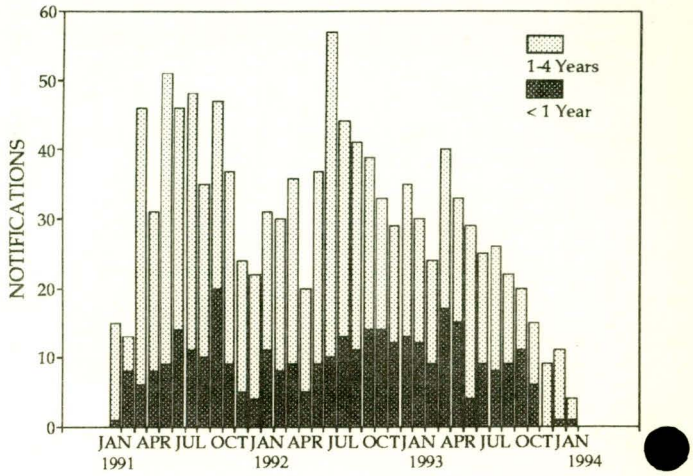
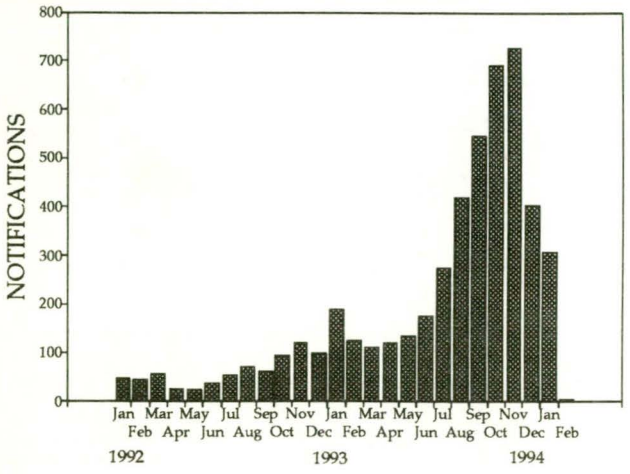


Figure 9. *Haemophilus influenzae* type b infection notifications, January 1992 to February 1994, by month of onset and age group



- There were two cases of **leptospirosis** notified for the period. Both cases were in males aged in the 20-24 years and 25-29 years age groupsand resident in rural Queensland.
- One case of **listeriosis** was notified for a male in the 55-59 years age group who was resident in South Australia. The recorded onset date was in January.
- There were six cases of **malaria** reported; 5 cases were male and one was female. Recorded ages ranged between the 15-19 and 30-34 years age groups. One case was a resident of the 'malaria receptive zone'. Five of the cases had an onset date in January and the onset date was unrecorded for the remaining case.
- Ninety-four cases of **measles** were notified in the period. Forty-six cases were male and 47 were females. Sex was unrecorded in one case. Fourteen cases were aged less than one year and the mean age was 11.9 years. There were 13 apparent clusters with up to 7 cases each in separate postcode areas. Apparent clusters were in New South Wales (7) and Queensland (6).
- There were 13 notifications of **meningococcal infection**; 7 were male and 6 were female. Cases ranged in age between the 0-4 and 90-94 years age groups, with 5 cases reported in persons less than 20 years. There were no apparent clusters.
- One hundred and seventy-nine cases of **pertussis** were notified; 97 cases were males and 82 were females (Figure10). Cases ranged in age between the 0-4 and 85-89 years age groups. Ten cases were less than one year old and 33 cases were less than 5 years old. There were 27 apparent clusters with 2 to 7 cases each in separate postcode areas. Appar-

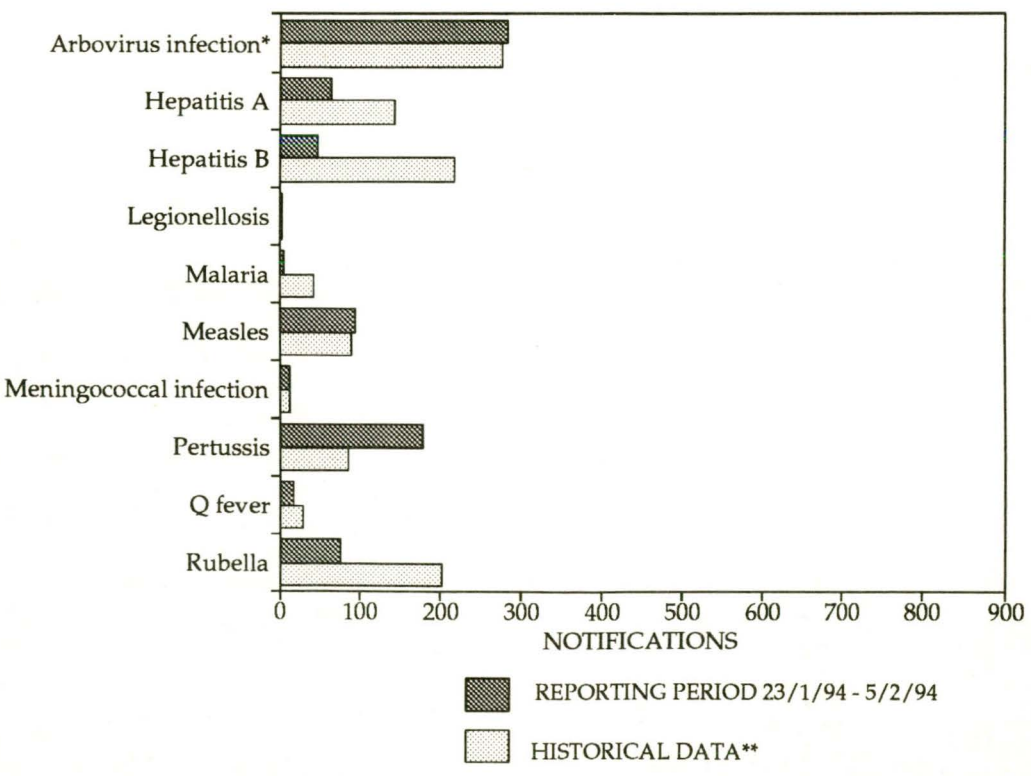
Figure 10. Pertussis notifications, January 1992 to January 1994, by month of onset.



ent clusters were in New South Wales (5), Queensland (16), and South Australia (6).

- There were 18 notifications for **Q fever** reported in the period. Fifteen cases were male and 3 were female. Cases ranged in age between the 15-19 and 60-64 years age groups. Five cases were residents in Statistical Divisions in rural New South Wales, one case was resident in the Brisbane Statistical Division, and 12 cases were resident in Statistical Divisions in rural Queensland.
- There were 75 notifications for **rubella**; 54 cases were male and 21 were female. Cases ranged in age between 0-4 and 90-94 years age groups. Age was not recorded in 3 cases. The mean age of cases was 51.5 years and 6 cases were recorded for females in the 15-44 years age group. There were 10 apparent clusters with up to 5 cases each in separate postcode areas. Apparent clusters were in the Australian Capital Territory (one), Queensland (one), Northern Territory (one), and Western Australia (7).
- Fifty cases of **syphilis** were notified in the period. Twenty-four cases were male and 26 were female. One case was reported for a female aged less than one year.
- There were 13 notifications for **tuberculosis** in the period; 6 cases were male and 7 were females. Cases ranged in age between 5-9 and 80-84 years age groups. Recorded onset dates were December (one), January (11), and February (one).
- Twenty-three cases of **yersinosis** were notified; 14 cases were male, 8 cases were female, and sex was unrecorded in one case. There were two apparent clusters with 2 cases each in the same postcode area reported in Queensland.

Figure 11. Selected National Notifiable Diseases Surveillance System reports, and historical data **



* Includes Ross River virus and Dengue

** The historical data are the averages of the number of notifications in 6 previous 2-week reporting periods: the corresponding periods of the last 2 years and the periods immediately preceding and following those.

Table 3. Notifiable Diseases preventable by vaccines recommended by the NHMRC for routine childhood immunisation for the reporting period 23 January to 5 February 1994

DISEASES	ACT	NSW	NT	Qld	SA	Tas	Vic	WA	TOTALS FOR AUSTRALIA[1] This Period 1994	This Period 1993	Year to Date 1994	Year to Date 1993
Diphtheria	0	0	0	0	0	0		0	0	0	1	0
Haemophilus influenzae b infection	0	1	0	0	0	0		0	1	31	17	53
Measles	6	44	0	41	0	1		2	94	118	381	222
Mumps	0	0	NN	NN	0	NN		0	0	0	0	0
Pertussis	0	48	0	79	45	0		7	179	141	447	219
Poliomyelitis	0	0	0	0	0	0		0	0	0	0	0
Rubella[2]	5	1	4	16	7	0		42	75	381	209	719
Tetanus	0	0	0	NN	0	0		0	0	2	0	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.
2. NT, Tas: CRS only.

NN Not Notifiable.

Table 4. Other Notifiable Diseases[1], for the reporting period 23 January to 5 February 1994

DISEASES	ACT	NSW	NT	Qld	SA	Tas	Vic	WA	TOTALS FOR AUSTRALIA[2] This Period 1994	This Period 1993	Year to Date 1994	Year to Date 1993
Arbovirus infection (NEC)[3]	0	1	2	11	0	1		0	15	27	48	40
Ross River virus infection	0	12	43	207	4	NN		2	268	273	553	396
Dengue	0	-	0	2	-	NN		NN	2	10	3	15
Campylobacteriosis[4]	14	-	5	99	66	21		39	244	736	610	1283
Chlamydial infection (NEC)[5]	1	NN	6	91	28	5		41	172	456	468	810
Donovanosis	0	NN	1	1	NN	NN		2	4	3	6	5
Gonococcal infection[6]	0	4	3	21	7	0		26	61	209	209	388
Hepatitis A	2	10	5	38	2	0		8	65	180	140	286
Hepatitis B	4	1	2	35	0	1		5	48	164	169	328
Hepatitis C	14	0	12	70	0	12		44	152	305	460	575
Hepatitis (NEC)	0	0	0	1	0	0		NN	1	7	5	9
Legionellosis	0	1	0	1	1	0		0	3	4	9	8
Leptospirosis	0	0	0	2	0	0		0	2	17	11	25
Listeriosis	0	0	NN	0	1	0		0	1	5	2	10
Malaria	1	2	0	1	1	1		0	6	42	14	80
Meningococcal infection	0	2	0	6	0	0		5	13	20	29	39
Ornithosis	0	NN	0	0	0	1		0	1	9	1	16
Q fever	0	5	0	13	0	0		0	18	35	45	60
Salmonellosis (NEC)	4	44	36	90	18	12		28	232	461	452	777
Shigellosis[4]	0	-	1	8	3	1		14	27	85	62	147
Syphilis	1	17	18	11	0	0		3	50	152	143	248
Tuberculosis	0	3	0	0	6	0		4	13	57	42	105
Typhoid[7]	0	0	0	0	0	0		0	0	7	1	13
Yersiniosis (NEC)[4]	0	-	0	15	7	1		0	23	48	56	84

1. For HIV and AIDS, see Tables 2 and 3 *CDI* 1994;18:69. For rarely notified diseases, see Table 5.
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. SA, Tas: includes Ross River virus and dengue.
4. NSW: only as 'foodborne disease' or 'gastroenteritis in an institution'.
5. WA: genital only.
6. NT, Qld, SA and Vic: includes gonococcal neonatal ophthalmia.
7. NSW and Vic: includes paratyphoid.

NN Not Notifiable.

NEC Not Elsewhere Classified.

\- Elsewhere Classified.

Table 5. Rarely Notified Diseases[1] for the reporting period 23 January to 5 February 1994

DISEASES	Total This Period	Reporting States or Territories	Year to Date 1994
Botulism	0		0
Brucellosis	0		1
Chancroid	0		0
Cholera	0		0
Hydatid infection	0		1
Leprosy	0		0
Lymphogranuloma venereum	0		0
Plague	0		0
Rabies	0		0
Yellow fever	0		0
Other viral haemorrhagic fevers	0		0

1. Fewer than 50 cases of each of these diseases were notified each year during the period 1987 to 1992.

Table 6. Laboratory reports by State or Territory[1] for the reporting period 27 January to 9 February 1994, historical data[2], and total reports for the year

	State or Territory [1]								Total this fortnight	Historical data [2]	Total reported this year
	ACT	NSW	NT	Qld	SA	Tas	Vic	WA			
MEASLES, MUMPS, RUBELLA											
Measles virus	1	1		46	1		4		53	15.3	358
Mumps virus				2			2		4	1.8	19
Rubella virus	1	3		6	2			3	15	29.7	194
HEPATITIS VIRUSES											
Hepatitis A virus	1	8		7				1	17	18.0	58
Hepatitis B virus		34		51	3	1	16	12	117	79.8	383
Hepatitis C virus	17	37		59	49	5	2	53	222	87.3	898
Hepatitis D virus				1					1	1.0	6
ARBOVIRUSES											
Ross River virus		4	1	151	1			1	158	34.8	367
Barmah Forest virus			1	3				3	7	4.7	46
ADENOVIRUSES											
Adenovirus type 1		1					3		4	4.2	15
Adenovirus type 2		3					1		4	2.7	17
Adenovirus type 3		1							1	2.5	6
Adenovirus type 5		1							1	.8	3
Adenovirus type 8							8		8	.5	24
Adenovirus not typed/pending		11			11		7	12	41	30.5	286
HERPES VIRUSES											
Herpes simplex virus type 1	2	17		93	34	3	42	33	224	152.3	860
Herpes simplex virus type 2		25	1	108	24		28	49	235	162.8	999
Herpes simplex not typed/pending	5	6		6				4	21	27.7	118
Cytomegalovirus		3		12	1		10	5	31	60.8	256
Varicella-zoster virus		3		29	1		5	3	41	33.7	192
Epstein-Barr virus	3	3		26	3		13	4	52	65.8	286
OTHER DNA VIRUSES											
Parvovirus				1			1	3	5	6.3	12
PICORNA VIRUS FAMILY											
Coxsackievirus A16							2		2	.2	11

Table 6. Laboratory reports by State or Territory[1] for the reporting period 27 Januaray to 9 February1994, historical data[2], and total reports for the year, continued

	State or Territory [1]								Total this fortnight	Historical data [2]	Total reported this year
	ACT	NSW	NT	Qld	SA	Tas	Vic	WA			
Coxsackievirus B1		1							1	2.7	16
Coxsackievirus B2		1							1	.2	5
Coxsackievirus B3		1							1	.5	1
Coxsackievirus B4		3							3	.5	3
Coxsackievirus B5							1		1	3.5	5
Echovirus type 9		2							2	2.2	3
Echovirus type 11		1							1	1.5	22
Echovirus type 30						2	17	1	20	.2	121
Poliovirus type 1 (uncharacterised)		2					1		3	2.2	6
Poliovirus type 2 (uncharacterised)		4							4	1.7	5
Poliovirus type 3 (uncharacterised)		2							2	.8	5
Rhinovirus (all types)		2					9		11	22.5	193
Enterovirus not typed/pending				4			7	2	13	22.8	271
ORTHO/PARAMYXOVIRUSES											
Influenza A virus					15		1		16	4.0	96
Influenza B virus					3				3	1.8	68
Parainfluenza virus type 1							2		2	4.3	16
Parainfluenza virus type 3		3			2		2	1	8	13.7	54
Respiratory syncytial virus		1	2		1		5	11	20	8.5	137
OTHER RNA VIRUSES											
HIV-1				4					4	2.2	14
Rotavirus	16	9			1		5	4	35	24.0	180
Calici virus		1							1	1.2	4
Norwalk agent							3		3	.2	4
Coronavirus		1							1	1.0	1
OTHER											
Chlamydia trachomatis not typed	3	11		50	15		1	34	114	98.7	422
Chlamydia psittaci					1		2		3	5.2	15
Mycoplasma pneumoniae		4		42	4	1	3		54	55.8	239
Coxiella burnetii (Q fever)		5		8			3		16	12.0	97
Streptococcus group A		1		12			1		14	5.8	50
Bordetella pertussis							8	7	15	3.2	112
Bordetella species		7		38					45	5.7	126
Treponema pallidum		15		3			1		19	13.5	59
Toxoplasma gondii		1					1		2	1.0	5
Schistosoma species		1							1	.0	1
Echinococcus granulosus							1		1	.2	6
TOTAL	49	240	5	762	172	12	218	246	1,704	1,146.3	7,776

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 7. Laboratory reports by clinical information for the reporting period 27 January to 9 February 1994

	Encephalitis	Meningitis	Other CNS	Congenital	Respiratory	Gastrointestinal	Hepatic	Skin	Eye	Muscle/joint	Genital	Other/unknown	Total
MEASLES, MUMPS, RUBELLA													
Measles virus					4			24				25	53
Mumps virus	1											3	4
Rubella virus								5				10	15
HEPATITIS VIRUSES													
Hepatitis A virus							13					4	17
Hepatitis B virus							14					103	117
Hepatitis C virus					1	2	20					199	222
Hepatitis D virus												1	1
ARBOVIRUSES													
Ross River virus								3		59		96	158
Barmah Forest virus					1			1		1		4	7
ADENOVIRUSES													
Adenovirus type 1					2			1				1	4
Adenovirus type 2					1	2						1	4
Adenovirus type 3									1				1
Adenovirus type 5					1								1
Adenovirus type 8									8				8
Adenovirus not typed/pending					10	17			3			11	41
HERPES VIRUSES													
Herpes simplex virus type 1					2			121	6		71	24	224
Herpes simplex virus type 2							1	47	1		172	14	235
Herpes simplex not typed/pending								10			5	6	21
Cytomegalovirus					6		3			1		21	31
Varicella-zoster virus								31			1	9	41
Epstein-Barr virus					5							47	52
OTHER DNA VIRUSES													
Parvovirus					1			2				2	5
PICORNA VIRUS FAMILY													
Coxsackievirus A16								2					2
Coxsackievirus B1		1											1
Coxsackievirus B2												1	1
Coxsackievirus B3												1	1
Coxsackievirus B4			1		1							1	3
Coxsackievirus B5		1											1
Echovirus type 9			1									1	2
Echovirus type 11												1	1
Echovirus type 30		17			1							2	20
Poliovirus type 1 (uncharacterised)			1									2	3
Poliovirus type 2 (uncharacterised)					1							3	4
Poliovirus type 3 (uncharacterised)						1						1	2
Rhinovirus (all types)					10							1	11
Enterovirus not typed/pending		4			1			1				7	13
ORTHO/PARAMYXOVIRUSES													
Influenza A virus					11							5	16

Table 7. Laboratory reports by clinical information for the reporting period 27 january to 9 February1994, continued

	Encephalitis	Meningitis	Other CNS	Congenital	Respiratory	Gastrointestinal	Hepatic	Skin	Eye	Muscle/joint	Genital	Other/unknown	Total
Influenza B virus					2							1	3
Parainfluenza virus type 1					2								2
Parainfluenza virus type 3					6							2	8
Respiratory syncytial virus					19							1	20
OTHER RNA VIRUSES													
HIV-1												4	4
Rotavirus						35							35
Calici virus						1							1
Norwalk agent						3							3
Coronavirus						1							1
OTHER													
Chlamydia trachomatis not typed									3		100	11	114
Chlamydia psittaci					2							1	3
Mycoplasma pneumoniae					19							35	54
Coxiella burnetii (Q fever)					1							15	16
Streptococcus group A										1		13	14
Bordetella pertussis					15								15
Bordetella species					30							15	45
Treponema pallidum				1							5	13	19
Toxoplasma gondii												2	2
Schistosoma species												1	1
Echinococcus granulosus												1	1
TOTAL	1	23	3	1	155	62	51	248	22	62	354	722	1704

Table 8. Laboratory reports by contributing laboratories for the reporting period 27 January to 9 February 1994

STATE OR TERRITORY	LABORATORY	REPORTS
Australian Capital Territory	Woden Valley Hospital, Canberra	49
New South Wales	Institute of Clinical Pathology & Medical Research, Westmead	41
	Royal Alexandra Hospital for Children, Camperdown	6
	South West Area Pathology Service, Liverpool	120
Queensland	Queensland Medical Laboratory, West End	833
South Australia	Institute of Medical & Veterinary Science, Adelaide	172
Tasmania	Northern Tasmanian Pathology Service, Launceston	4
	Royal Hobart Hospital	6
Victoria	Monash Medical Centre, Melbourne	13
	Royal Children's Hospital, Melbourne	57
	Victorian Infectious Diseases Reference Laboratory, Fairfiel	151
Western Australia	Princess Margaret Hospital, Perth	31
	State Health Laboratory Services, Perth	221
TOTAL		1704