Hepatitis A in Australia in the 1990s: future directions in surveillance and control

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Abstract

The national notification data from 1952 to 1997 was examined in order to characterise hepatitis A virus (HAV) infection in Australia in the 1990s, and to determine whether currently available surveillance data are sufficient to inform disease control strategies and vaccination policies. Hepatitis A annual notification rates declined dramatically from a high of 123 notifications per 100,000 persons in 1961, to 3 per 100,000 in 1989. During 1991-97, the hepatitis A notification rate was 12 per 100,000 persons per year, although rates varied substantially between States and Territories. The Northern Territory had the highest notification rate of 52 per 100,000 persons per year. Seventy-six per cent of cases were adults, although in most regions notification rates were significantly higher in children than adults. Nationally, the male to female ratio was 1.7:1 ($p<0.001$). The Northern Territory was the only area with no significant difference in notifications between the sexes. Large outbreaks were detectable through the notification system but risk factors for transmission could only be inferred from age and sex distribution of notifications, and from previous outbreak reports. National hepatitis A surveillance would be improved by collecting basic risk factor data, which identify cases as food-borne, sporadic, related to another case, or travel related. In addition, a population based serosurvey to measure age-specific hepatitis A susceptibility would assist vaccination policy development. Serosurveillance data are also needed, in conjunction with enhancements of the notification data, to provide baseline information against which the impact of changes in vaccination policy can be assessed. *Commun Dis Intell* 1999;23:113-120.

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Introduction

Hepatitis A is an infectious disease caused by an RNA virus.[1] Humans are considered the main reservoir for the hepatitis A virus (HAV),[2] and HAV is the predominant cause of infectious hepatitis transmitted by the faecal-oral route. HAV is primarily transmitted from person to person, and this type of transmission is most evident between household contacts and within institutions. Point source outbreaks also arise as a result of faecal contamination of water, transmission from infected food handlers, and contamination of raw or under-cooked foods.[2]

Age is the most important determinant of morbidity and mortality, with severity of the illness and its complications increasing with age.[2] In young children most infections are either asymptomatic or cause a mild non-specific anicteric illness. The duration of the illness varies, but most commonly cases are symptomatic for three weeks. Complications during the acute illness phase are unusual, with fulminant hepatitis and death being uncommon.[2]

During the past decade in Australia there have been several reports of wide scale hepatitis A activity including: a large food-borne outbreak related to consumption of oysters,[3] outbreaks in men who have sex with men,[4,5] high endemic rates in some parts of the country,[6] and continued concern about transmission within institutions,[7-9] particularly child care centres.[10]

This article aims to analyse the national notification data from 1952 to 1997, Australian Bureau of Statistics (ABS) mortality data, and liver transplant data and to interpret these data in view of reported hepatitis A outbreaks. Whether national data sources are sufficient for planning national hepatitis A control strategies and vaccination policies, will also be discussed. Currently the National Health and Medical Research Council (NHMRC) recommends HAV vaccination only for certain high risk groups[11] (see Box). Examination of national surveillance data is also necessary to establish a baseline prior to the possible introduction of a universal childhood hepatitis A vaccination program.

Methods

Hepatitis A is notifiable by doctors and laboratories in all States and Territories of Australia. In 1991, the National Notifiable Diseases Surveillance System (NNDSS) was established, and since then notification data has been collated nationally in a de-identified format. The variables uniformly reported to NNDSS for hepatitis A are age, sex, postcode of residence, date of onset, and date the notification was received by the State or Territory.

The NHMRC definition for hepatitis A cases is:

a) anti-HAV (antibody to hepatitis A virus) IgM positive, in the absence of recent vaccination;

or

b) demonstration of a clinical case of hepatitis (jaundice +/- elevated aminotransferase levels without a non-infectious cause) and epidemiologically linked to a serologically confirmed case.[12]

All States and Territories report cases on the basis of the NHMRC definition, except New South Wales (NSW) and Western Australia (WA) where positive anti-HAV IgM serology is required.

Notification data for 1952 through 1990 were obtained from the National Centre for Disease Control in summary format (Htoo Myint, data manager, personal communication, 1997). Until the 1970s, cases of hepatitis A included those classified as infectious/infective hepatitis. In March 1998 unit notification data with onset from 1 January 1991 to 31 December 1997 were extracted from the NNDSS database. Mortality figures for hepatitis A (ICD 9 codes 0700 and 0701) and mid-year population estimates were obtained from the Australian Bureau of Statistics (ABS). Notification rates were calculated using the average of the mid-year populations occurring within the period as the denominator, and were adjusted for age, sex and State as appropriate. All rates were reported per 100,000 population per year. For categorical analyses of age, persons aged less than 15 years were defined as children, and persons aged 15 years or older as adults. From 1991 to 1997 the Australian population increased from 17.3 million to 18.5 million, and persons aged less than 15 years accounted for 21-22% of the population during this period.

Box 1. Groups for whom hepatitis A vaccination is recommended[11]*

- Travellers to areas of intermediate or high endemicity
- Occupations with significant risk of exposure:
  - carers for children in day care centres
  - teachers and close contacts of the intellectually disabled
  - staff and residents of residential facilities for the intellectually disabled
  - health workers and teachers in remote Aboriginal and Torres Strait Islander Communities
  - health care workers with paediatric, emergency and/or intensive care unit exposure
  - sewerage workers
- Men who have sex with men
- Individuals with chronic liver disease
- Recipients of blood products
- Food handlers

*Prevaccination screening is recommended for some groups

Figure 1. National hepatitis A notifications 1952 to 1995

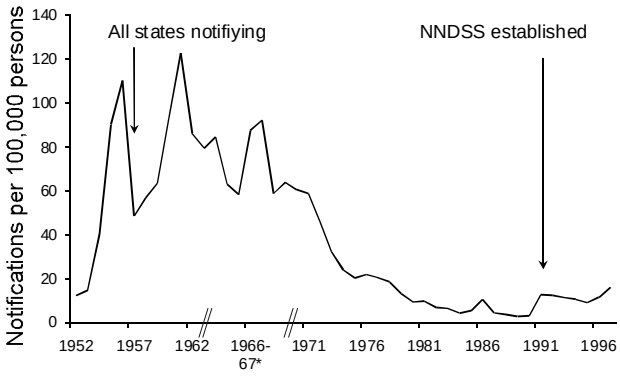
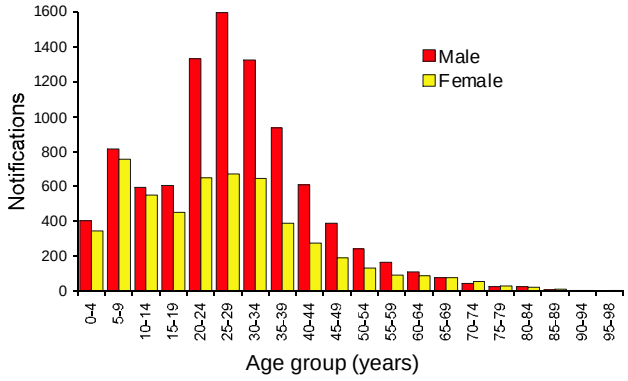


Figure 2. Hepatitis A notifications by age group and sex, 1991 to 1997



* Only notifications tallied by financial year available from 1964 through 1969

Year States/Territories began notifying- 1952: ACT, NT, Vic, WA; 1953 NSW; 1954 SA, Tas; 1957 Qld

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Significance values were calculated using Pearson's Chi-square. The case fatality rate (CFR) was calculated by dividing the number of hepatitis A deaths from 1979 to 1996 by the number of hepatitis A notifications for the same time period. Liver transplant data were obtained from the Australian National Liver Transplant Unit, Clinical Experience Report, 1986 to 1997.[13] Outbreaks (peak periods of notifications) for the seven year period 1991 to 1997 were determined using a probability distribution methodology developed by the authors. The method used involves calculating the threshold number of notifications above which an outbreak is defined to occur. The outbreak level (O) is defined as the number of notifications occurring in a month for which the probability of that number occurring is less than 0.05, based on the expected number of notifications per month being the average number of notifications per month. Expected and outbreak numbers of notifications were calculated for adult males, adult females and children. For analysis of the national data all expected monthly counts were greater than 45, therefore the outbreak level was determined on the basis of a chi-square distribution. For each State/Territory where the expected monthly count was less than 45, a Poisson error distribution was used as the basis for determining the outbreak level.

SAS version 6.12 , Excel 5.0 and Epi Info 6 were used for analysis and presentation of data.

Results

Notification data

Secular trends

The summary data from 1952 to 1997 (Figure 1) showed peaks in crude notification rates in 1956, 1961 and 1968 with a gradual drop in notification rates occurring from

Table 1. Distribution of hepatitis A notifications by State/Territory and age, 1991 to 1997

State/Territory	Number of notifications (Notification rates per 100,000 population per year)					
	0-14 years		≥15 years or over		Total	
ACT	56	(11.8)	147	(9.0)	205	(9.7)
NSW	1,352	(14.9)	4,767	(14.2)	6,138	(14.4)
NT	230	(68.5)	386	(43.4*)	633	(51.7)
Qld	1,084	(21.8)	3,093	(17.8*)	4,207	(18.9)
SA	146	(7.0)	405	(5.0*)	551	(5.4)
Tas	7	(0.9)	55	(2.2*)	62	(1.9)
Vic	320	(4.8)	1,833	(7.4*)	2,214	(7.0)
WA	313	(11.6)	681	(7.4*)	1,002	(8.4)
Australia	3,508	(13.0)	11,367	(11.6*)	5,012	(12.0#)

*Significant difference between children and adults, p<0.001.

All State/Territory total rates were significantly different to that for Australia as a whole, p<0.001

1971 to 1985. There were three small peaks in notification rates in 1986, 1991 and 1997. There was no apparent national seasonal pattern to notifications.

Age and sex

For the time period 1991 through 1997 the age distribution was bimodal, markedly so in males, with peaks in the 5 to 9 and 20 to 39 year age groups (Figure 2). Because of the bimodal age distribution of notifications, further analysis of the impact of age was made by comparison of those aged 0-14 years (children) to those aged 15 years or older (adults). Seventy-six per cent of notifications were for adults. The national notification rate in children, 13.0 per 100,000 persons per year, was significantly but not substantially higher than for adults, 11.6 per 100,000 per year (p<0.001) (Table 1).

Significantly more notifications occurred for males than females, resulting in a male to female ratio of 1.7:1 (p<0.001). This ratio differed significantly by age group, with the ratio for adults (2.0:1) being significantly greater than that for children (1.1:1) (p<0.001). More males than females were notified in all age groups up to 64 years. The median age for males and females was 27 and 24 years respectively.

Regional differences

The mean annual crude notification rate for States and Territories during 1991-97 ranged from 1.9 to 51.7 per 100,000 per year (Table 1). New South Wales had the highest number of cases and the NT had the highest notification rate. The NT, Queensland and NSW all had significantly higher notification rates than Australia overall. In all States/Territories adults accounted for the majority of notifications (State/Territory range 61-89%, Australia 76%). However, the notification rate in children was greater than that in adults in all States and Territories except Tasmania and Victoria.

Table 2. Male to female ratio by State/Territory, 1991 to1997

State/Territory	Male : Female
ACT	1.6:1*
NSW	1.9:1*
NT	1.1:1
Qld	1.4:1*
SA	1.5:1*
Tas	2.4:1*
Vic	2.4:1*
WA	1.4:1*
Australia	1.7:1*

*Significant difference between males and females, p<0.001.

The male to female ratio also varied by State and Territory. The NT was the only area with no significant difference between the number of male and female notifications (Table 2). Tasmania, Victoria, and NSW had male to female ratios greater than Australia as a whole.

Table 3. Outbreak threshold number of notifications* for States/Territories by age and sex

State/ Territory	Adult males (notifications per month)	Adult females (notifications per month)	Children (notifications per month)
ACT	4	3	3
NSW	48	24	23
NT	6	6	7
Qld	30	21	19
SA	7	5	5
Tas	3	2	2
Vic	23	11	8
WA	9	8	8
Australia	120	65	60

* See methods for details of calculation.

Secular trends, 1991 to 1997, by age, sex and region

Nationally, there were 15,012 notifications of hepatitis A from 1991 through 1997, equivalent to a crude annual notification rate of 12 per 100,000 persons.

Adults

Analysis of monthly outbreak data from 1991 through 1997 for Australia shows the extent of temporal variability in hepatitis A notifications (Figure 3). The expected number of notifications per month and the outbreak number of notifications for males were estimated to be 100 and 120 notifications respectively, and 50 and 65 notifications respectively for females (Table 3).

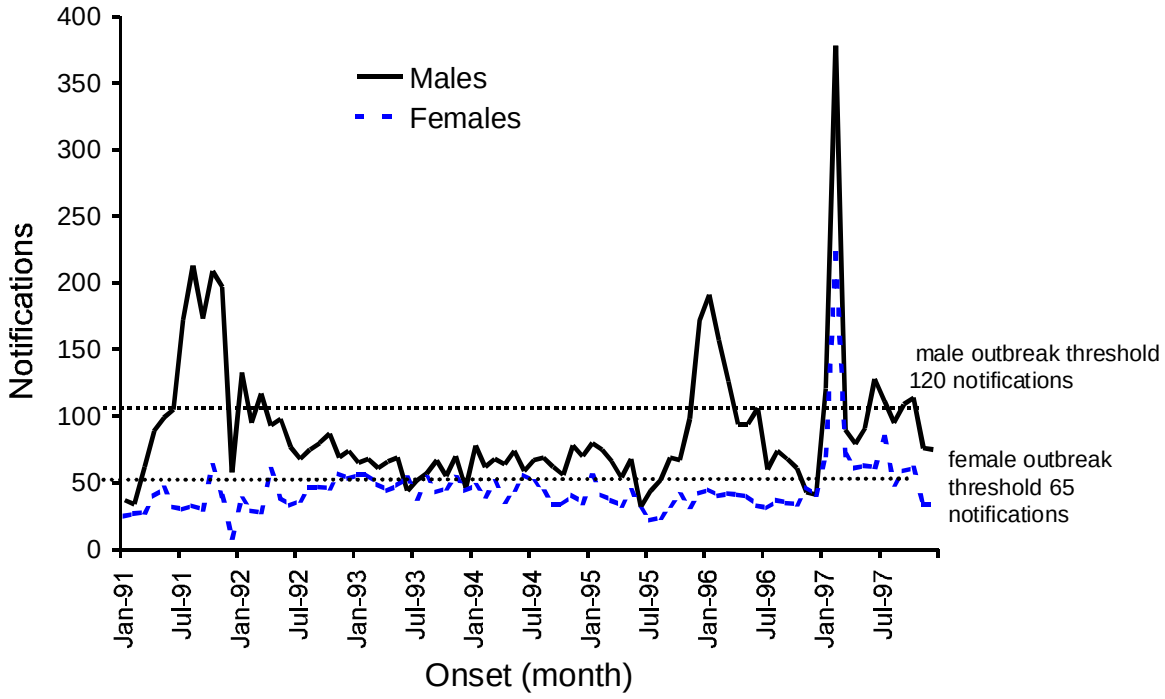
a) Peaks 1 and 2

Two sustained peaks occurred when the number of notifications for males exceeded the outbreak level of 120 notifications per month (Figure 3). Peak 1 occurred from July 1991 to January 1992, and peak 2 from December 1995 to March 1996. The male to female ratio for notifications was 4.8:1 in peak 1 and 3.8:1 in peak 2. States and Territories reporting higher numbers of male notifications than their respective outbreak levels (Table 3) during the peak periods were: the Australian Capital Territory (ACT), NSW and Victoria for both peaks; South Australia (SA) for peak 1 only; and Western Australia (WA) for peak 2 only.

b) Peak 3

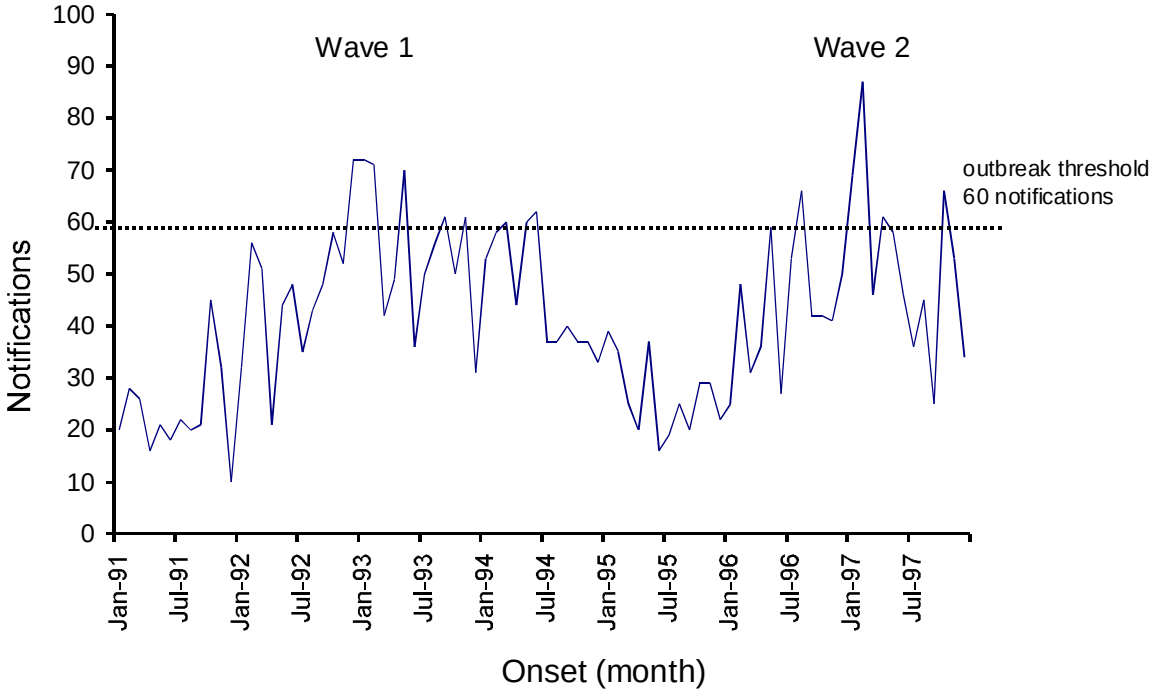
There was a sharp peak in the notifications in early 1997 (peak 3) (Figure 3). Notifications were higher than the outbreak level for men from January 1997 to February 1997 and for females from January 1997 to March 1997. The States reporting a higher number of notifications than their respective outbreak levels for males and females (Table 3) during this peak were: the ACT , NSW,

Figure 3. Adult hepatitis A notifications by sex and month of onset, 1991 to 1997, Australia



* Peak periods are where plot is above the outbreak threshold, see methods for detail

Figure 4. Childhood hepatitis A notifications by month of onset, 1991 to 1997



Queensland and Victoria. SA reported outbreak numbers for females only in this period.

Children

For children the expected number of notifications nationally per month was 47 and the outbreak number of notifications was estimated to be 60 notifications (Table 3).

The national notification pattern for children showed two waves of increased notifications, during 1993-94 and 1996-97. However, the number of notifications exceeded the outbreak level of 60 for only relatively short periods (Figure 4). During the first wave, all State/Territories except Tasmania reported notification counts above their respective threshold levels (Table 3). During the second wave, the ACT, NSW, NT, Queensland and SA reported

notification counts above their respective outbreak threshold levels (Table 3).

Transplants

From 1986 through 1997 three patients with hepatitis A were assessed for transplantation.[13] One person, with acute fulminant hepatic failure, received a transplant and survived 5 years post transplantation.

Mortality

Mortality data for hepatitis A specifically have been collected by the ABS since 1979. From 1979 to1996, 57 deaths (median 3, range 0 to 6 per year) were attributed to hepatitis A, resulting in an average mortality rate of 0.02 per 100,000 population per year, and a case fatality rate (CFR) of 0.2%. Three deaths occurred in children under 10 years of age, 20 in adults 20 to 59 years and 34 in those aged 60 years or over. CFRs for age groups could not be calculated as age groups for notifications prior to 1991 were not available. The male to female ratio of deaths was 1.4:1, compared to 1.7:1 for notifications. The number of deaths by region was: NSW 22; Queensland 11; and all other States/Territories less than 10.

Discussion

The epidemiology of hepatitis A in Australia has changed dramatically in the last four decades. In the 1950s and 60s notification rates were high, peaking at 123 per 100,000 persons in 1961. These rates fell steadily to low levels through the 1970s and early 1980s. These data need to be interpreted with caution as the fall in notifications may be partly attributable to increased specificity of hepatitis A diagnosis following the introduction of a serological test for the HAV antibody in the 1970s. The 1990s were characterised by low baseline notification rates, with epidemic peaks related to the re-emergence of hepatitis A amongst particular risk groups and a large foodborne outbreak. The peaks may also have been more obvious because of improvements in the surveillance system from the 1980's onward. While the majority of notifications during the 1990s were for adult males, notification rates in children were higher than for adults overall. The NT was the only area with no significant sex difference. The overall epidemiology of hepatitis A in Australia in terms of notification rates and sex differences is similar to that reported in other western countries.[14,15] Rates of hepatitis A mortality and transplantation were very low, in keeping with the known course of illness for hepatitis A. The impact of hepatitis A differed between States and Territories, most notably during peak periods.

In order to assess i) how well the peaks in notification data reflect hepatitis A epidemiology at a State/Territory level; and ii) how the notification data can be used to interpret trends at a national level, the data presented here need to be compared to local reports of hepatitis A outbreaks.

Recent outbreaks

Men who have sex with men

The finding that the adult notifications for males exceed those for females is likely to be a result of a real increase in transmission of HAV between men who have sex with men. Since 1990 there have been two reported major hepatitis A outbreaks in men who have sex with men. The first occurred in 1991 in Victoria, NSW and SA;[4,5,16,17] the second in 1995-96 in south-eastern Sydney.[18] Both these outbreaks were discernible in our analysis of the national notification data (Figure 3). The 1991 peak in notifications (peak1) may be partly attributable to improved reporting of hepatitis A following the introduction of NNDSS. However, the striking adult sex difference in notifications during this time suggests a real increase in adult male HAV infections. The States'/Territories' reports of notification numbers exceeding their respective outbreak threshold levels for adult males during the peak periods also suggests that both outbreaks may have been more widespread than previously reported.

Food-borne

A large scale outbreak associated with contamination of oysters occurred in NSW in 1997.[3] Peak 3 in the national notification data (Figure 3) corresponds with this outbreak, and the dramatic peak (short duration) in above-threshold notifications, moreover across both sexes, is indicative of a common source outbreak. Analysis of the national data found that adults and children were affected by the outbreak and that the outbreak affected residents of a number of States and Territories.

Children

While only 24% of notifications were for children, the notification rate in children was significantly higher than for adults. The true rate of HAV infection in children is likely to be even higher than reported, since infection of children is often asymptomatic or anicteric. A more accurate indication of the role of children in HAV transmission can be ascertained by active case finding. The notification data show that from 1991 to 1997 there were two waves of hepatitis A notifications for children under 15 years of age which occurred in 1991-94 and 1996-97. Considering the extended time for which these elevated rates were reported, the waves are unlikely to be a result of single point source outbreaks. The wave pattern of notification has been previously described[19] and is probably a result of successive cohorts of susceptible children becoming infected. Outbreaks in child care centres and schools, and spread of infection from these places to older siblings and household members have been reported.[14,20-23] These modes of transmission probably account for most childhood infections. The wave pattern may also have occurred as result of distinct outbreaks in separate states overlapping in time resulting in large numbers of cases nationally.

These comparisons show that large outbreaks are discernible through a probability analysis of NNDSS data and that patterns of infection in terms of region, sex and age are reflected in those described at a national level. Analysis of the notification data did indicate that outbreaks might have been more widespread than have been previously reported; however this is difficult to determine without additional risk factor information. Other outbreaks of hepatitis A related to food,[24] institutions,[7] injecting drug use[25] and person to person spread[26] have been reported in Australia. While these small outbreaks were not detected in the present analysis of national-level data, they can in principle be determined by probability analysis at the State and Territory level.

Seroepidemiology

The usefulness of notification data for determining the incidence of HAV infection and anti-HAV seroprevalence is limited by under reporting generally and as a result of asymptomatic and anicteric HAV infection specifically. It has been postulated that the true incidence of clinical hepatitis A in developed countries is at least five times greater than reported, with the prevalence of infection many times higher.[19] As such, seroprevalence studies are a useful adjunct for estimating age-specific incidence and prevalence.

To date there have been no Australia wide HAV serosurveys. Comparison of Australian hospital-based serosurveys conducted in the 1950s to those in the 1970s[27] indicate, as reflected in the notification data, a fall in the transmission of HAV over this time period as seroprevalence in the 1950s was higher than in the 1970s. These surveys also show an age cohort effect to have occurred, as during the 1950s seroprevalence was close to 100% from the age of 41, but 20 years later seroprevalence only reached 100% at age 60 years or older. A hyperendemic pocket has been identified in a rural aboriginal community in the NT.[6] This study found seroprevalence rates of 90% in all age groups. There may also be other areas of the NT in which hepatitis A is highly endemic or hyperendemic. If so, this would account for the high notification rate and lack of sex difference in this territory.

The National Centre for Immunisation Research and Surveillance of Vaccine Preventable Diseases plans to carry out a national HAV serosurvey. Information from such a survey conducted periodically, in conjunction with analysis of notification and other morbidity data, will be important for estimating incidence, determining seroprevalence and targeting and evaluating prevention strategies, such as vaccination.

Future directions

While analysis of the NNDSS data did allow large outbreaks to be detected, the impact of point source versus person-to-person outbreaks of hepatitis A was not discernible due to the lack of risk factor information at a national level. However, a number of characteristics of the NNDSS data can be used to signify possible point-source outbreaks, including their characteristic short outbreak periods, and the spread of infection across the sexes and age groups. Additionally, as postcode information is on the NNDSS dataset, it is possible to do more detailed regional or small-area analyses to investigate possible outbreaks. The probability methodology described in this paper would be well suited to predictive outbreak detection and for setting an alert level to indicate unusually high counts for closer monitoring, and action levels at which point active surveillance or intervention would commence.

A number of risk factors have been documented to be associated with hepatitis A.[14,28] It would be most useful and efficient to collect all risk factor information at a State/Territory level. Cases could then be classified as: food-borne, sporadic, related to another case, travel related, and/or occurring in an aboriginal person. These classifications, along with routinely collected case information, could be relayed to the NCDC to enable outbreaks with a common source or risk factor to be identified across borders and allow national interventions to be instigated if warranted. Collation of this information would also help in developing and evaluating control strategies.

Interventions to control hepatitis A historically have involved improving sanitation and hygiene and control of outbreaks through the use of human immunoglobulin.[29] Recently there has been discussion regarding the use of hepatitis A vaccines for the control of outbreaks, including mass vaccination of defined communities and incorporation into routine childhood vaccination schedules.[30,31] Current surveillance data provides little information regarding groups for whom HAV vaccination should be recommended. The differences between States/Territories in terms of notification rate, sex and age of those notified, suggests that vaccination policies may need to be tailored to regional epidemiology. In conjunction with the surveillance data presented here, risk factor data, timely serosurveillance, and HAV hospitalisation data, are essential for informed HAV vaccination and control policies as we enter the next century.

Acknowledgments

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The epidemiology of acute hepatitis A in North Queensland, 1996-1997

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Abstract

Details on all cases of hepatitis A notified in North Queensland in 1996 and 1997 were prospectively collected. There were two substantial outbreaks and a total of 225 cases during this period. The total incidence rate (per 100,000) was 11.0 in 1996 and 27.0 in 1997. Aborigines and Torres Strait Islanders constituted 29% of cases and had incidence rates of 75.2 and 62.7 per 100,000 for 1996 and 1997 respectively. Thirty-nine cases (17.3%) were admitted to hospital for a total of 202 bed-days and a 4 year old died with fulminating hepatitis. A probable source of infection was identified for 69% of cases. The common risk categories for infection were: living in or visiting a rural Aboriginal or Torres Strait Islander community, injecting drug use, contact with a known case of hepatitis A, and travel to countries with endemic hepatitis A. *Commun Dis Intell* 1999;23:120-124.

Introduction

Infection with the hepatitis A virus (HAV) causes considerable morbidity in North Queensland. (Figure 1 illustrates the geograpical extent of Far North Queensland and the North Queensland Public Health Zone.) For example, Far North Queensland was subjected to a prolonged community-wide epidemic from 1992 to 1994 (Figure 2). During this epidemic numerous episodes of transmission in child day-care centres were documented and many occupational exposures were identified. [1,2,3]

An inactivated hepatitis A vaccine was first licensed in Australia in 1993 and recommendations for its use were subsequently published by the National Health and Medical Research Council (NHMRC).[4] The Tropical Public Health Unit (TPHU) promoted vaccination of at-risk groups, including staff at child day-care centres and some health care providers, in response to the Far North Queensland epidemic.

This prospective study was undertaken to describe the current epidemiology of hepatitis A in North Queensland, and to reassess the risk factors for what is now a vaccine preventable disease.

Methods

The TPHU collected details on all notified cases of hepatitis A in the North Queensland Public Health Zone for 1996 and 1997 (Figure 1). The Zone has a population of 592,000, 8.1% of whom are Aborigines or Torres Strait

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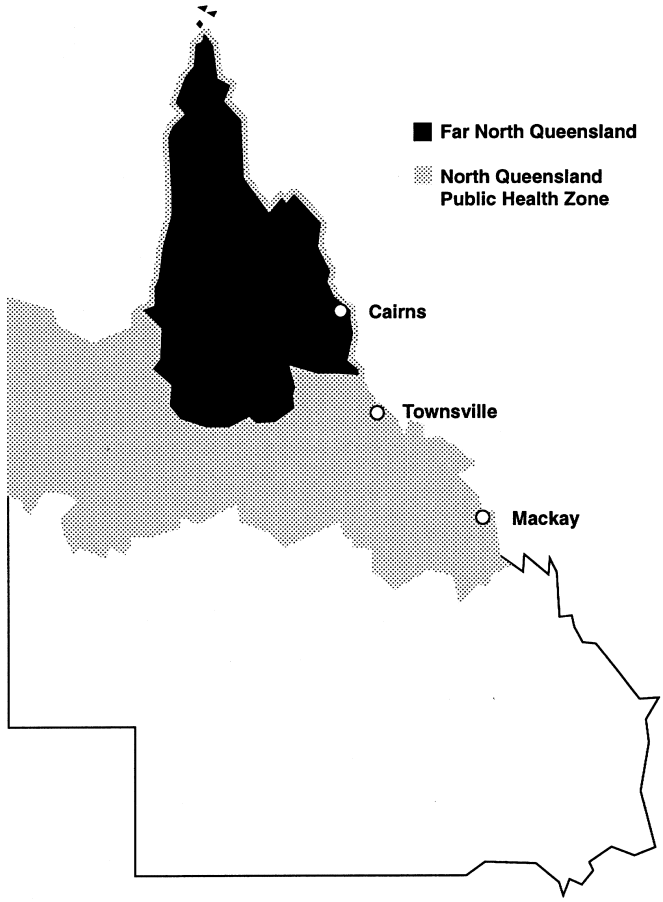
Figure 1. Map of North Queensland



Islanders. [5] Acute hepatitis A is a notifiable disease in Queensland and all public and private laboratories in North Queensland are therefore required to notify the TPHU when IgM antibodies to hepatitis A (anti-HAV IgM) are detected in serum. In addition, clinicians are requested to report any clinical case of acute hepatitis, regardless of whether confirmatory serology is available. All cases in this report were thus confirmed by the presence of anti-HAV IgM in the absence of recent vaccination, or had an illness consistent with hepatitis and an epidemiological link to a serologically confirmed case.[6]

The staff at TPHU contacted the treating doctor of each notified case and, when possible, interviewed the patient directly. The following details were sought: onset date of illness, age, sex, race, address, occupation, potential risk categories for infection,[7] management, immediate health outcome and contacts eligible for hepatitis A prophylaxis. Normal human immunoglobulin (NHIG) was provided to contacts in conjunction with the treating doctor and further public health measures were instituted as required.

Incidence rates were calculated for the total and the indigenous population in North Queensland using

Figure 2. Notifications of hepatitis A in Far North Queensland, by quarter of year

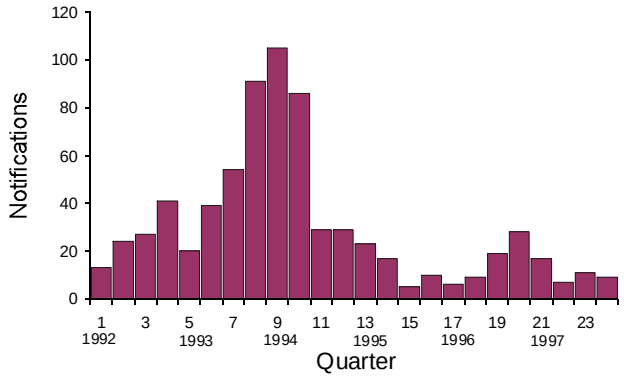


Figure 3. Notifications of hepatitis A, North Queensland, 1996-97

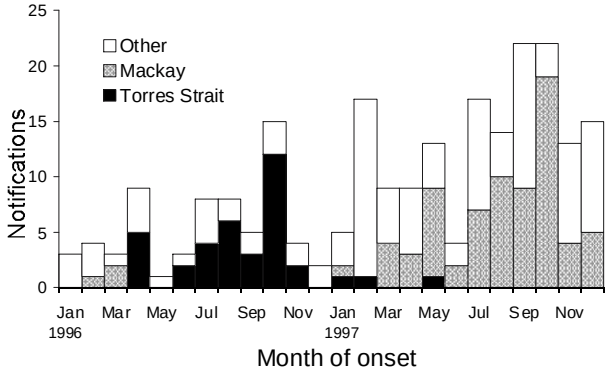
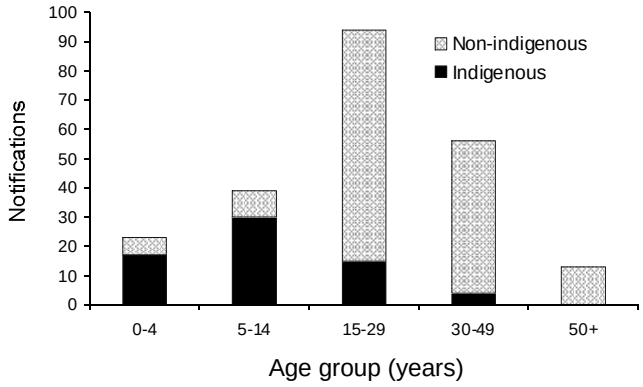


Figure 4. Notifications of hepatitis A, North Queensland, 1996-97, by age and indigenous status



denominator data from the 1996 Australian Bureau of Statistics Census.[5]

Results

A total of 225 cases were notified; 65 in 1996 and 160 in 1997. Total incidence rates were thus 11.0 and 27.0 per 100,000 persons for 1996 and 1997 respectively. Only one case was not serologically confirmed. In addition to sporadic cases, there were two substantial outbreaks; one in the Torres Strait during 1996 (34 cases) and a second in Mackay during 1997 (72 cases) (Figure 3). There were also discrete clusters in two rural Aboriginal communities during 1997, both resulting in 9 notifications. The majority of cases were Caucasian (151), but Aboriginal and Torres Strait Islanders were clearly over-represented as they accounted for 29% (66) of all cases. Incidence rates for indigenous persons were 75.2 and 62.7 per 100,000 persons for 1996 and 1997 respectively.

Hepatitis A was more common in children and young adults, and the mean age of Aboriginal and Torres Strait Islander cases (12.6 years) was significantly lower than that of non-indigenous cases (30.0 years, $p<0.001$) (Figure 4). There were more male than female cases (140 and 85 respectively).

Thirty-nine cases (17.3%) were admitted to hospital, for a total of 202 bed days (median length of hospital stay 4 days, range 1-18 days). Four people required transfer from a peripheral hospital to a regional base hospital and a 45 year old Australian resident was evacuated by air from Papua New Guinea (PNG) to Cairns acutely ill with hepatitis A.

There was one death during 1997. A 4 year old Aboriginal boy developed severe hepatic encephalopathy due to fulminating hepatitis A and died in the Royal Childrens Hospital in Brisbane before liver transplantation was possible.

Risk categories indicating a probable source of infection were identified for 69% of cases, a possible source for a further 2%, and for 29% the source was unknown. Some cases had more than one risk factor identified (Table 1). For example, among those who had contact with a known case, 17 were also identified as injecting drug users, 7 were in the rural indigenous group, 2 had travelled overseas to HAV endemic areas and 1 had child day-care contact. Only a few cases in other groups had multiple risk factors. People were allocated to risk categories on the basis of available information and misclassification may have occurred for some cases. For example, it is likely that some cases had unrecognised contact with an HAV-infected person and that not all injecting drug users (IDUs) were identified.

The most common risk category indicating a probable source of infection was either living in or visiting a rural Aboriginal or Torres Strait Islander community. The majority of people infected in these communities were

Table 1. Risk category for source of hepatitis A infection, North Queensland, 1996-97

Probable source of infection	Number	Percent
Rural indigenous community resident or visitor	59	26
Contact with a known case	43	19
Injecting Drug Use	41	18
Overseas travel to an endemic country	17	8
Urban indigenous community resident	10	4
Child day-care contact (children, staff and parents)	10	4
Homosexual male	4	2
Oysters (Wallis Lake outbreak)	4	2
Sewage exposure	1	0
Total cases with one or more probable risk categories	155	69
Possible source of infection		
Cleaner	5	2
Unknown source	65	29
Total notified cases	225	100

local indigenous residents, but two non-indigenous residents (a doctor and a hospital clerk) and three visitors also acquired HAV infection. Other common risk categories were injecting drug use (IDU), contact with a known case of hepatitis A, and travel to overseas countries with endemic hepatitis A.

A total of 41 cases were identified as IDUs, 34 of whom were involved in the Mackay outbreak. A variety of different drugs, including amphetamines and heroin, were injected by the affected IDUs and no common drug source was identified. Very few cases reported sharing needles, but many described sharing other objects such as cigarettes and bongs. Many lived in houses with other IDUs. During 1997 several other outbreaks of HAV infection in IDUs were identified in southern Queensland but no epidemiological links with the Mackay outbreak were established.

Seventeen cases probably acquired their infection overseas. None had received HAV vaccination or NHIG prior to travel. The most common regions implicated were Papua New Guinea (9 cases) and South East Asia (3 cases).

Other risk categories accounting for small numbers of cases included urban indigenous residents, association with child day-care centres, homosexual males, eating oysters implicated in an outbreak [8] and exposure to sewage. Cleaners could feasibly be exposed to HAV in their workplace and were over-represented as an occupational group among cases; they were included as a possible risk category.

Discussion

Hepatitis A results in considerable morbidity and expense for the North Queensland community. The death of a 4 year old child during this period also indicates how serious this infection can be. While the reported case fatality rate for HAV infection is low (<1/1000), higher rates have been reported in children under the age of 5 years (1.5/1000) and adults over the age of 50 years (27/1000).[9] As a safe and effective vaccine is available, there is a need to consider whether more can be done to prevent HAV infection in north Queensland.

This report identifies a number of key population groups that continue to be at increased risk of contracting HAV infection. These include rural and urban indigenous community residents and visitors, IDUs and people travelling to endemic regions overseas.

Many rural Aboriginal and Torres Strait Islander communities have endemic HAV infection. Nearly all residents (98.5%) from a sample of remote Aboriginal communities in the Northern Territory (NT) were immune to HAV by the age of 10 years when assessed in 1994.[10] In such endemic circumstances HAV infection is usually acquired in early childhood and confers life-long immunity. Acute infection at this early age is often mild and anicteric, thus the disease may be largely invisible despite very high real incidence rates. Clinical notifications will considerably under-estimate infection in such communities.

Paradoxically, hepatitis A initially becomes a more obvious problem as communities experience lower incidence rates in response to changes such as improved sewage disposal, housing and water supply. Fewer early childhood infections result in a susceptible pool of older children, adolescents and adults, and infection in this older group is more likely to be clinically apparent. The outbreak that occurred in the Torres Strait during 1996 and the clusters of cases seen in several rural communities during 1997 probably reflect this transitional process. Further outbreaks and clusters can be expected as this transition continues.

The hepatitis A vaccine has been used to interrupt transmission in communities with high incidence rates but its use needs to be tailored to specific situations. Following the NT seroprevalence study noted above, it was concluded that vaccination was not indicated in those communities as only 1.5% of people over the age of 10 years of age were susceptible to HAV.[10]

There is thus a need for appropriate seroprevalence studies before considering an immunisation program in indigenous communities in North Queensland. Other issues such as the expense of the vaccine and the logistics involved in maintaining such a program would also need to be considered. Community-wide HAV vaccination programs often target young children (aged 3 to 5 years) on the basis that they are a vulnerable group, and because they are recognised as key transmitters of disease within a community.[11]

Non-indigenous staff employed in indigenous communities are also at risk of HAV infection. The NHMRC currently recommends HAV vaccination for teachers and health staff in remote indigenous communities,[12] and this should be arranged prior to their arrival in the community.

Injecting drug use is well recognised as a risk category for HAV infection [7, 13] but had not previously been associated with an outbreak in North Queensland. The exact mechanism by which infection is acquired is unclear, but is likely to involve the associated lifestyle and possibly the use of shared equipment such as bongs. Although the NHMRC recommends that IDUs be considered for HAV vaccination,[12] they are likely to be a difficult group to access.

The number of cases in travellers to endemic areas is of concern. Hepatitis A is the most frequent infection in travellers that can be prevented by immunisation,[14] and it should be possible to more readily identify and target this group for vaccination prior to travel.

Ten cases were associated with child day-care centres; 4 were children in care, 1 was a day-care staff member and 5 were parents of children in care. All were isolated cases and all but four had another risk factor. There were no outbreaks in child day-care centres, which is in marked contrast to the 1992-94 epidemic in Far North Queensland.[1,2]

There were 4 cases attributed to oysters from the Wallis Lakes in New South Wales, but no other food-borne cases were identified.

In conclusion, we have identified a number of important risk factors for HAV infection in North Queensland. There is a need for greater use of the HAV vaccine to protect at risk groups identified in the current NHMRC guidelines including IDUs and travellers to endemic areas. Vaccination also needs to be further considered for some indigenous groups.

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Report of the Australian National Polio Reference Laboratory
1 January to 31 December 1998

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Abstract

The Australian National Polio Reference Laboratory was established at the Victorian Infectious Diseases Reference Laboratory (VIDRL) in late 1994 to carry out virological confirmation of the eradication of poliomyelitis in Australia. The laboratory is responsible for transporting samples from all Australian patients with acute flaccid paralysis (AFP) to VIDRL for poliovirus culture, identification and intratypic differentiation. The laboratory also performs polio serology on selected serum samples from AFP patients when faecal samples are not available. In 1998, faecal specimens were received from 11 patients with AFP. Adenovirus type 2 was isolated from 1 patient and an untypable non-polio enterovirus from another. No viruses were isolated from the other 9 patients. Since 1995, over 820 isolates have been transported to VIDRL from laboratories in five Australian states for testing. Three hundred and seventy three (45%) were confirmed as Sabin vaccine-like polioviruses, 416 (51%) were non-polio enteroviruses and 24 (3%) yielded no virus or viruses other than enteroviruses. Eight polioviruses are still uncharacterised. *Commun Dis Intell* 1999;23:124-128.

Introduction

In 1988, the World Health Assembly passed a resolution which committed the World Health Organization (WHO) to the global eradication of poliomyelitis by the year 2000. The eradication strategy is three-fold: routine and supplementary immunisation, surveillance and, where required, outbreak response.[1,2]

Almost all countries of the six WHO regions are committed to polio eradication. Each country is required to verify the absence of wild poliovirus circulation in the presence of high quality surveillance. Acute flaccid paralysis (AFP) has been proven to be a sensitive indicator for detecting wild poliovirus and was used successfully in the Americas prior to certification of that region as being free of wild poliovirus.[3] In a country where polio is not endemic, there is normally a background incidence of at least one AFP case for every 100,000 children under 15 years of age per year. Surveillance of cases of AFP by most recently endemic and some non-endemic countries of the Western Pacific has resulted in the documentation of AFP rates of at least one per 100,000.[4] Based on Australia's population

distribution, there are likely to be approximately 40 children with AFP each year.[5]

The laboratory plays a crucial role in surveillance as AFP may have many aetiologies. Wild poliovirus infection can only be confirmed by virological investigation. Faecal samples collected within 14 days of onset of paralysis are transported to a WHO approved laboratory for enterovirus isolation and identification. If poliovirus is isolated, the strain is characterised as Sabin or wild type.

This report describes the functions of the polio reference laboratory and its activities during 1998.

Terms of reference of the Australian National Polio Reference Laboratory

The Australian National Reference Laboratory at the Victorian Infectious Diseases Reference Laboratory (VIDRL) was established in late 1994. It is one of three WHO Western Pacific regional reference laboratories, the other two being in Tokyo and Beijing. Faecal samples from all reported AFP cases in Australia are transported to VIDRL and cultured for entero and poliovirus. Poliovirus strains isolated from these samples, and those isolated from non-AFP patients by virus laboratories throughout Australia are then referred to VIDRL, and identified and characterised as wild or Sabin vaccine-like. Polio neutralisation antibody tests are carried out on selected serum samples and for serosurveys.

Implementation of the terms of reference

Acute Flaccid Paralysis surveillance was commenced in Australia in March 1995 through the Australian Paediatric Surveillance Unit (APSU), the National Centre for Disease Control (NCDC) and the Australian National Polio Reference Laboratory at VIDRL. Prior to May 1996, AFP faecal samples were cultured in virology laboratories in Australian states and, if polio strains were isolated, these were referred to VIDRL. Since May 1996 all AFP faecal samples have been processed at VIDRL which is accredited by WHO as both a regional and national polio reference laboratory. The process of accrediting all state virology laboratories was not feasible considering the small numbers of specimens expected.

When VIDRL is notified of an AFP case under investigation or if enterovirus isolates are ready for shipment, the appropriate instructions for packing and, if necessary, containers and documentation are provided to

Table 1. Samples received for enterovirus culture from Australian AFP cases in 1998

State/City/District	Epid No.	Date received	Result
AFP			
Papua New Guinea/TSI	Aus001/98	Anal25-02-98	Negative
		TS 25/2/98	Negative
		F 25/2/98	Negative
		F 25/2/98	Negative
		F 25/2/98	Negative
NSW/ Hunter Valley	Aus002/98	F 3/4/98	Adeno 2
		F 3/4/98	Negative
Vic/Belgrave	Aus003/98	F 30/4/98	Negative
ACT/Canberra	Aus004/98	F 7/5/98	Negative
		F 7.5.98	Negative
Qld/Brisbane	Aus005/98	F 21/5/98	Negative
Qld/Cairns	Aus006/98	F 25/8/98	NPEV
NSW/Tweed Heads	Aus007/98	F 25/8/98	Negative
		F 25/8/99	Negative
Qld/Tugun	Aus008/98	F 23/9/98	Negative
		F 23/9/99	Negative
SA/Adelaide	Aus/009/98	F 02/10/98	Negative
SA/Adelaide	Aus/10/98	F 02/10/99	Negative
		F 13/10/98	Negative
Qld/Townsville	Aus/011/98	F 21/10/98	Negative
NON AFP			
Vic/Melbourne		F26/8/98	Negative

the requesting laboratory by VIDRL. The laboratory is instructed to pack the samples and then contact the laboratory's preferred carrier who collects these containers and ships them overnight to VIDRL for testing.

Faecal samples are processed and inoculated into the WHO-supplied cell lines, HEp2, RD and L20B. The use of the L20B cell line, which is a mouse cell line with receptors for human poliovirus, facilitates the preliminary separation of polio and most non-polio enteroviruses which have been isolated from AFP specimens or submitted from laboratories in all Australian states. The L20B positive strains are identified using type-specific poliovirus antisera and, if polioviruses, are characterised as Sabin vaccine or wild type using nucleic acid probe hybridisation (NAPH). The L20B negative virus isolates from all AFP samples are fully identified while those submitted from other laboratories are identified on request.

Polio antibody testing is performed only on samples from patients with paralysis when faecal samples have not been collected within 14 days of onset of paralysis. The interpretation of results may be difficult as type-specific antibodies are often raised at the time of onset of symptoms. In addition, the test does not distinguish between antibodies against wild and vaccine virus. Serosurveys are carried out in collaboration with researchers usually as part of immunisation studies.[6]

During 1997, a Polio site on the internet was established (*www.vidrl.org.au*). The information available includes global, regional and Australian updates on poliomyelitis eradication; AFP surveillance in Australia; the polio laboratory network; instructions for collection and transportation of stool samples and results of testing on enteroviruses referred to VIDRL for intratypic differentiation. The homepage has links to other related global sites and is up-dated annually and when new information is available.

The annual *Australian Polio Network Newsletter*, which contains similar material to that found on the homepage, is posted to Australian laboratories involved either with enteroviruses or which are likely to transport faecal samples and isolates to VIDRL for polio testing. The newsletter is also available to interested public health and other medical personnel (see below for subscription address).

Table 2. Cumulative summary of identification of enteroviruses and intratypic differentiation of polioviruses from Australian laboratories tested at VIDRL from 1995 to 1998

State	Year	Polio Sabin Like	Polio Pending ITD	Reovirus	Non-Polio Enterovirus	Herpes Simplex/ Negative	Total
Victoria	1995	9					9
	1996	16	1				17
	1997	5	1				6
	1998	7					7
Queensland	1995	41			5	8	54
	1996	98	1		4	9	112
	1997	40	1				41
	1998	8			15	2	25
Western Australia	1995-6	125	1	5	359		490
	1997	9			33		42
	1998						0*
Tasmania	1995	1					1
	1996	3					3
	1997	3	1				4
	1998	3	1				4
NSW	1995-8						0**
South Australia	1997	3					3
	1998	2	1				3
Total	1995-1998	373	8	5	416	19	821

* PCR has replaced culture for enteroviruses, so isolates are no longer available.

** Polioviruses stored for shipment in 1999

Activities in 1998

During 1998, 19 faecal samples, one anal swab and one throat swab were received from 11 patients with AFP (Table 1). A faecal sample was also cultured to detect viral shedding in a Victorian transplant patient who had been given Sabin vaccine. Four AFP patients were from Queensland, two each from New South Wales (NSW) and South Australia and one each from the Torres Strait Islands, Victoria and Canberra. Duplicate samples were collected from only seven of the eleven patients. No samples yielded polioviruses and 20 of the 22 samples did not yield any virus. Adenovirus type 2 was isolated from the initial sample from one patient in NSW (Aus002/98). An enterovirus, isolated from a patient in Cairns in August, (Aus006/98) could not be identified with available antisera and will be further investigated using molecular techniques.

The first case (Aus001/98) was one of three young men from the same village on the south coast of Papua New Guinea who often travelled in the Torres Strait and visited islands there. All young men developed AFP in late 1997 or early 1998 and were admitted to hospitals in Port Moresby and Thursday Island. No samples were available from the first patient. Serum samples were collected from the second patient 5 days after admission and 31 and 91 days later. Rising antibody levels were detected between days 5 and 31 to all three polioviruses. The interpretation of these findings is uncertain. It suggests that this patient received polio vaccine after the first bleed was taken. However it is known that this did not occur as he was in the Thursday Island Hospital during this time (personal communication, Dr Jeffrey Hanna, Tropical Public Health Unit, Cairns). It appears that there may have been a mixup with the first sample which was transported via several pathology departments to VIDRL. Four stool samples and a throat and anal swab from the third patient were negative for enteroviruses. Serum samples collected from this patient on admission and after 3, 25 and 69 days showed elevated but stationary antibody levels to types 1, 2 and 3, suggestive of past immunisation or infection.

Fewer polio and enterovirus isolates were submitted for identification and characterisation in 1998 than in 1996 and 1997. The State laboratories in Western Australia and Queensland have introduced molecular methods for enterovirus detection and no longer culture samples for enterovirus isolation. The State laboratory in NSW has stored all polioviruses identified since 1995 and plans to ship them to VIDRL in early 1999. During 1998, of 39 'enteroviruses' submitted, 22 were polioviruses (Table 2). Twenty poliovirus isolates were confirmed as poliovirus Sabin-like and intratypic differentiation results are still pending for two. Echovirus types 6 (two isolates) and 19 (11 isolates) were cultured at the Queensland Health Scientific Services Laboratory in Brisbane from healthy children who lived in the same village in Papua New Guinea as the three young men with AFP. Coxsackievirus type A9 was isolated from two samples from another Queensland patient and two did not yield virus.

Further intratypic differentiation and enterovirus identification has been performed on isolates submitted in 1996 and 1997 (Table 2). Overall since early 1995, there were 373 (45%) Australian polioviruses characterised as Sabin-like, 416 (51%) were non-polio enteroviruses and 19 (3%) were enterovirus negative or herpes simplex virus.

Five isolates submitted from Western Australia in 1995-96 which produced a non-enterovirus cytopathic effect in L20B cells were confirmed by electron microscopy as reoviruses. There is some evidence that the eight polioviruses still uncharacterised are Sabin vaccine-like but further testing is necessary as titres were low or mixtures were detected.

Neutralisation tests to detect poliovirus antibodies were performed on serum samples of three Australian patients with suspected paralysis. Only single bleeds were collected. All patients had elevated antibody levels to poliovirus type 1, 2 or 3 which may suggest past immunisation or infection.

Discussion

Certification of non-endemic industrialised countries was discussed at the second meeting of the Global Commission for the Certification of the Eradication of Poliomyelitis in Geneva 1997.[7] The commission reaffirmed that the absence of wild poliovirus in the presence of high quality routine AFP surveillance among children aged less than 15 years should be regarded by all countries, regardless of their endemic status, as the gold standard of polio eradication. The WHO has set standard performance indicators for AFP surveillance. Reports must be timely and reporting sites must represent the geography and demography of the country. The surveillance system should be sensitive with all AFP cases being investigated soon after onset of symptoms and there should be a follow-up examination after 60 days. At least 80% of AFP cases should have two adequate stool specimens collected and tested in an accredited laboratory.[8]

Although some non-endemic countries have decided that it is impracticable to establish routine AFP surveillance, Australia has chosen to do so.[9] Since the last case of poliomyelitis was most likely to have occurred in the 1970s,[10] it is unlikely that indigenous wild poliovirus transmission will be detected. However there should still be a background of about 40 cases of AFP in children, mainly due to Guillain-Barre syndrome and transverse myelitis. Since AFP surveillance commenced in Australia in 1995, reporting of AFP cases and stool sample collection has not reached the WHO targets. Efforts are being made to address this problem.[9]

Under-reporting of AFP cases has been demonstrated in a recent Victorian study. The two-source capture-recapture method was used to estimate ascertainment of AFP cases in Victoria. The APSU study was used as the primary source and children admitted to two major teaching hospitals as the secondary source.[11] Only 27% of patients with symptoms of AFP had been reported to APSU in Victoria between 1995 and 1997.

There has been a positive response from staff in laboratories in hospitals around Australia who have been contacted by letter and telephone to advise them of the program. On some occasions, these scientists have been responsible for alerting clinical staff of the need to report AFP cases to the APSU and the NCDC.

The state virology laboratories have also supported VIDRL in its task to characterise all polioviruses isolated since 1995. It is fortunate that the program for characterisation of isolates was well established in 1995 since with the

introduction of molecular methods for enterovirus detection, there would be fewer isolates available in future years. To date, nearly 400 poliovirus isolates have been proven to be Sabin vaccine-like. Once the large number of isolates from NSW has been screened, over 950 enteroviruses will have been tested to exclude wild poliovirus.

With the expected global eradication of wild poliovirus by the end of the year 2000 or 2001, the next task for laboratories will be to either transfer wild poliovirus strains to designated repositories or destroy them. The continued cooperation of virologists in Australia will be sought to carry out this task.

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Acute Flaccid Paralysis surveillance in Australia Progress Report 1995-1998

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Introduction

The World Health Organization (WHO) aims to eradicate poliomyelitis from the Western Pacific Region by the year 2000. As part of the certification process, active surveillance of acute flaccid paralysis (AFP) was initiated in Australia in March 1995.

While it is unlikely that Australia does have indigenous wild poliovirus, adequate investigation of all cases of AFP is required for WHO certification. A number of indicators have been established to monitor the performance of AFP surveillance systems.[1] Most importantly, even in the absence of wild poliovirus circulation, surveillance systems should be capable of detecting at least one case of AFP per 100,000 population aged less than 15 years or 40 cases per year in Australia. Secondly, at least 80% of AFP cases should have two specimens collected within 14 days of onset of paralysis, 24 hours apart, even if the clinician involved is confident of an alternative diagnosis. Stool specimens must be tested for poliovirus in a WHO accredited laboratory which for Australia is the Victorian Infectious Diseases Reference Laboratory. Stool testing also has the additional advantage of identifying other viruses which can cause poliomyelitis-like illness.

The objectives of this project are:

- to determine the incidence, aetiology and clinical picture of AFP in children under 15 years, and

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- to determine whether paralytic 'wild' poliovirus has been eradicated from Australia.

Methods

Active AFP surveillance was initiated in March 1995 thorough the Australian Paediatric Surveillance Unit (APSU). The APSU sends out a monthly report card to all paediatricians and asks them to indicate the number of AFP cases they have seen in the last month. Conditions which may present as AFP include wild and vaccine-acquired poliomyelitis, Guillain-Barré syndrome or transverse myelitis or traumatic paralysis. Paediatricians are also requested to report all cases of AFP by telephone to the National Centre for Disease Control (NCDC). Clinicians arrange for collection of stool specimens and provide further clinical and laboratory information on the case by postal questionnaire. In addition a 60-day follow-up questionnaire is sent to paediatricians to ascertain the presence of residual paralysis.

Case Definition

Any child aged less than 15 years with:

acute onset of flaccid paralysis in one or more limbs or acute onset of bulbar paralysis.

Results

There were 171 reports of AFP for the period March 1995 to December 1998, for which further information was available on 145 (85%). Of these, 27 were duplicate reports and 4 were errors. One hundred and eleven cases of AFP were confirmed, of which 30 occurred in 1995, 24 in 1996, 26 in 1997 and 31 in 1998. 'Confirmed' cases are those reported to the APSU and confirmed by information supplied in the questionnaire. Three cases aged over 15 years were excluded for the purposes of calculating incidence, which was estimated at 0.71/100,000 children less than 15 years old for the study period. This may be an under-estimate as further clinical information is currently unavailable on 6 cases reported in 1998. The annual reported incidence of AFP is shown in Table 1.

The ages of the AFP cases ranged from 2 months to 15 years. Fifty-eight per cent of the cases were male. One-hundred and five cases were hospitalised and 35

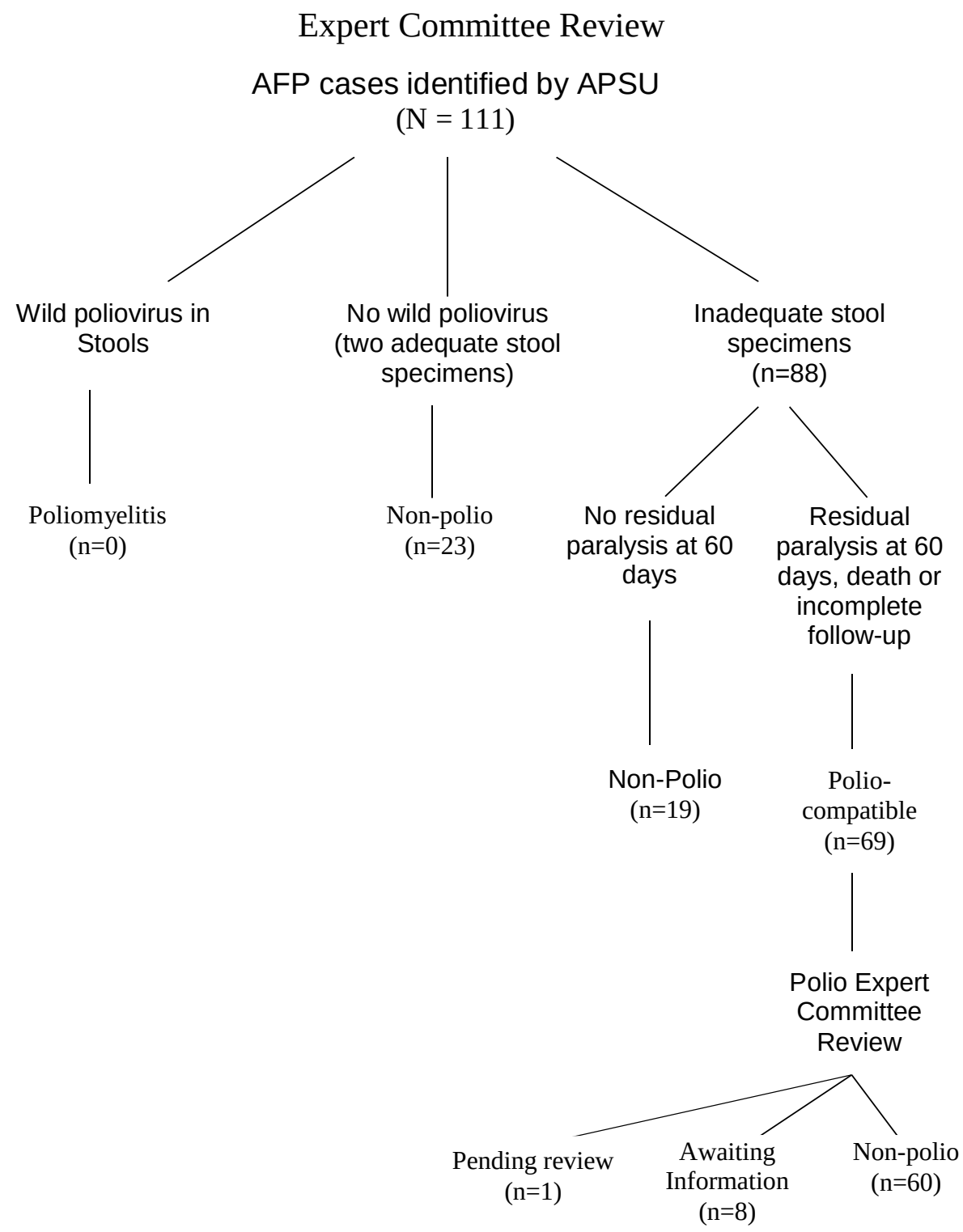
Table 1. Annual reported incidence of AFP/100,000 children < 15 years of age

Year of Diagnosis	Confirmed cases	Incidence (95% CI)
1995	30	0.9
1996	24	0.6
1997	26	0.66
1998	31	0.79
1995-8	111	0.71

Table 2. Diagnoses of 111 cases of AFP, March 1995 - December 1998

Diagnosis	N	%
Guillain-Barre syndrome	52	46.9
Transverse myelitis	21	18.9
Trauma	5	4.5
Encephalomyelitis / myelitis	5	4.5
Demyelination (includes 1 with viral encephalitis and 1 post- viral demyelination)	5	4.5
Ischaemic cord damage	4	3.6
Tick bite paralysis	3	2.7
Spinal cord damage/spinal surgery	3	2.7
X-linked recessive remittent /acquired myaesthenia gravis	2	1.8
Myelitis secondary to mycoplasma infection	1	0.9
Polio like illness due to enterovirus (Echo 9)	1	0.9
Infant botulism	1	0.9
Lumbar radiculopathy	1	0.9
Hypotensive brain stem necrosis or demyelination	1	0.9
Post drug polyneuromyopathy	1	0.9
Post intensive care polymyopathy	1	0.9
Neuroblastoma	1	0.9
Post-viral myositis	1	0.9
Not specified	2	1.8
Total	111	100.0

Figure 1. Virological classification of AFP cases[2] and case outcomes following Polio Expert Committee Review



required intensive care admission. Information is not available on hospitalisation status of 3 cases while another 3 cases were not hospitalised.

Follow-up information on the presence of residual paralysis at 60-days after diagnosis is available on 50 (45%) of the 111 cases. This information was available for 48% cases notified in 1998, 65% cases in 1997, 29% of cases notified in 1996 and 36% of cases notified in 1995. Of the 50 cases for whom follow-up information was available, 25 (50%) had residual paralysis at 60 days and one child with Transverse Myelitis died. Stool testing in accordance with the study protocol was undertaken in only 23 (21%) cases.

Diagnoses of the 111 confirmed cases are shown in Table 2. There were no cases of poliomyelitis identified during the study period. According to WHO's virological classification 42 (38%) of cases would be classified as 'non-polio' cases either because they had two poliovirus negative stool cultures *or* because there was no residual paralysis at 60 day follow-up. However, using this classification, 69 (62%) would be classified as 'polio compatible' because of incomplete follow-up data or failure

of provision of specimens for stool culture (Figure 1). Following review by the Polio Expert Committee, 60 of the 69 (87%) of the 'polio compatible' cases were reclassified as 'non-polio' based on information provided by the reporting paediatrician and the investigating laboratory.

In summary, 102 (92%) of the 111 confirmed AFP cases have been classified as 'non-polio.' One case is pending review by the Polio Expert Committee and for 8 cases further clinical and laboratory information which could allow them to be reclassified as 'non-polio' is not yet available.

Discussion

There have been no identified cases of AFP due to 'wild' or vaccine poliovirus in Australia during the study period. The surveillance has provided a large number of AFP cases especially Guillain-Barré Syndrome and Transverse Myelitis and provided information on their aetiology.

The surveillance system is a very useful part of the WHO Polio Eradication certification process. However, incomplete information on cases is a continuing problem. As polio has not been seen in Australia for over 20 years, it is sometimes not considered as a differential diagnosis in a child with AFP and appropriate stool testing may not be undertaken. Similarly paediatricians may omit to report cases of AFP when the child is proved to have an alternative diagnosis to polio. This may result in under reporting of AFP cases to APSU.

In cases of AFP where the stool sampling was inadequate for WHO's requirements, the Polio Expert Committee has been able to review case notes and classify the majority of these cases as non-polio.

Retrospective searches of in-hospital databases are currently being undertaken to identify additional cases of AFP not reported to the APSU. Medical records of these cases will be reviewed for clinical and laboratory information and the Polio Expert Committee will use this information to classify these cases as polio compatible or non-polio. The combination of active surveillance and hospital searches will enable Australia to meet the WHO certification criteria to be declared polio-free. Although it is anticipated that retrospective hospital searches will increase the number of cases identified, it is time consuming, costly and unlike active surveillance, does not allow timely investigation of AFP cases.

If Australia is to be declared polio-free, it is vital that all cases of AFP are reported promptly to APSU and NCDC, that questionnaires are returned and that stool samples are collected from all cases, including those in whom poliomyelitis is not considered to be the diagnosis. Clinicians can assist by ensuring that the specimens are ordered in accordance with the study protocol and that hospital laboratories forward stool specimens and all poliovirus isolates whatever their source to the Victorian Infectious Diseases Reference Laboratory.

Acknowledgment

We acknowledge the contribution made by Dr Ana Herceg in her role as chief investigator from 1995 to 1997. The authors thank the APSU for facilitating this study, all paediatricians who reported and investigated cases of AFP and the staff of the National Polio Reference Laboratory and other laboratories in Australia for collection, transport and processing of stool and serum samples. APSU is funded by the Financial Markets Foundation for Children. We also thank the Polio Expert Committee for reviewing and classifying AFP cases according to WHO's virological classification and Dr Jennifer Peat for assistance with data analysis.

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1. Regional Commission (Regional Poliomyelitis Eradication Certification issues) WPR/VID/EPI(3)97.

Communicable Diseases Surveillance

Highlights

Communicable Diseases Surveillance consists of data from various sources. The National Notifiable Diseases Surveillance System (NNDSS) is conducted under the auspices of the Communicable Diseases Network Australia New Zealand. The *CDI* Virology and Serology Laboratory Reporting Scheme (LabVISE) is a sentinel surveillance scheme. The Australian Sentinel Practice Research Network (ASPREN) is a general practitioner-based sentinel surveillance scheme. In this report, data from the NNDSS are referred to as 'notifications' or 'cases', whereas those from ASPREN are referred to as 'consultations' or 'encounters' while data from the LabVISE scheme are referred to as 'laboratory reports'.

Vaccine Preventable Diseases

Pertussis

Pertussis notifications remain lower than those seen in the epidemic period of late 1997 and early 1998. The number of notifications with onset in February is the lowest for any month since July 1997. For the current reporting period, the male to female ratio is 1:1.36 and the majority of cases are in the 0 to 4 (13%), 10 to 14 (11%) and 15 to 19 (11%) age groups.

Measles

The number of measles notifications is higher than for the same period of last year, reflecting the outbreak in Victoria which started on 11 February 1999 (Figure 1). Current figures at 7 May 1999 are:

- 74 cases have been reported to the Victorian Department of Human Services;
- 28 of these have been hospitalised;
- 63 cases are in the 18 to 30 year age groups;
- 5 cases of vaccine failure have been identified (all had received one dose of a measles-only vaccine);
- 7 cases are in children aged 8 years or under, all unimmunised.

The frequency of laboratory confirmed cases appears to be decreasing with now only isolated chains of transmission, mainly occurring outside of metropolitan Melbourne (personal communication, Dr Stephen Lambert, Victorian Department of Human Services).

Arbovirus infections

Ross River Virus infection notifications are higher than for the same period of last year with a total of 765 received for this period. The highest numbers are from the Statistical Divisions of Brisbane (292 notifications) and Moreton, Queensland (122).

For the current reporting period the male to female ratio is 1:1.1 and the age distribution follows a bell shaped curve with a flattened peak at the three age groups from 35 to 49 (107 to 110 cases each).

Zoonoses

Large numbers of notifications of leptospirois continue to be received particularly from the Statistical Division (SD) of Far North Queensland (Figure 2).

In the 1997 Annual Report of the National Notifiable Diseases Surveillance System, a total of 126 cases of leptospirosis were reported for Australia with the highest reporting SD being Far North Queensland with 19 cases. In 1998, Far North Queensland reported 62 cases out of an Australian total of 192 and for the year to date for 1999, Far North Queensland has reported 82 cases (Australian total 149). In this reporting period, 37 of the Australian total of 51 notifications have been received from Far North

Figure 1. Notifications of measles, Australia, 1992 to 1999, by month of onset

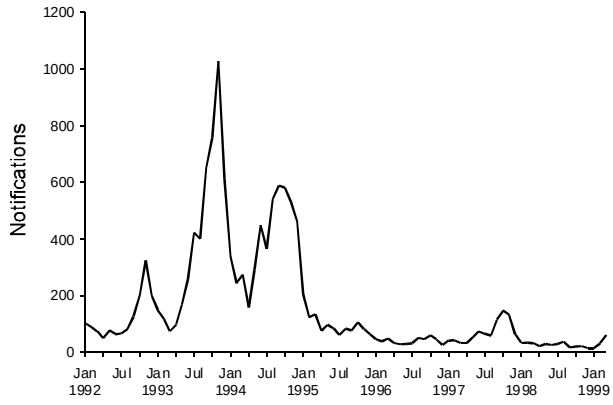
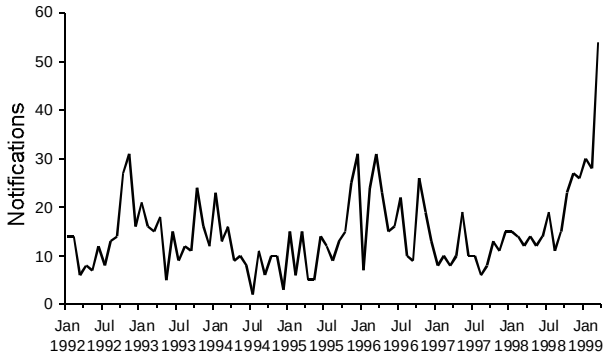


Figure 2. Notifications of leptospirosis, Australia, 1992 to 1999, by month of onset



Queensland. For the current reporting period all but 3 notifications are in males and 71% are from the 20 to 44 year age groups.

It is likely that the high numbers are associated with the prolonged and heavy wet season in Far North Queensland this year. The floods that followed Cyclone Rona in the Innisfail-Ingham area have probably also contributed (personal communication, Dr Jeffrey Hanna, Tropical Public Health Unit, Cairns). An association between heavy rainfall and floods and an increased incidence of leptospirosis is well recognised.[1]

It presents a difficult public health problem as the prevailing conditions clearly cannot be controlled and leptospirosis infection can result in severe disease.[2] While education of at-risk workers is a possible intervention there is no guarantee that such intervention would change behaviour.

The 1997 population estimates for the Statistical Divisions mentioned in this report are:

Brisbane	1,548,346
Moreton	639,024
Far North Queensland	215,518

References

1. Trevejo, RT, Rigau-Perez JG, et al. Epidemic leptospirosis associated with pulmonary hemorrhage-Nicaragua, 1995. *J Inf Dis* 1998;178:1457-63.
2. Simpson, FG, Green KA, et al. Leptospirosis associated with severe pulmonary haemorrhage in Far North Queensland. *Med J Aust* 1998;169:151-3.

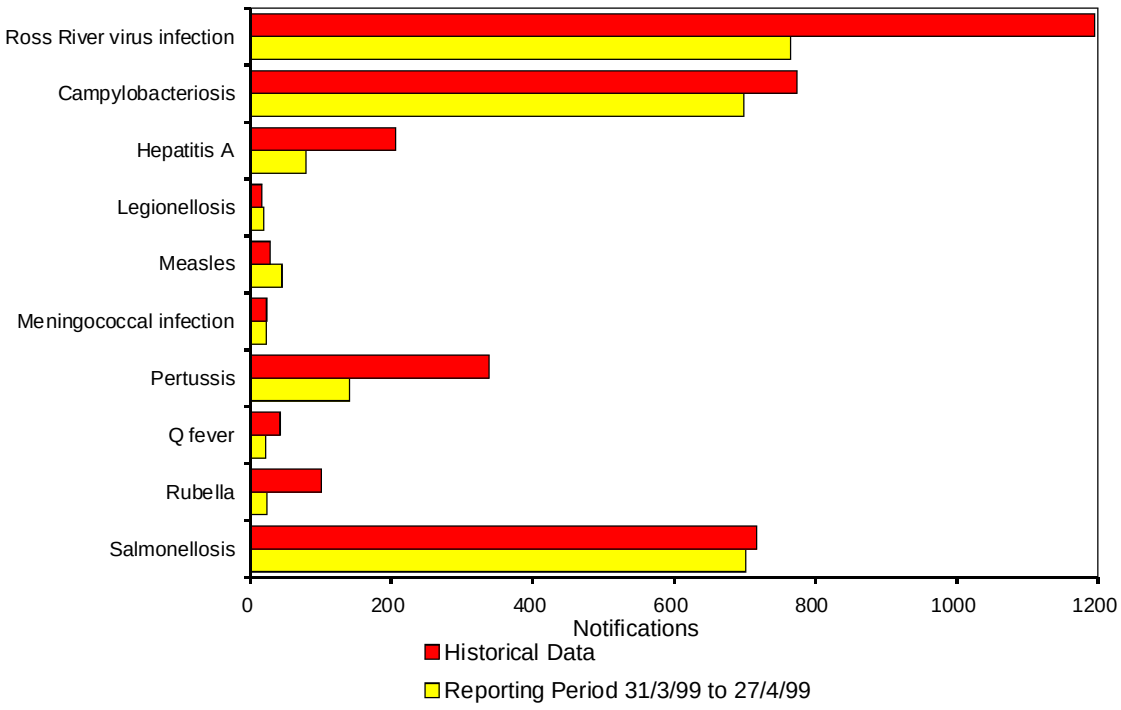
Tables

There were 5,560 notifications to the National Notifiable Diseases Surveillance System (NNDSS) in the four week period, 31 March to 27 April 1999 (Tables 1 and 2). The numbers of reports for selected diseases have been compared with historical data for corresponding periods in the previous three years (Figure 3).

There were 1,403 reports received by the *CDI* Virology and Serology Laboratory Reporting Scheme (LabVISE) in the four week period, 25 March to 21 April 1999 (Tables 3 and 4).

The Australian Sentinel Practice Research Network (ASPREN) data for weeks 13 to 16, ending 25 April 1999, are included in this issue of *CDI* (Table 5).

Figure 3. Selected National Notifiable Diseases Surveillance System reports, and historical data[1]



1. The historical data are the averages of the number of notifications in the corresponding 4 week periods of the last 3 years and the 2 week periods immediately preceding and following those.

Table 1. Notifications of diseases received by State and Territory health authorities in the period 31 March to 27 April 1999

Disease[1,2,3,4]	ACT	NSW	NT	Qld	SA	Tas	Vic	WA	This period 1999	This period 1998	Year to date 1999	Year to date 1998[5]
Arbovirus infection (NEC)	0	0	0	2	0	0	2	0	4	11	62	28
Barmah Forest virus infection	0	21	0	46	0	0	2	4	73	67	248	247
Brucellosis	0	0	0	3	0	0	0	0	3	2	8	16
Campylobacteriosis[6]	22	-	14	207	144	21	208	83	699	675	4,137	3,724
Chancroid	0	0	0	0	0	0	0	0	0	0	0	0
Chlamydial infection (NEC)[7]	2	NN	46	271	61	16	225	127	748	765	4,155	3,195
Cholera	0	0	0	0	0	0	0	0	0	0	1	2
Dengue	0	1	0	11	1	0	0	1	14	42	138	227
Donovanosis	0	NN	0	0	NN	0	0	0	0	2	5	19
Gonococcal infection[8]	1	60	60	66	17	0	58	67	329	400	1,757	1,594
Haemolytic uraemic syndrome[9]	NN	1	NN	0	0	0	NN	0	1	3	10	5
Hepatitis A	0	23	2	33	7	2	4	8	79	204	575	1,046
Hepatitis B incident	0	0	1	4	1	0	6	5	17	30	98	98
Hepatitis B unspecified[10]	7	140	0	69	0	3	63	10	292	453	1,886	2,250
Hepatitis C incident	3	2	0	-	2	0	1	3	11	16	95	80
Hepatitis C unspecified[5,10]	22	321	15	206	58	24	493	52	1,191	1,533	6,312	7,109
Hepatitis (NEC)[11]	0	0	0	0	0	0	0	NN	0	1	2	7
Hydatid infection	0	0	0	0	2	0	1	0	3	2	9	11
Legionellosis	1	3	0	1	1	0	9	4	19	18	111	73
Leprosy	0	0	0	0	0	0	0	0	0	0	0	1
Leptospirosis	0	3	0	47	0	0	1	0	51	8	149	46
Listeriosis	0	0	0	0	1	0	0	0	1	3	15	23
Malaria	1	9	1	22	0	1	9	2	45	30	268	192
Meningococcal infection	0	10	1	2	0	2	6	2	23	25	115	72
Ornithosis	0	NN	0	0	1	1	11	0	13	3	29	9
Q Fever	0	6	0	14	0	0	2	0	22	42	149	167
Ross River virus infection	1	135	3	517	4	20	27	58	765	438	2,696	1,542
Salmonellosis (NEC)	3	132	38	236	59	24	129	80	701	689	3,772	3,280
Shigellosis[6]	1	-	13	12	7	0	8	4	45	41	225	221
SLTEC, VTEC[12]	NN	0	NN	NN	1	0	NN	NN	1	1	11	5
Syphilis[13]	0	29	26	41	0	0	0	1	97	89	569	410
TTP[14]	0	0	0	0	0	0	0	0	0	0	0	0
Tuberculosis	0	26	1	5	1	4	35	3	75	86	450	406
Typhoid[15]	0	1	0	0	0	0	0	1	2	7	21	42
Yersiniosis (NEC)[6]	0	-	0	7	1	0	3	0	11	16	68	100

1. Diseases preventable by routine childhood immunisation are presented in Table 2.
2. For HIV and AIDS, see Tables 7 and 8.
3. 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.
4. No notifications have been received during 1999 for the following rare diseases: lymphogranuloma venereum, plague, rabies, yellow fever, or other viral haemorrhagic fevers.
5. Data from Victoria for 1998 are incomplete.
6. Not reported for NSW because it is only notifiable as 'foodborne disease' or 'gastroenteritis in an institution'.
7. WA: genital only.
8. NT, Qld, SA and Vic: includes gonococcal neonatal ophthalmia.
9. Nationally reportable from August 1998.
10. Unspecified numbers should be interpreted with some caution as the magnitude may be a reflection of the numbers of testings being carried out.
11. Includes hepatitis D and E.
12. Infections with *Shiga*-like toxin (verotoxin) producing *E. Coli* (SLTEC/VTEC) became nationally reportable in August 1998.
13. Includes congenital syphilis.
14. Thromotic thrombocytopaenic purpura became nationally reportable in August 1998.
15. NSW, Qld: includes paratyphoid.

NN Not Notifiable.

NEC Not Elsewhere Classified.

\- Elsewhere Classified.

Table 2. Notifications of diseases preventable by vaccines recommended by the NHMRC for routine childhood immunisation, received by State and Territory health authorities in the period 31 March to 27 April 1999

Disease[1,2]	ACT	NSW	NT	Qld	SA	Tas	Vic	WA	This period 1999	This period 1998	Year to date 1999	Year to date 1998
Diphtheria	0	0	0	0	0	0	0	0	0	0	0	0
H. influenzae type b infection	0	0	0	0	1	0	0	0	1	1	13	7
Measles	3	0	0	2	1	0	32	7	45	24	141	124
Mumps	0	1	0	3	0	0	7	3	14	14	42	62
Pertussis	6	43	0	17	8	1	64	2	141	350	1,112	3,014
Rubella[3]	3	1	0	7	2	2	7	2	24	44	109	223
Tetanus	0	0	0	0	0	0	0	0	0	0	0	2

NN. Not Notifiable

1. No notification of poliomyelitis has been received since 1978.
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. Includes congenital rubella.

Table 3. Virology and serology laboratory reports by State or Territory[1] for the reporting period 25 March to 21 April 1999, and total reports for the year

	State or Territory[1]								Total this period	Total reported in *CDI* in 1999
	ACT	NSW	NT	Qld	SA	Tas	Vic	WA		
Measles, mumps, rubella										
Measles virus					3		46	4	53	101
Mumps virus								2	2	18
Rubella virus		1			3		2		6	27
Hepatitis viruses										
Hepatitis A virus			7	8	10			13	38	149
Hepatitis D virus					1				1	2
Arboviruses										
Ross River virus		5	4	99	10		8	48	174	743
Barmah Forest virus		2	1	14			1	3	21	55
Dengue not typed								4	4	21
Murray Valley encephalitis virus								1	1	1
Kunjin virus								1	1	2
Flavivirus (unspecified)				2					2	13
Adenoviruses										
Adenovirus type 1					1		1		2	12
Adenovirus type 2							1		1	7
Adenovirus type 3					2		2		4	17
Adenovirus type 5					2				2	3
Adenovirus type 37							4		4	8
Adenovirus type 40								2	2	20
Adenovirus not typed/pending		3		3	58		4	12	80	441
Herpes viruses										
Cytomegalovirus				10	81		8	11	110	421
Varicella-zoster virus		2	2	10	44	1	21	15	95	615
Epstein-Barr virus		2	3	28	174		7	32	246	975

Table 3. Virology and serology laboratory reports by State or Territory[1] for the reporting period 25 March to 21 April 1999, and total reports for the year (continued)

	State or Territory[1]								Total this period	Total reported in *CDI* in 1999
	ACT	NSW	NT	Qld	SA	Tas	Vic	WA		
Other DNA viruses										
Papovavirus group							1		1	1
Parvovirus				2	4		12	4	22	136
Picorna virus family										
Echovirus type 2					1				1	2
Echovirus type 6		2			1				3	12
Echovirus type 7							1		1	1
Echovirus type 9		3							3	20
Echovirus type 11		5							5	30
Echovirus type 22		1							1	12
Echovirus type 30		1							1	19
Poliovirus type 2 (uncharacterised)					1				1	10
Rhinovirus (all types)					10		2	3	15	128
Enterovirus not typed/pending			3	1			3	30	37	264
Ortho/paramyxoviruses										
Influenza A virus		1		1	25		2	4	33	227
Influenza A virus H3N2							2		2	3
Influenza B virus					9			1	10	38
Parainfluenza virus type 1					7				7	16
Parainfluenza virus type 2		1			6			1	8	16
Parainfluenza virus type 3					23		2	5	30	278
Respiratory syncytial virus		1		12	25		1	21	60	333
Other RNA viruses										
HTLV-1								2	2	6
Rotavirus		1						24	25	311
Calici virus							1		1	1
Norwalk agent							1		1	30
Other										
Chlamydia trachomatis not typed		4	5	29	76	2		34	150	948
Chlamydia psittaci							10		10	30
Chlamydia species				2					2	5
Mycoplasma pneumoniae			3	9	24		33	1	70	452
Mycoplasma hominis		1							1	2
Coxiella burnetii (Q fever)				4			2	1	7	55
Bordetella pertussis		1		5			32		38	209
Legionella pneumophila					1				1	6
Legionella longbeachae					2			1	3	23
Cryptococcus species		2							2	3
TOTAL		39	28	239	604	3	210	280	1,403	7,278

1. State or Territory of postcode, if reported, otherwise State or Territory of reporting laboratory.

Table 4. Virology and serology laboratory reports by contributing laboratories for the reporting period 25 March to 21 April 1999

State or Territory	Laboratory	Reports
New South Wales	Institute of Clinical Pathology & Medical Research, Westmead	18
	Royal Prince Alfred Hospital, Camperdown	10
Queensland	Queensland Medical Laboratory, West End	243
	Townsville General Hospital	18
South Australia	Institute of Medical and Veterinary Science, Adelaide	604
Tasmania	Northern Tasmanian Pathology Service, Launceston	3
Victoria	Royal Children's Hospital, Melbourne	68
	Victorian Infectious Diseases Reference Laboratory, Fairfield	141
Western Australia	PathCentre Virology, Perth	209
	Princess Margaret Hospital, Perth	33
	Western Diagnostic Pathology	56
TOTAL		1,403

Table 5. Australian Sentinel Practice Research Network reports, weeks 13 to 16, 1999

Week number	13		14		15		16	
Week ending on	4 April 1999		11 April 1999		18 April 1999		25 April 1999	
Doctors reporting	50		49		51		49	
Total encounters	5411		5993		6817		6563	
Condition	Reports	Rate per 1,000 encounters	Reports	Rate per 1,000 encounters	Reports	Rate per 1,000 encounters	Reports	Rate per 1,000 encounters
Influenza	11	2.0	16	2.7	14	2.1	13	2.0
Rubella	0	0.0	0	0.0	0	0.0	0	0.0
Measles	0	0.0	1	0.2	0	0.0	0	0.0
Chickenpox	8	1.5	15	2.5	11	1.6	6	0.9
New diagnosis of asthma	8	1.5	17	2.8	8	1.2	5	0.8
Post operative wound sepsis	10	1.8	3	0.5	8	1.2	5	0.8
Gastroenteritis	30	5.5	62	10.3	68	10.0	46	7.0

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 1999;23:55.

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 every four weeks. 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 1999;23:58.

ASPREN currently comprises about 100 general practitioners from throughout the country. Up to 9,000 consultations are reported each week, with special attention to 12 conditions chosen for sentinel surveillance in 1999. CDI reports the consultation rates for seven of these. For further information, including case definitions, see CDI 1999;23:55-56.

Additional Reports

Serious Adverse Events Following Vaccination Surveillance Scheme

The Serious Adverse Events Following Vaccination Surveillance Scheme is a national surveillance scheme which monitors the serious adverse events that occur rarely following vaccination. More details of the scheme were published in CDI 1999:23;58.

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

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

Results for the reporting period 1 February 1999 to 30 April 1999.

There were 219 reports of serious adverse events following vaccination for this reporting period (Table 6). Onset dates were from 1997 to 1998, the majority (96%) being in 1998. Reports were received from Australian Capital Territory (19), Northern Territory (2), Queensland (99), South Australia (11), Tasmania (1), Victoria (15) and Western Australia (72). No reports were received from New South Wales for this period. The majority of reports received were from Queensland (45%).

The most frequently reported events following vaccination were persistent screaming (119 cases, 55%), followed by other reactions (44 cases, 20%), temperature of 40.5^{o} C or more (25 cases, 11.4%) and hypotonic/hyporesponsive episodes (18 cases, 8%).

Twenty-one of the 219 cases were hospitalised of which 20 had recovered at the time of reporting. There was incomplete information on recovery status on 17 cases while all the other cases had recovered at the time of reporting.

One hundred and ninety-six (89%) cases were associated with Diptheria-Tetanus-Pertussis (DTP), vaccine either alone or in combination with other vaccines. Of these 61% of reports were associated with the first dose of DTP and 32% with the second dose.

Table 6. Adverse events following vaccination reported in the period 1 February to 30 April`1999[1]

	Vaccines													
Event	DTP	DTP/Hib	DTP/OPV/ Hib	DTP/OPV/Other	DTP/OPV/MMR	DTP/OPV/Hib/ Hep B	DTP/MMR	MMR	Hib/OPV/Other	Hep B/Other	Hep B	Other [1]	Reporting States or Territories	Total reports for this period
Persistent screaming	74	3	40	1									ACT, Qld, Vic, WA	118
Hypotonic/ Hyporesponsive	8	1	8									1	Qld, SA, Vic, WA	18
Temperature	18		6								1		ACT,Qld, Vic, WA	25
Convulsions	3	1	1						1				ACT, Qld, SA, Vic, WA	6
Anaphylaxis		1					1	2	1					5
Other	19	2	8		1	1		2	2	1	2	6	ACT, NT, Qld, SA, Vic, WA	44
Total	122	8	63	1	1	1	1	4	4	1	3	7		216[2]

1. Includes influenza vaccination, DTPa, CDT, OPV, Hepatitis B vaccine, pneumococcal vaccination, BCG, ADT and rabies immunoglobulin (HRIG)
2. 3 cases have missing events

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.

abulations of diagnoses of HIV infection and AIDS are based on data available three months after the end of the reporting interval indicated, to allow for reporting delay and to incorporate newly available information. More detailed information on diagnoses of HIV infection and AIDS is published in the quarterly Australian HIV Surveillance Report, and annually in HIV/AIDS and related diseases in Australia Annual Surveillance Report. The reports are available from the National Centre in HIV Epidemiology and Clinical Research, 376 Victoria Street, Darlinghurst NSW 2010. Telephone: (02) 9332 4648; Facsimile: (02) 9332 1837; http://www.med.unsw.edu.au/nchecr.

HIV and AIDS diagnoses and deaths following AIDS reported for 1 to 31 December 1998, as reported to 31 March 1999, are included in this issue of CDI (Tables 7 and 8).

Table 7. New diagnoses of HIV infection, new diagnoses of AIDS and deaths following AIDS occurring in the period 1 to 31 December 1998, by sex and State or Territory of diagnosis

										Totals for Australia			
		ACT	NSW	NT	Qld	SA	Tas	Vic	WA	This period 1999	This period 1998	Year to date 1999	Year to date 1998
HIV diagnoses	Female	0	2	0	0	0	1	0	2	5	4	90	77
	Male	0	14	2	8	1	0	11	0	36	53	622	703
	Sex not reported	0	1	0	0	0	0	0	0	1	1	8	13
	Total[1]	0	17	2	8	1	1	11	2	42	58	622	794
AIDS diagnoses	Female	0	1	0	0	0	1	0	0	2	3	15	27
	Male	1	12	1	0	0	0	4	0	18	27	249	324
	Total[1]	1	14	1	0	0	1	4	0	21	30	265	351
AIDS deaths	Female	0	0	0	0	0	0	0	0	0	0	8	14
	Male	0	2	0	2	0	0	5	0	9	11	136	220
	Total[1]	0	2	0	2	0	0	5	0	9	11	144	235

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

Table 8. Cumulative diagnoses of HIV infection, AIDS and deaths following AIDS since the introduction of HIV antibody testing to 31 December 1998, by sex and State or Territory

		State or Territory								
		ACT	NSW	NT	Qld	SA	Tas	Vic	WA	Australia
HIV diagnoses	Female	22	580	8	131	57	5	199	102	1,104
	Male	186	10,499	104	1,863	648	77	3,749	869	17,995
	Sex not reported	0	259	0	0	0	0	24	0	283
	Total[1]	208	11,357	112	2,001	705	82	3,985	974	19,424
AIDS diagnoses	Female	8	169	0	45	20	3	67	25	337
	Male	85	4,514	33	784	326	43	1,583	343	7,711
	Total[1]	93	4,695	33	831	346	46	1,657	370	8,071
AIDS deaths	Female	2	113	0	30	15	2	47	16	225
	Male	63	3,115	24	547	224	28	1,235	244	5,480
	Total[1]	65	3,235	24	579	239	30	1,288	261	5,721

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

Bulletin Board

National Centre for Disease Control
Conference: *1999 Emergency Under Control*
1-2 June 1999
Rydges Canberra Hotel
Contact: Andrea Hodshon,
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Email: andrea.hodshon@health.gov.au

The Australian Society for Microbiology Inc.
The 11th International Conference
International Congress of Virology
9-13 August 1999
International Congress of Bacteriology and Applied Microbiology
9-13 August 1999
International Congress of Mycology
16-20 August 1999
Sydney, New South Wales
Fax: 03 9262 3135
Email: tourhosts@tourhosts.com.au

The International Leptospirosis Society
2nd International Scientific Conference
22-25 August 1999
Kooringa Lodge, Marysville, Victoria
Phone: 03 9905 4815
Fax: 03 9905 4811
Web page and conference registration:
http://www.med.monash.edu.au/micro/department/leptconf/ils99.htm

The Public Health Association of Australia Inc.
31st Annual Conference
26-29 September 1999
Carlton Hotel
Darwin, Northern Territory
Details: PO Box 319
Curtin ACT 2605
Email: conference@pha.org.au

Advance notice

Australian Society for Infectious Diseases Meeting
April 16-19, 2000
Fairmont Resort Leura organisers:
Dart Associates:
Phone: 02 94189396
For scientific content: Contact Tom Gottlieb,
Concord Hospital
Phone: 02-97677533
Fax; 02-97677868 or
E-mail: Tom@micr.crg.cs.nsw.gov.au

Royal North Shore Hospital
Conference: *Outpatient Parenteral Therapy - beyond 2000*
17-22 September 2000
Fairmont Resort
Leura, New South Wales
Phone: 02 9956 8333
Fax: 02 0056 5154
Email: confact@conferenceaction.com.au

The Australasian Society for HIV Medicine
12th Annual Conference
16-19 November 2000
The Carlton Crest, Melbourne, Victoria
Phone: 02 9382 1656
Fax: 02 9382 3699
Email: B.Pearlman@unsw.edu.au

The CDI Bulletin Board is provided as a service to readers. Every effort has been made to provide accurate information, but readers are advised to contact the relevant organisation for confirmation of details. Information about the availability of resources is included when space allows. Inclusion of a resource on the Bulletin Board does not imply endorsement of the resource by either the Communicable Diseases Network Australia New Zealand or the Commonwealth Department of Health and Aged Care.

Contributions to the Bulletin Board are invited from those organisations with forthcoming events relevant to communicable disease control.

Overseas briefs

Source: World Health Organization (WHO)
This material has been condensed from information on the WHO internet site. A link to this site can be found under 'Other Australian and international communicable diseases sites' on the CDI homepage.

Meningococcal disease

Sudan - Update

The outbreak of meningococcal meningitis that started in Northern Darfur State in early December 1998 has spread to 18 out of 26 states. From the beginning of the epidemic up to 22 April more than 16,000 cases, of which nearly 1,000 died, have been notified. The epidemic is expected to last until the end of June-July, depending on the geographical location of the states affected.

Since the start of the epidemic 7.6 million people have been vaccinated. The vaccination campaigns, as well as other epidemic control measures are being implemented through a national task force chaired by the Federal Ministry of Health. This task force includes the members of the International Coordinating Group on Vaccine Provision for Epidemic Meningitis Control (ICG), namely the IFRC, MSF, UNICEF and WHO.

Current work includes strengthening of the surveillance system and training for health care and laboratory workers in order to improve the detection and management of cases. Health education messages have also been widely distributed.

The task force estimated that a further 4.5 million people need to be vaccinated during the next three weeks and stressed the need for availability of medicines for treatment of the patients at peripheral health centres. The ICG is requesting an additional US$ 2.1 million from the international community with a view to meeting these needs immediately.

Polio

Angola

On 23 March, the paediatric hospital in Luanda reported that a total of 21 cases of acute flaccid paralaysis with 3 deaths had been registered. An investigation by the Ministry of Health demonstrated that by 3 April, 102 cases of AFP had been recorded in Luanda and neighbouring areas of Bengo province. Cases were primarily in children aged < 5 years and 90% of cases had received 2 doses or less of oral polio vaccine. Only 6% had received 4 doses.

On 8 April, the National Institute of Virology in South Africa reported that wild poliovirus type 3 had been isolated from 11 of 22 stool specimens taken from AFP cases in Angola. By 25 April, the number of polio cases was reported to be 661 with 41 deaths. Field investigation confirmed 6 cases of AFP in children aged < 5 years in Benguela, a city 500 km south of Luanda. Investigation of the outbreak is ongoing with the assistance of a WHO team. In response to the outbreak, 634 000 children were immunized with OPV in Luanda on 17 and 18 April. A national immunization campaign is being planned to start in June.

Cholera

Somalia - Update

A total of 7860 cases with 233 deaths has been reported since the beginning of the current epidemic which started in December 1998. Areas where cases were still being registered at the end of March and first half of April are Mogadishu, Kismayo, and Baidoa. The disease is still appearing in new areas - Bay, Lower Shabelle, Upper and Lower Juba - which are difficult to access and figures are not available.

WHO is continuing to make supplies available and supporting the health authorities in strengthening cholera preparedness plans since more outbreaks are expected later in the year following the seasonal pattern

Acute haemorrhagic fever syndrome

Democratic Republic of Congo

An outbreak of suspected viral haemorrhagic fever has been reported in Durba, Watsa Zone, in the northeastern Democratic Republic of Congo (DRC). Clinical features include fever, headache, lassitude, gastrointestinal bleeding, coughing up blood and agitiation. The first cases are believed to have occurred in January 1999. Between January and 28 April, 50 cases, with 46 deaths have been recorded (CFR=92%). The earliest cases appear to have occurred in gold miners, but now cases are occurring among those living in the community. The WHO Office in DRC and the WHO Regional Office for Africa in Harare, Zimbabwe and MSF(Belgium and Holland) are preparing a team to investigate. The security situation in the area is poor and access to the affected area may be extremely difficult.

Sudan

The outbreak of cholera which began in early March is continuing. The areas of Padak, Mading, Wanding, Lankien, Akobo and Burmat have reported a total of 892 cases with 24 death up to 27 April 1999.

These figures are cases admitted to hospital and are provisional. The epidemic mainly affects the Jonglei region in areas south of the river Sobat. As it is the beginning of the rainy season people have started moving with their animals from locations along the river to inland sites where other areas are likely to be affected.

A cholera response team coordinated by UNICEF is meeting twice weekly to review the situation, share information and plan the response strategy. UNICEF currently has ORS and tetracycline on standby for use as the need arises. WHO has sent an epidemiologist to assist local health authorities to assess the situation in the affected areas.

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Contributions

Contributions covering any aspects of communicable diseases are invited. All contributions are subject to the normal refereering process. Instructions to authors can be found in *CDI* 1999;23:59.