The epidemiology of rubella and congenital rubella in Australia, 1992 to 1997

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Abstract

Selective rubella vaccination of schoolgirls commenced in 1971 and was followed by a significant reduction in congenital rubella. Infant vaccination with MMR was introduced in 1989 to interrupt circulation of the virus in young children, and in 1994/95 the adolescent school based rubella vaccination program was changed to MMR for both boys and girls. This report reviews the epidemiology of rubella and congenital rubella between 1992 and 1997 using reports to the National Notifiable Diseases Surveillance System (NNDSS) and the Australian Paediatric Surveillance Unit (APSU). Notification rates for rubella exceeded 20 per 100,000 in 1992, 1993 and 1995 and declined to 7.2 per 100,000 in 1997. Sixty-one per cent of notifications occurred between September and December and 68% occurred in males. The incidence rate in males aged 15–22 years peaked at 152.6 per 100,000 in 1995 reflecting the lack of immunisation in this cohort. From 1993 to 1997, 19 children were reported with congenital rubella syndrome, representing 1 in 67,000 live births. Of these, 17 had multiple defects (4 died) and 2 had deafness only. There were also 5 infants with congenital rubella infection but no defects. Australia's rate of congenital rubella syndrome exceeded that of the United Kingdom and the United States of America but this may be partly attributable to differences in reporting practices. The impact of changing the second dose of MMR vaccine to 4 years of age in 1998 will require careful monitoring. *Commun Dis Intell* 1999;23:209-214.

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Introduction

In 1971, Australia commenced selective vaccination of 10–14 year old girls to prevent congenital rubella. By the mid-1980s this had significantly reduced the occurrence of congenital rubella from approximately 120 to fewer than 20 cases per year.[1,2] However, to eliminate rubella in pregnancy, the United States (US) strategy of interrupting the circulation of virus through vaccinating young children (the primary transmission group), was required.[3] A combined infant and adolescent strategy was adopted in Australia in 1989 when measles-mumps-rubella vaccine (MMR1) was introduced for infants at 12 months of age. Further revision was made to the immunisation schedule in 1994/95 when selective rubella immunisation of schoolgirls ceased and a second dose of MMR (MMR2) was recommended for both boys and girls aged 10–16 years.

Rubella vaccination policy has changed once more. In response to the longer-term plan to eliminate measles, the Australian Technical Advisory Group on Immunisation recommended that, as part of the 1998 National Measles Control Campaign, the existing MMR school based program cease and the second dose of MMR be given at 4 years of age to coincide with school entry diphtheria-tetanus-pertussis (DTP) and oral polio (OPV) vaccinations. Changing the schedule will lengthen the time between MMR2 vaccination and pregnancy. The impact of this change on population susceptibility to rubella infection and on immunity during pregnancy will require careful monitoring.

This report describes the epidemiology of rubella and congenital rubella cases notified to the National Notifiable Diseases Surveillance System (NNDSS) and the Australian Paediatric Surveillance Unit (APSU) between 1992 and 1997.

Methods

Data sources

Under the Public Health legislation of each State and Territory, medical practitioners (except in New South Wales; NSW) and laboratories (except in Western Australia; WA) are required to notify rubella; congenital rubella is mandatorily notifiable only in 5 of the 8 States and Territories. Since 1991 these data have been collated nationally by the NNDSS. This report includes NNDSS rubella data with a stated onset of disease between 1 January 1992 and 31 December 1997. Australian Bureau of Statistics mid-year population estimates for each year (1992–1997) were used to calculate age-specific and crude notification rates. Seasonal trends were reported by month of disease onset.

Congenital rubella data were derived from the APSU, which has maintained a national system of active surveillance since 1993. Analysis of congenital rubella cases relate to children born in Australia between January 1993 and December 1997.

Surveillance case definitions

Rubella

Rubella is defined by the National Health and Medical Research Council (NHMRC) (1994) as:

(a) a generalised maculopapular rash and a fever, and one or more of: arthralgia, arthritis or lymphadenopathy or conjunctivitis and an epidemiological link to a confirmed case; or

(b) demonstration of rubella-specific IgM antibody, except following immunisation; or

(c) a fourfold or greater change in rubella antibody titre between acute and convalescent phase sera obtained at least 2 weeks apart; or

(d) isolation of the virus from a clinical specimen. [4]

Congenital rubella infection

A case of congenital rubella infection is defined as an infant with no rubella defects, but with congenital infection confirmed by isolation of virus, or detection of IgM or persistent IgG rubella antibody in the infant.

Congenital rubella syndrome

A case of congenital rubella syndrome is defined by the NHMRC (1994) as a live or stillborn infant with clinically compatible defects and at least one of the following:

(a) isolation of rubella virus from a clinical specimen from the infant; or

(b) demonstration of rubella-specific IgM antibody in the infant's serum; or

(c) persistence of rubella-specific IgG antibody titre higher than expected from passive transfer of maternal antibody; or

(d) laboratory confirmed maternal rubella in the first trimester of pregnancy. [4]

Results

Rubella, 1992-1997

Between January 1992 and December 1997, a total of 19,599 cases of rubella were reported to the NNDSS. Notifications remained elevated between 1992 and 1995, with rates of infection ranging between 18.9 to 25.4 per 100,000 population. The incidence of notified rubella subsequently declined to 7.2 per 100,000 population in 1997. Both rates of infection and the time at which disease activity peaked varied by State (Table 1). Overall, Queensland reported 35.4% of notifications, Victoria 27.6% and NSW 14.6%. Western Australian data were absent for 1992, while Tasmania reported no cases of rubella until 1995. Seasonal fluctuation was evident, with 61% of all notifications occurring between the months of September and December (Figure 1).

On average, males accounted for 68% of rubella notifications reported annually, with individuals aged 15–22 years most frequently affected. Age-specific notification rates for this cohort (15–22 years) ranged from 113.5 per 100,000 population in 1992 rising to a peak of 152.6 cases per 100,000 population in 1995 and falling to 35.7 per 100,000 in 1997. Figures 2,3 and 4 show the age and sex distribution for those years. Table 2 gives the overall rates for all years 1992 to 1997.

The incidence of notified disease in females of an equivalent age (15–22 years) was considerably lower (Table 2). In 1992, the notification rate was 16.7 per 100,000 population (Figure 2) increasing to 22–23 per 100,000 between 1993 and 1995 (Figure 3)before declining to 9.2 per 100,000 in 1997 (Figure 4). In 1992 and 1995 when notified disease activity was at its highest, the male:female ratio was 7:1, falling to 4:1 in 1997.

Table 1. National rubella notifications and notification rates by State, 1992–1997

State	Notifications						Rate per 100,000 population					
	1992	1993	1994	1995	1996	1997	1992	1993	1994	1995	1996	1997
ACT	609	127	49	173	70	32	206.6	42.4	16.2	56.7	22.7	10.3
NSW	339	812	113	1,213	254	142	5.7	13.5	1.9	19.8	9.7	2.3
NT	NN	(18)*	(45)*	10	7	7	-	-	-	5.6	3.8	3.7
Qld	788	1,404	2,148	1,073	979	539	7.5	45.1	67.4	32.9	29.3	15.8
SA	121	275	75	87	382	184	8.3	18.8	5.1	5.9	25.9	12.4
Tas	NN	NN	NN	169	32	18	-	-	-	35.6	6.7	3.8
Vic	2,230	491	211	1,468	667	337	50.0	10.9	4.7	32.5	14.6	7.3
WA	1	509	730	396	161	84	0.06	30.3	42.9	22.8	9.1	4.5
Aust	4,088	3,636	3,371	4,589	2,552	1,343	23.4	20.6	18.9	25.4	13.9	7.2

NN Not Notifiable.

* Rubella was not notifiable in NT until December 1994.

Figure 1. Notifications of rubella, Australia, January 1992 to December 1997, by month of onset

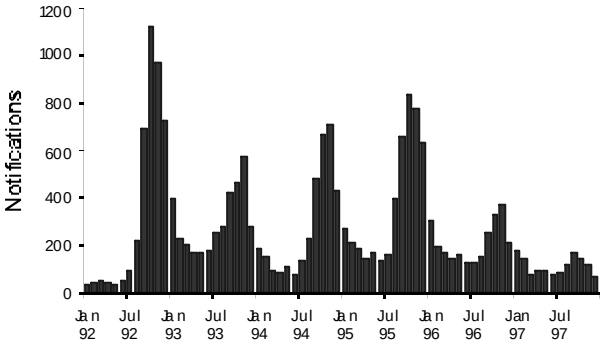

Figure 2. Notification rates per 100,000 population for rubella, Australia, 1992, by age and sex

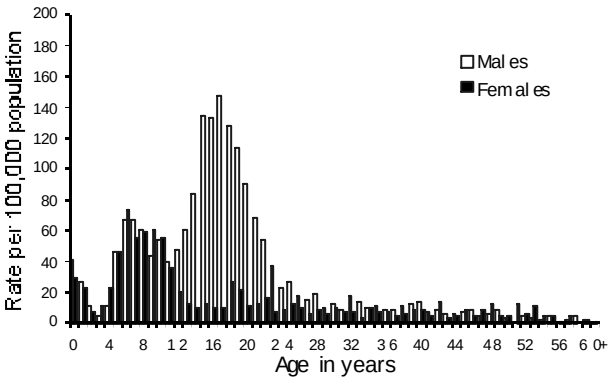

Figure 3. Notification rates per 100,000 population for rubella, Australia, 1995, by age and sex

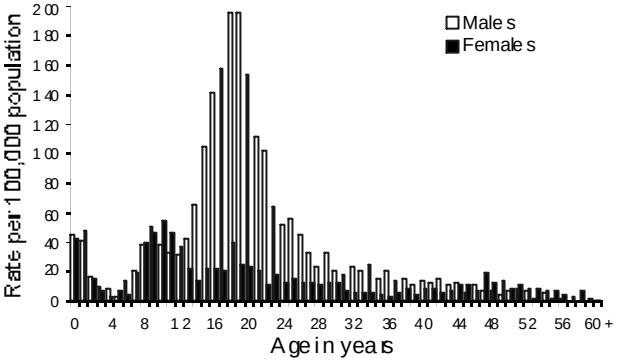

Figure 4. Notification rates per 100,000 population for rubella, Australia, 1997, by age and sex

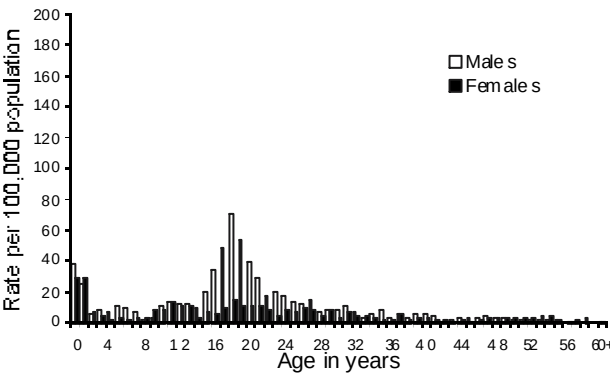

Table 2. National rubella notification rates per 100,000 population in Australia, 1992–1997, by age group and sex

	2–14 years		15–22 years		All ages	
Year	Male	Female	Male	Female	Male	Female
1992	48.7	39.4	113.5	16.7	31.5	14.3
1993	29.6	27.9	98.2	21.7	26.5	14.3
1994	22.2	20.4	99.9	21.7	25.0	12.1
1995	31.6	26.7	152.6	23.2	36.2	13.6
1996	13.3	14.0	81.3	16.1	18.8	8.9
1997	8.2	5.9	35.7	9.2	9.6	4.9

Table 3. Congenital rubella births in Australia per year, 1993–1997

Year of birth	Live births*	Congenital rubella syndrome	Birth incidence per 10,000 live births
1993	258,626	4	0.15
1994	258,426	5	0.19
1995	254,942	4	0.15
1996	251,000	5	0.20
1997	253,673	1	0.04

* Australian Bureau of Statistics

For women of childbearing age (15–44 years) the incidence of rubella notifications remained around 12 cases per 100,000 population between 1992 and 1993 (Figure 2), declining to 5.6 per 100,000 in 1997 (Figure 4). As a proportion of total notifications (2,660 /20,286), the rate in this age group remained fairly constant at around 14% of annual notifications.

For children aged 2 to 14 years the incidence of disease fell progressively from 42 per 100,000 population in 1992 to 7 per 100,000 in 1997.

The decline in the childhood incidence of notified disease is more apparent when the proportion of cases reported in children aged 2–14 years is compared over time with other selected age groups. The proportion of notified disease in males aged 2–14 years and 15–22 years has changed as notification of infection in younger males declined from 30% in 1992 to 16% in 1997. Over the same period, the proportion of notified cases reported in the 15–22 year old cohort remained at around 47%. The trend in the female population has also changed, with the proportion of reported cases in 2–14 year olds falling by 29% to 21% by 1997, equating to that of females aged 15–22 years.

Congenital rubella syndrome, 1993–1997

From 1993 to 1997, 24 children born in Australia with congenital rubella were reported to the APSU; 19 had congenital rubella syndrome and 5 were congenitally infected but had no defects; only 1 of these 5 children was exposed in the first 16 weeks of gestation. In 1997 only 1 case of congenital rubella syndrome was reported to the APSU. This figure may be an underestimate of the true incidence since not all cases may yet (at 30 July 1999) have been identified although all cases but one were reported to the APSU within the first year of life. Incidence at birth is shown in Table 3. Over the same period only 8 cases of congenital rubella were notified to the NNDSS, all from NSW.

APSU records indicate that over this 5 year period 13 (54%) children with congenital rubella with or without defects were born in NSW, with the remainder born in Queensland 6 (25%), the Australian Capital Territory (ACT) 2 (8%) and Victoria 3 (13%). No cases were reported in the NT, South Australia (SA) or WA during this period. The ratio between congenital rubella births and reports of rubella in women of childbearing age varied markedly between States reporting cases (Table 4).

Multiple defects were reported in 17 of 19 (90%) defective children (4 died, 1 from sudden infant death syndrome), while deafness, reported as a single defect, was diagnosed in 2 (10%) children (Table 5). A further 5 children demonstrated rubella specific IgM positivity neonatally but showed no evidence of defects.

Fourteen of the 24 mothers (58%) whose infants had congenital rubella infection were born in Australia; 8 mothers were born elsewhere, and the place of birth was not recorded for two. Maternal age ranged from 16 to 42 years. Six women gave a history of rubella vaccination. During the first trimester of pregnancy 15 women reported either being aware of a rash or rubella-like illness, having come in contact with a person with rubella or having serological confirmation of rubella. Sixteen infants were the outcome of a first pregnancy, 4 of a second, 3 of a third, and 1 of a fourth pregnancy.

Discussion

Between 1992 and 1997 the striking features of the epidemiology of rubella in Australia were the seasonality (peaking in spring), the low reported incidence in 1997, and the high notification rates in adolescent males compared with adolescent females. This difference between adolescent males and females appears to reflect the fact that males were not included in the adolescent vaccination program until 1994/95. It is likely that outbreaks will continue to occur in males aged around

Table 4. Ratio of congenital rubella cases to the number of rubella cases reported in women aged 15–44 years, by State, Australia, 1993–1997

State	Rubella notifications*	Rubella notificati ons in women 15–44 years. Number (% of total)		Congenitally infected infants. Number (number without defects)	Ratio of congenital rubella infection to reported infection in women 15–44 years
ACT	1,057	94	(8.9%)	2	1:47
NSW	2,873	376	(13.1%)	13 (4)	1:29
Qld	6,931	1,553	(22.4%)	6 (1)	1:259
Vic	5,404	327	(6.0%)	3	1:109

* Notification of rubella cases to NNDSS

17–24 years until younger immunised males reach this age or until immunisation coverage of younger children is sufficiently high to interrupt rubella transmission.

The occurrence of at least 2,660 cases of rubella in women of childbearing age resulted in the birth of 24 infants with congenital rubella (19 with defects). APSU data indicated that missed opportunities to immunise and failure to confirm apparent infection in pregnancy were major contributing factors to this outcome.[5] The fact that 8 of the mothers had been born overseas (7 in countries currently without rubella vaccination programs) was also important.

In comparison, from 1993 to 1996 (4 years) the British Paediatric Surveillance Unit and the National Congenital Rubella Surveillance Program in the United Kingdom (UK), which uses a definition consistent with that of the APSU, reported the birth of 20 infants with congenital rubella syndrome.[6] The mothers of 4 of these 30 cases acquired their infection abroad. The increase in cases of congenital rubella in 1996 in the UK followed a springtime outbreak among young men who had not been vaccinated.[7] The birth cohort in the UK is more than double that of Australia. Much lower congenital rubella syndrome rates were reported in the US (7 indigenous cases in the 3 years 1993 to 1996)[8] although previous rates were higher.[9] However, it is estimated that because a passive surveillance system is used, only 40%–70% of all US cases are detected.[8] Notwithstanding this, a much more restrictive case definition of congenital rubella syndrome is applied than that used in Australia or the UK and only 13 of the 19 Australian cases would fit the US definition.[10]

Of the 6,931 reports of rubella in Queensland, close to 1 in 4 cases occurred in women of childbearing age. In Victoria, which reported a total of 5,404 cases, the ratio was closer to 1:16, while in NSW, which reported 2,873 cases, the ratio was 1:7. The apparent disparity across States may be a consequence of differential reporting of rubella, variable immunisation coverage of women of childbearing age or use of laboratory diagnosis in women of this age. This may also account for the marked variation between States and Territories in the ratio of rubella reported in women of childbearing age and the occurrence of congenital rubella. In NSW, one congenital rubella birth occurred for every 29 women reported infected. All congenital rubella cases were reported from the more populous Eastern States and none from the NT, SA, Tasmania or WA.

Table 5. Clinical manifestations and deaths recorded in 19 cases of congenital rubella syndrome, 1993–1997*

Manifestation	Number of children	
Deaths		4
Deafness		11
Eye defects		10
cataract(s)	8	
retinopathy	2	
keratitis	1	
Central nervous system defects		8
microcephaly	4	
intracerebral calcification	6	
haemorrhage	1	
cerebral palsy	1	
Cardiovascular defects		11
patent ductus arteriosus	9	
pulmonary artery stenosis	2	
other	4	
Pneumonitis		2
Intrauterine growth retardation/ failure to thrive		9
Developmental delay		4
Other		12

*Not all infants had been fully assessed at the time of notification, so this may be an underestimation of the actual defects particularly deafness. Seventeen infants had more than one defect.

The absence of standardised surveillance methods across State and Territory health authorities may in part explain these disparities. At present neither the method of diagnosis (clinical, cultural or serological) nor the mechanism of notification (whether by doctor, hospital or laboratory) is specified on the database. Since an element

of bias is associated with each, and variation exists in the reporting requirements of each State, interpretation of and comparison between State and Territory data at a national level is difficult.

Pregnancy status at the time of infection and rubella-associated terminations of pregnancy are not monitored nationally, so the actual number of adverse outcomes associated with rubella infection in pregnancy is unknown and may be higher than current data suggest. Three first trimester terminations for rubella are known to have occurred in WA between 1993 and 1994.[1] In SA, it was reported that between two and four rubella-associated terminations were carried out each year,[11] but the last one was carried out in 1993.[2] In the UK, where rubella-associated pregnancies are notifiable, births of babies with congenital rubella and terminations associated with rubella disease or contact have both fallen between 1971 and 1996.[7]

Rubella surveillance is complicated by the nature of the disease itself, because up to 50% of infections occur without a recognised rash, and clinical diagnosis is unreliable.[12] While laboratory confirmation is recommended practice, reluctance on the part of many doctors to take blood from children means that an excess of laboratory confirmed cases in adults is likely to occur.[13] Conversely, it has been found that misdiagnoses in children may both distort age-specific incidence and increase background incidence between epidemic years, obscuring the true size of resurgence in disease activity when it does occur.[14]

In recognition of these limitations, efforts are in progress to develop uniform standards of communicable disease surveillance in Australia at the national level.[15] More specifically, in the light of the 1998 National Measles Control Campaign and the subsequent change to the childhood immunisation schedule, enhanced serosurveillance of measles and rubella susceptibility is being initiated by the National Centre for Immunisation Research and Surveillance of Vaccine Preventable Diseases (NCIRS). Modelled on the British system,[14] the NCIRS will monitor the effect of revised disease control strategies on rubella susceptibility in children and women of childbearing age through a national system of age-stratified serosurveillance.[16] Information derived from this source will also be used to construct mathematical models of rubella epidemiology for the purpose of predicting outbreaks. Collation of this information with immunisation coverage data derived from the Australian Childhood Immunisation Register, and from other surveillance systems such as NNDSS, APSU and LabVISE (the national sentinel Virology and Serology Reporting Scheme) will in the future provide a more accurate estimate of the effectiveness of rubella prevention and control strategies.

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An outbreak of *Campylobacter* enteritis on an island resort, north Queensland

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Abstract

An outbreak of *Campylobacter* enteritis among staff on a resort island in north Queensland is reported. Untreated rainwater and food from the staff dining room were initially suspected as possible sources of infection but *Campylobacter* species were not isolated from any environmental samples. Faecal contamination was detected in four rainwater tanks. A case control study involved a total of 23 cases (7 confirmed and 16 probable), 3 of whom required hospitalisation. There was a strong association between gastrointestinal illness and consumption of water from a dispenser in the staff restaurant that had probably been filled from one of the contaminated tanks. We conclude that this was probably a waterborne outbreak and postulate that *Campylobacter* species were introduced into one or more of the tanks by contamination with the faeces of wild animals. *Commun Dis Intell* 1999;23:215-219.

Introduction

Campylobacter is a common cause of gastrointestinal illness in Australia[1] and overseas,[2,3] and while the majority of cases are sporadic in nature, outbreaks are occasionally detected. This report describes an outbreak of *Campylobacter* enteritis among staff on an island resort in north Queensland for which untreated rainwater was the likely source of infection. Waterborne outbreaks of *Campylobacter* enteritis are well documented[2-10] but, to the best of the authors' knowledge, have not previously been reported in Australia.

The setting for this outbreak was a large island off the north Queensland coast. The only population centre on the island was a tourist resort that had over 600 staff and 900 guests resident at the time. Food was available to guests from 14 restaurants and four cafes, and there was one staff restaurant. A reticulated water supply distributed a mixture of chlorinated dam water and the output from a reverse-osmosis plant, and staff also had access to a number of untreated rainwater tanks. Staff were accommodated in a variety of widely separated quarters, some of which had food preparation facilities. Domestic animals were banned on the island but birds, bats and other native animals were abundant.

The Tropical Public Health Unit (TPHU) was notified on 17 June 1997 when *Campylobacter* was isolated from the faeces of a resort employee admitted to the district hospital. Preliminary investigations identified a further two staff members who had been admitted with abdominal pain and diarrhoea in the preceding week, both of whom subsequently had *Campylobacter* isolated. The resort's medical practitioner identified over 20 additional staff members who had presented with gastrointestinal illness during the same period. No visitors with gastrointestinal illness were seen. Further environmental, microbiological and epidemiological investigations were then initiated by TPHU staff to define the circumstances of the outbreak.

Investigations

Environmental investigations

Environmental Health Officers (EHOs) arrived at the resort on 19 June. The staff restaurant and all guest restaurants were inspected and, in the staff restaurant, swabs from bench tops and samples of pre-cooked diced chicken and chicken leg were collected.[11] These were forwarded to the Centre for Public Health Sciences, Brisbane (CPHS) for *Campylobacter* isolation. Samples were analysed using Prestons *Campylobacter* broth and subsequent subculture onto Prestons Agar.[12] There were no food samples remaining from the meals served prior to the outbreak.

Seven functioning rainwater tanks were identified (designated Tanks 1 to 7). All tanks drained directly from adjacent rooftops and none were routinely treated. Water samples were collected from as many as possible between 19 and 22 June. Samples for routine microbiological analysis were collected from Tanks 2, 3 and 4 and from the reticulated supply. A separate collection of up to 1,000 mL was obtained from Tanks 2, 3, 4, 5 and 6 for *Campylobacter* isolation. Samples were collected directly from taps into sterile containers and were transported on ice to CPHS to arrive within 24 hours of collection. Taps were not flamed prior to sample collection. Where there was sufficient volume, *Campylobacter* isolation was attempted using two different methods:

(i) filtration through a 45 micron filter followed by suspension of the filter in Prestons *Campylobacter* broth; and

(ii) centrifugation of the water to produce a concentrated sample which was then placed on a 65 micron filter overlaid on a blood agar plate.

The plate was incubated at 37^{0}C for 20 minutes then the filter paper was removed. The blood agar plate was then incubated in a special atmosphere (80% nitrogen, 10% hydrogen, 10% carbon dioxide) at 37^{0}C. This second method was designed to detect non-thermophylic *Campylobacter* species.

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Tank 1 had been emptied on 19 June, prior to the arrival of the EHOs, but they were able to obtain a sample of sludge from the bottom of the tank, which was submitted to CPHS for *Campylobacter* isolation. In addition, the Water Treatment Officer on the island had collected water from the tank immediately prior to the tank being emptied. It was subjected to routine microbiological analysis at a private laboratory used by the resort, and a portion was subsequently forwarded to CPHS for *Campylobacter* isolation.

Staff also had access to drinking water from dispensers at several locations. These were inverted clear plastic containers with a capacity of approximately 20 litres that were available commercially but were capable of being refilled from other sources. One such dispenser was available in the staff restaurant. Water samples were not collected from these dispensers because they were not suspected as potential sources of infection during the initial investigation.

Microbiological investigations

The EHOs requested faecal specimens from all staff with a recent history of gastrointestinal illness. Eleven samples were obtained and transported on ice to CPHS for *Campylobacter* culture. Isolates of *Campylobacter jejuni* were then further analysed. Penner sero-groups were determined using specific *Campylobacter jejuni* antisera (Mast Diagnostics) by extracting the heat-stable lipopolysaccharide O antigens from the isolates and reacting these against sensitised chicken red blood cells. In addition, the genetic relatedness of isolates was compared with Pulsed Field Gel Electrophoresis (PFGE), using Sma1 as a cutting enzyme.[13]

Epidemiological investigations

A case control study was initiated on 7 July as initial testing did not identify a source of infection. Potential cases were identified using the list compiled by the EHOs on their initial visit, from other staff being questioned, and from a complete review of records maintained by the medical practitioner on the island. Resort management had written to all staff at the time of the outbreak requesting that those with gastrointestinal symptoms immediately attend the doctor. People who developed their illness after a household member were considered to be secondary cases and were therefore excluded. In addition, because Tank 1 had been emptied on 19 June, those who became unwell after 24 June were also excluded.

Confirmed cases were defined as staff members with onset of a compatible clinical illness between mid-May and 24 June who had *Campylobacter jejuni* isolated from their faeces. Probable cases were staff members who became unwell between mid-May and 24 June with an illness consisting of either diarrhoea for two or more days or at least four of the following; diarrhoea for one day, nausea, vomiting, stomach pain, fever, headache, myalgia or malaise. Diarrhoea was defined as two or more loose watery stools per day. Controls were adult staff members who had been at the resort during the outbreak but did not meet the criteria for cases and had not shared a room with a case. They were nominated by cases or approached directly at their place of residence to ensure that the different staff accommodation areas were equally represented among cases and controls.

The following details were collected: name, date of birth, sex, accommodation, occupation, symptoms, treatment, and time off work. Potential exposures were sought from cases for the week prior to their illness and from controls for the week prior to the onset of illness in a case from the same accommodation area. Potential exposures sought were: source of water for drinking, dining at the staff restaurant, other sources of food, contact with animals and use of swimming pools. All who had eaten at the staff restaurant were asked about specific foods consumed there. Detailed menus for this period were unavailable, however the restaurant served a limited number of dishes on a weekly cycle and it was possible to establish a generic list of dishes served. This list was used to aid recall.

Cases and controls were not individually matched but were grouped by accommodation area and unmatched analyses

Table 1. Bacterial isolates from analyses of water samples, by water source

Water source	*Campylobacter* isolation	Routine microbiological analysis	
		Coliform count (colonies per 100mL	*E. coli* count (colonies per 100ml)
Tank 1	Not isolated*	500#	55#
Tank 2	Not isolated	51	13
Tank 3	Not isolated	Positive♦	Positive ♦
Tank 4	Not isolated	Positive♦	27
Tank 5	Not isolated	Not tested	Not tested
Tank 6	Not isolated	Not tested	Not tested
Tank 7	Not tested	Not tested	Not tested
Reticulated supply	Not tested	Not detected	Not detected

*Two samples tested: a water sample forwarded from a private laboratory and sludge from the bottom of the tank

result from private laboratory (all other results were from the CPHS)

♦ Detected but no count was possible due to the presence of confluent growth.

were performed using Epi Info.[14] Matched analyses were also performed.

Results

Environmental results

Significant food hygiene problems were identified in the staff dining room kitchen. Food residues had collected on several bench surfaces as a result of inadequate cleaning and few staff understood the correct storage temperatures for food or the dangers of cross-contamination. There was also no quality assurance program in place to monitor the safe handling and preparation of food.

Microbiological results

Campylobacter species were not isolated from the bench swabs or the food samples from the staff restaurant, nor from any of the water samples. However, Tanks 1 to 4 all showed evidence of faecal contamination, with particularly high coliform counts noted in Tank 1 (Table 1). There was no evidence of faecal contamination of the reticulated water supply.

Campylobacter jejuni was isolated from seven cases and all isolates were subjected to further testing. Two genetically distinct 'pulsovars' were identified by PFGE. Within each pulsovar isolates were genetically indistinguishable. All 5 isolates in one pulsovar were Penner sero-group O(19), and of the 2 in the other pulsovar, 1 was Penner sero-group G(8) and the other was un-groupable. There were therefore at least 2 distinct strains of *Campylobacter jejuni* involved in this outbreak.

Epidemiological results

The case control study identified 23 people who met the case definition (7 confirmed and 16 probable cases), all of whom were resort staff. Twenty-four controls were interviewed.

Most cases had their onset in the period 7 to 17 June, but there were a few cases in late May (Figure 1). The most common symptoms were nausea (100%), diarrhoea (96%), abdominal pain (96%) and fever (87%). Other symptoms included headache (61%), vomiting (39%), joint pain (22%) and blood in faeces (17%). Symptoms lasted from 1 to 28 days, with a median duration of 4 days.

Nearly three-quarters (74%) of cases required time off work, ranging from 1 to 28 days (median of 2 days and an overall total of 93 days). Three cases were admitted to the district hospital, one of whom had an appendicectomy. There were no deaths.

Cases and controls had similar age characteristics. The median age of cases was 25.5 years (range 19.4-60.4), and for controls was 25.3 years (range 17.9-57.7). No

Figure 1. Cases of illness included in the epidemiological investigation, by date of onset and confirmation of *Campylobacter* diagnoses

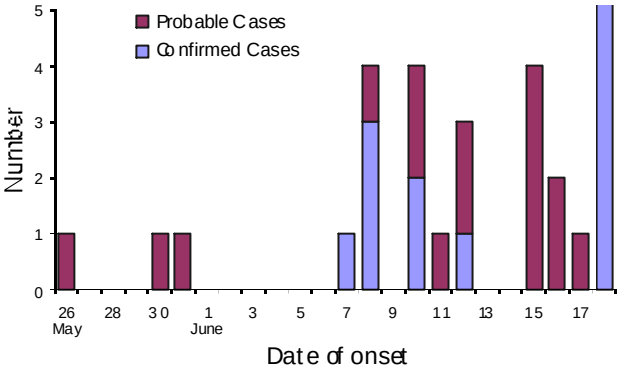

Table 2. Number of cases and controls, by exposure to potential sources of infection, contains matched and unmatched analyses

Exposure	Confirmed cases (n = 7)	Probable cases (n = 16)	Total cases (n = 23)	Controls (n = 24)	Unmatched OR (for total cases)	Cornfield 95% CI	Matched OR (for total cases)	Exact 95% CI
Staff restaurant dispenser	7	10	17	6	8.5	1.92 - 40.8	12.0	1.78 - 512.97
Tank 1	5	15	13	8	2.6	0.68 - 10.28	7.0	0.9 - 315.48
Tank 2	0	2	2	1	2.19	0.14 - 67.33	-	
Tank 3	0	1	1	2	0.5	0.02 - 8.03	-	
Tank 4	0	4	4	2	2.32	0.3 - 21.14	2.0	0.29 - 22.3
Tank 5	0	4	4	5	0.8	0.15 - 4.28	0.75	0.11 - 4.43
Tank 6	0	2	2	1	2.19	0.14 - 67.33	-	
Tank 7	0	0	0	0	-			
Any tank water	5	15	20	14	4.76	0.93 - 27.34	4.5	0.93 - 42.8
Tank 1 sources	7	12	19	10	6.65	1.45 - 33.06	11.0	1.60 - 473.47
Contaminated sources	7	14	21	12	10.5	1.71 - 83.27	-	

children were affected. There were more females among cases than in the control group (69.6% vs. 45.8%, p=0.1)

The strongest association between gastrointestinal illness and a single water source was with the water dispenser in the staff restaurant (OR 8.5, CI 1.9-40.8) (Table 2). All 7 confirmed cases and 10 of the 16 probable cases recalled drinking from the dispenser in the week prior to their illness. Restaurant employees reported that the dispenser had been filled from a rainwater tank in the period prior to the outbreak, however it is not possible to be absolutely sure which tank was used for this purpose. Tank 5 had been the usual source of water for the dispenser, but it had twice run dry during this time. Records from the Bureau of Meteorology are consistent with this. Rainfall totals for the island for the months of March, April and May were 402.1 mm, 71.3 mm and 73.4 mm respectively and for the fortnight starting 17 May a total of only 15.2 mm of rain was recorded. One employee reported that Tank 1, which was the closest tank to the restaurant, had probably been used in early June and it is thus possible that the dispenser was filled with water from Tank 1 immediately prior to the outbreak.

Drinking water obtained directly from any one rainwater tank was not significantly associated with illness but associations were also tested for 'grouped' sources of untreated drinking water. Tank 1 and the staff restaurant water dispenser were grouped as 'Tank 1 sources' on the basis that the dispenser was probably filled from this tank. A second group of 'contaminated sources' included each of the four tanks with demonstrated faecal contamination (Tanks 1 to 4) and the staff restaurant water dispenser. Being a case was associated with drinking water from either the 'Tank 1 sources' (OR 6.65, CI 1.45-33.1) or from the 'contaminated sources' (OR 10.5, CI 1.7-83.3).

Analysis of the association between illness and eating food at the staff restaurant was confounded by the presence of the water dispenser. For those who did not drink water from the dispenser, there was no significant association between illness and eating at the restaurant (OR 2.0, CI 0.2-21.7). No significant associations with specific foods were identified. No unpasteurised dairy products had been consumed.

Staff with access to cooking facilities were asked to identify the source of any food used when preparing their own meals. No associations between illness and food sources were identified.

None of those questioned reported contact with pets and there was minimal contact with other animals.

Response to the outbreak

Resort management cooperated with efforts to investigate and curtail the outbreak. They wrote to all staff in the main accommodation areas on 17 June to advise them of the outbreak and to warn that rainwater was a suspected source of infection. Staff were advised not to consume rainwater and to empty any existing containers. All those with symptoms were requested to immediately attend the island doctor. Island staff drained and removed Tank 1 on 19 June, and several other tanks had taps locked or removed. Management subsequently agreed that water dispensers should not be filled from rainwater tanks.

In response to the food handling issues identified, a number of staff were immediately replaced and a series of staff training sessions run by EHOs were initiated. These commenced in July 1997 and involved one of the first 'Foodsafe' food handler training programs in Australia. This joint development by the Australian Institute of Environmental Health and Healthway (Western Australia) was designed to improve food safety practices in commercial premises and may be implemented on a nationwide basis. In addition, management has instigated an extensive program of structural improvement within food premises on the island.

Discussion

The epidemiological and microbiological data from this investigation indicate that untreated rainwater was the most likely source of *Campylobacter* infection in this outbreak. Outbreaks of *Campylobacter* infection have been traced to contaminated water sources in Europe,[2,6,8,9] North America,[4,5,7] New Zealand[10] and elsewhere, but the authors are not aware of any previous reports from Australia.

The epidemiological evidence that this was a waterborne outbreak is strong. Gastrointestinal illness was significantly associated with drinking water from each of the following:

(i) the 'contaminated sources' (that is, the staff restaurant water dispenser and Tanks 1 to 4);

(ii) the 'Tank 1 sources' (that is, the staff restaurant water dispenser and Tank 1); and

(iii) the staff restaurant water dispenser alone. This dispenser was being refilled with tank water prior to the outbreak.

The microbiological evidence is not conclusive but the presence of faecal contamination in Tanks 1 to 4 supports the possibility that *Campylobacter* species were also present in one or more of these tanks. However, *Campylobacter* species were not isolated from any of the water samples in this investigation and other outbreaks of *Campylobacter* enteritis presumed to be waterborne on epidemiological grounds, have had similar difficulties.[2,4,5,10] These factors all underline the need to develop more sensitive detection techniques. *Campylobacter* species are notoriously difficult to culture from food and environmental sources, partly due to their ability to enter a non-cultivatable state.[15] The development of specific molecular-based methodologies for detection of *Campylobacter* species in such samples, and the demonstration of viability when they are detected, will offer more accurate identification of these organisms than currently available with standard cultural detection methods.

The source of faecal contamination in Tanks 1 to 4 is not known but possibilities include birds, bats and possums whose faeces could have collected on the roofs or in the gutters associated with these tanks. *Campylobacter* have been found in the intestines of many domestic and wild animals including rodents and a variety of birds,[16,17] and faecal droppings from such infected animals can introduce *Campylobacter* into water supplies.[5,6] We postulate that the droppings of wild animals contaminated one or more of the rainwater tanks with *Campylobacter* and that the dispenser was filled from a contaminated tank. Tank 1 was the most likely source, but this cannot be proven.

At least two distinct strains of *Campylobacter jejuni* were involved in this outbreak. Multiple strains have been

reported in other outbreaks[7,8,9] and this may reflect contamination by animals carrying more than one strain.

Other potential sources of infection were considered, including food from the staff dining room, food prepared by self-catering staff, direct contact with animals and swimming pools. No significant associations with illness were identified for any of these factors.

This outbreak resulted in substantial morbidity and time off work and raises a number of additional public health issues. It is a reminder that untreated rainwater is a potential source of infection for pathogens such as C*ampylobacter* and is a risky source of drinking water. Furthermore, while food was not implicated in this outbreak, the investigation did identify food handling practices that posed a considerable risk for foodborne illness. These were directly addressed at the staff restaurant and a broad ongoing program is being introduced at this resort and others like it.

This investigation was subject to a number of limitations. The delay between onset of illness and the case control study was over four weeks for some cases, and this may have affected the accuracy of recall for food and water consumed. This was, to some extent, mitigated by the use of food lists when the questionnaire was administered and by the publicity given to both the restaurant food and rainwater tanks as possible sources of infection at the time of the outbreak. Most cases and controls were certain about which water sources they had used and whether they had eaten at the staff restaurant. There was less certainty about specific foods consumed. The fact that *Campylobacter* were not isolated from any environmental samples and that no water sample was available from the water dispenser in the staff restaurant were also problematic.

In summary, untreated rainwater contaminated by the faeces of wild animals appears to have been the source of infection in this outbreak of *Campylobacter* enteritis.

Acknowledgements

We gratefully acknowledge the assistance of Dr Jeffrey Hanna, Public Health Physician Tropical Public Health Unit, in the planning and analysis of this investigation and the preparation of this report.

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Editorial
Outbreak of *Campylobacter* enteritis, north Queensland

The accompanying article by Merritt et al describes an outbreak of gastrointestinal illness in which *Campylobacter jejuni* is identified as the causative organism. The article allows us to focus on an organism which is a significant cause of morbidity in Australia and elsewhere. Recent reports from the National Notifiable Diseases Surveillance System (NNDSS) show that campylobacteriosis is reported more frequently than any other acute infection and that is without any reports from the most highly populated state, New South Wales.[1]

Although this article does not find a definitive source for the *Campylobacter*, there is a strong suspicion that certain water sources are contaminated. The article does, however, highlight some of the difficulties faced by those who are attempting to investigate such outbreaks. Timeliness was certainly an issue, with the case control study not able to be initiated until some weeks after the first report of illness. The potential for various forms of bias exists, especially in the choosing of controls.

Some reviewers considered the analysis should have been based on matched cases and controls and others thought it should be unmatched. Both sets of results are provided for comparison by the interested reader. In any event the differences in these calculations are not marked and may be overshadowed by the possibility of bias.

1. A higher number of reports is received for Hepatitis C unspecified, but these are not all incidentreports.

Notice

Update of subscription details

Tenders have been invited for the printing and mailing of *CDI.*

As part of the process a review of the current mailing list is currently in progress.

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This form will also appear in the next two issues of *CDI*. At the end of October the mailing list will be updated to include only those who have returned forms.

Neisseria canis infection: a case report

Sandie Safton,[1] Gavine Cooper,[1] Michael Harrison,[1] Lynne Wright[1] and Paul Walsh[2]

Abstract

The third case report, which is the first in Australia, of human infection with *Neisseria canis* is documented. This is the first case report in which the pathogenicity of this organism for humans is unequivocally demonstrated. *Commun Dis Intell* 1999;23:221.

Introduction

*Neisseria canis (N. canis)*was first described by Berger in 1962.[1] The bacterium's normal habitat is the throat of the cat and dog. It is regarded as a true *Neisseria* with phenotypic properties that allow its recognition as a distinct species.[2,3] Only two previous case reports of human infection have been found by the authors.[4,5] The first case of human infection with *N. canis* was published by Hoke and Vedros[4] in 1982. This isolate came from a cat bite wound on a child. No other clinical details were described. In 1989 *N. canis* was reported in a mixed culture that included *Pasturella multocida* (*P. multocida)* and *Eiknella corrodens* from a cat bite wound on the arm of a previously healthy 36 year old woman. The wound was inflamed and the patient was successfully treated with amoxycillin. *P. multocida* was regarded as the primary pathogen in this case.[5]

Clinical Features

The patient, a 50 year old male normally in good health, presented with a purulent wound to the sole of his foot, with surrounding cellulitis. The patient recalled having trod on a dog bone a few days previously. A swab for culture was taken and antibiotics commenced (metronidazole and amoxycillin/clavulanic acid). Seven days later he made a complete recovery apart from some residual induration.

Methods

Laboratory Diagnosis

Standard bacteriologic techniques as outlined in the *Manual of Clinical Microbiology* [6]were used. The Gram stain showed moderate numbers of polymorphs. A moderate pure growth of a small gram negative coccus was obtained on aerobic blood agar, with the formation of yellowish non-haemolytic, 2 - 4 mm slightly flat topped colonies after 48 hours. It grew well on nutrient agar but did not grow on MacConkey agar. The organism was a facultative anaerobe, non-capnophilic and growth at 37°C was better than at 30°C or 42°C. The remainder of the diagnostic tests were consistent with the identification of *N. canis,* and it was sensitive to benzylpenicillin, erythromycin, and tetracycline but resistant to vancomycin.

A conserved segment (441 base pairs) of the isolate's RNA was subject to molecular studies, using BLAST analysis with the GeneBank[7] data bank. A significant similarity was found with a sequence of 422 matching base pairs (95%) with GenBank Accession number L06170 - *Neisseria canis* ATCC 14687.

Discussion

It is considered without doubt that *N. canis* was pathogenic. Currently the organism is a very rare isolate associated with cat or dog contact, but it may be under reported. The laboratory diagnostic clues are the isolation of an oxidase positive, gram negative, non-fastidious coccus that is very strongly catalase positive and forms dull yellow flat-topped non-haemolytic colonies on Day 2. It is nitrate positive but otherwise essentially asaccharolytic and rather inert in its biochemical reactions. It is described in the literature as galactosidase negative, tributyrin hydrolysis negative, DN'ase negative, nitrite negative and polysaccharide synthesis negative.[3,6] Currently there is no reason to suspect that the organism would not be covered by the current *Australian Antibiotic Guidelines*[8] for the management of animal bites.

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Measles-Mumps-Rubella immunisation, autism and inflammatory bowel disease: update

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Early last year we reported[1] on a study by Wakefield and colleagues which suggested there may be an association between measles containing vaccine, inflammatory bowel disease (IBD) and autism.[2] The evidence for either association was very weak[1] and the study was conducted on a highly selected group of subjects. Since then several epidemiological investigations have found no evidence for any association with autism and/or IBD.[5,6,7,8,9] Also, specific virological assays in patients with IBD, the proposed aetiological link for autism after measles-mumps-rubella (MMR) vaccination, have not detected measles virus.[3,4] Following the publication of the Wakefield study[2] however, there has been a measurable decrease in the uptake of MMR in the United Kingdom (UK).[10]

In June this year two further reports were published that provide no support for a causal link between measles vaccine and autism.[11,12] The Working Party on MMR Vaccine of the UK's Committee on Safety of Medicine's study[11] evaluated the reports of autism, Crohn's disease, and similar disorders developing after MMR or MR vaccination, collected by a firm of solicitors. A systematic review of these cases lead the Working Party to conclude that the information available (which was of variable quality, subject to selection bias and lacked a control group) did not support the suggested causal association between measles vaccine and autism or Crohn's disease.

The second report, by Taylor et al,[12] is a population-based study that overcomes many of the limitations of the Working Party's study. Taylor's study investigated 498 children with autism born since 1979 in the North Thames Region. These children's measles vaccination status was determined from an independent register. The investigators found that:

- there was a steady increase in cases of autism over time, however there was no 'step-up' after the introduction of MMR in 1988;
- the age of diagnosis of children with autism was not dependent on when or if a child had been vaccinated;
- vaccination coverage rates in cases did not differ significantly from that for the region as a whole; and
- developmental regression was not clustered in the months after vaccination.

These results should alleviate concerns about the possibility of MMR causing autism or IBD and hopefully reassure parents and others as to the safety of MMR.

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The NCIRS was established by the National Centre for Disease Control, Commonwealth Department of Health and Aged Care. The Centre analyses, interprets, and evaluates national surveillance data on immunisation coverage and vaccine preventable diseases. NCIRS also identifies research priorities, and initiates and coordinates research on immunisation issues and the epidemiology of vaccine preventable diseases in Australia.

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

The 289 notifications of pertussis infection in this reporting period represent a further fall compared to historical figures which are high because of the large epidemic that occurred from mid 1996 to early 1998. When examined by month of onset, April is historically the month with the lowest number of cases. The number of pertussis cases with onset in April 1999 is the lowest since April 1993 (Figure 1). The male to female ratio for the current reporting period is 1:1.2 and most cases are in the 10-14 age group (12%) although there is a broad spread of age distribution with considerable activity across the range. Most notifications in this reporting period are from Queensland (122) and 55% of these are in the 20 to 49 year age groups. Figure 2 shows a comparative age distribution for the current and previous 4 week reporting periods and 5 years of historical data from 1 July 1994 to 30 June 1999.

Figure 1. Notifications of pertussis, Australia, 1991 to 1999, by month of onset

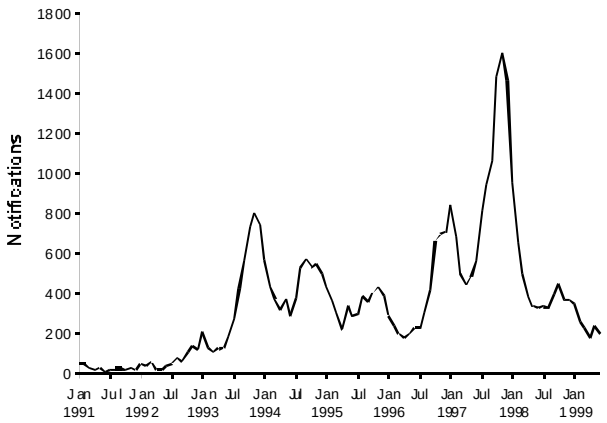
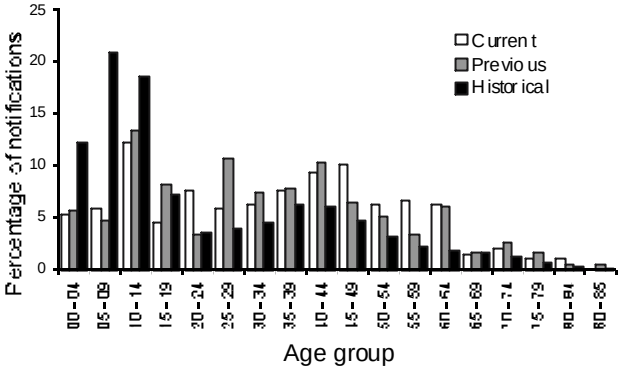


Figure 2. Notifications of pertussis, Australia, by age group, current and previous reporting periods and 5 years' historical data to 30 June 1999



Measles and rubella

Small numbers of notifications continue to occur although they continue to be low compared to historical data.

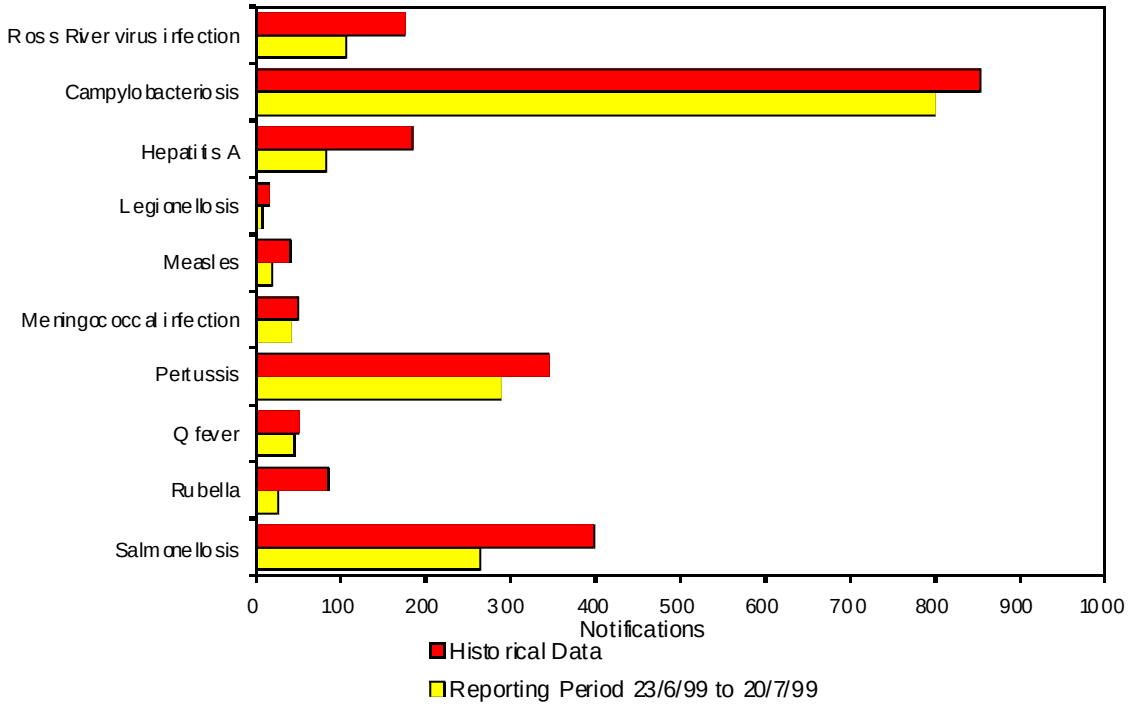
Tables

There were 4,907 notifications to the National Notifiable Diseases Surveillance System (NNDSS) in the four week period, 23 June to 20 July 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 1,946 reports received by the *CDI* Virology and Serology Laboratory Reporting Scheme (LabVISE) in the four week period, 17 June to 14 July 1999 (Tables 3 and 4).

The Australian Sentinel Practice Research Network (ASPREN) data for weeks 25 to 28, ending 18 July 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 preventable by vaccines recommended by the NHMRC for routine childhood immunisation, received by State and Territory health authorities in the period 23 June to 20 July 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	2	1	2	0	0	0	0	5	3	24	18
Measles	1	3	0	11	0	0	4	0	19	28	180	211
Mumps	3	1	0	2	1	1	7	1	16	8	93	88
Pertussis	3	48	0	122	20	27	61	8	289	313	1,876	4,029
Rubella[3]	3	2	1	15	0	0	6	0	27	55	221	431
Tetanus	0	0	0	1	0	0	0	0	1	0	1	3

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 2. Notifications of diseases received by State and Territory health authorities in the period 23 June to 20 July 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
Arbovirus infection (NEC)	0	0	1	1	0	0	1	0	3	4	89	62
Barmah Forest virus infection	0	11	0	19	0	0	0	1	31	25	444	376
Brucellosis	0	0	0	3	0	0	1	0	4	4	15	24
Campylobacteriosis[5]	21	-	18	202	142	38	278	101	800	932	6,842	6,256
Chancroid	0	0	0	0	0	0	0	0	0	0	0	1
Chlamydial infection (NEC)[6]	25	NN	62	337	77	28	88	132	749	853	6,316	5,900
Cholera	0	0	0	0	0	0	0	0	0	0	2	3
Dengue	0	0	0	1	1	0	0	0	2	55	155	340
Donovanosis	0	NN	1	0	NN	0	0	0	1	3	10	22
Gonococcal infection[7]	2	52	65	67	12	0	54	71	323	424	3,089	2,914
Haemolytic uraemic syndrome[8]	NN	0	0	0	0	0	NN	0	0	1	11	7
Hepatitis A	0	18	9	18	7	1	11	19	83	233	914	1,798
Hepatitis B incident	0	1	1	3	2	0	5	1	13	28	165	155
Hepatitis B unspecified[9]	4	143	0	52	0	5	184	37	425	567	3,745	4,080
Hepatitis C incident	0	0	0	-	5	0	4	2	11	17	166	152
Hepatitis C unspecified[9]	22	367	19	235	90	21	499	56	1,309	1,770	10,628	11,986
Hepatitis (NEC)[10]	1	1	0	2	0	0	0	NN	4	0	10	9
Hydatid infection	0	0	0	2	1	0	1	0	4	5	20	20
Legionellosis	0	2	0	2	1	0	2	1	8	29	162	153
Leprosy	0	0	0	1	0	0	0	0	1	0	1	2
Leptospirosis	0	6	0	11	0	0	0	0	17	13	257	91
Listeriosis	0	1	0	2	0	1	0	1	5	5	27	34
Malaria	1	3	3	26	2	0	2	0	37	99	401	480
Meningococcal infection	2	13	0	12	2	0	9	4	42	50	240	183
Ornithosis	0	NN	0	0	0	0	5	0	5	4	48	22
Q Fever	0	7	0	32	2	0	5	0	46	49	283	309
Ross River virus infection	0	25	4	51	0	4	4	18	106	80	3,839	2,295
Salmonellosis (NEC)	6	51	15	98	19	4	48	23	264	647	5,062	5,168
Shigellosis[5]	0	-	4	8	2	1	5	8	28	31	346	365
SLTEC, VTEC[11]	NN	0	0	NN	2	0	NN	NN	2	1	15	8
Syphilis[12]	0	18	11	85	1	1	2	7	125	132	1,060	771
TTP[13]	0	0	0	0	0	0	0	0	0	0	0	0
Tuberculosis	1	44	3	5	5	2	23	6	89	114	801	720
Typhoid[14]	0	2	0	1	0	0	0	1	4	2	43	48
Yersiniosis (NEC)[5]	0	-	0	7	1	0	1	0	9	8	94	146

1. Diseases preventable by routine childhood immunisation are presented in Table 1.
2. For HIV and AIDS, see Tables 8 and 9.
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. Not reported for NSW because it is only notifiable as 'foodborne disease' or 'gastroenteritis in an institution'.
6. WA: genital only.
7. NT, Qld, SA and Vic: includes gonococcal neonatal ophthalmia.
8. Nationally reportable from August 1998.
9. Unspecified numbers should be interpreted with some caution as the magnitude may be a reflection of the numbers of testings being carried out.
10. Includes hepatitis D and E.
11. Infections with *Shiga*-like toxin (verotoxin) producing *E. Coli* (SLTEC/VTEC) became nationally reportable in August 1998.
12. Includes congenital syphilis.
13. Thrombotic thrombocytopaenic purpura became nationally reportable in August 1998.
14. NSW, Qld: includes paratyphoid.

NN Not Notifiable.

NEC Not Elsewhere Classified.

- Elsewhere Classified.

Table 3. Virology and serology laboratory reports by State or Territory[1] for the reporting period 17 June to 14 July 1999, and total reports for the year

	State or Territory[1]									
	ACT	NSW	NT	Qld	SA	Tas	Vic	WA	Total this period	Total reported in *CDI* in 1999
Measles, mumps, rubella										
Measles virus							3		3	146
Mumps virus								2	2	39
Rubella virus	1			10		1	1		13	69
Hepatitis viruses										
Hepatitis A virus			4	5	9			6	24	253
Hepatitis D virus					1				1	3
Arboviruses										
Ross River virus		2		14	2		3	10	31	1,214
Barmah Forest virus		2		8				2	12	141
Japanese encephalitis virus								1	1	1
Flavivirus (unspecified)							1		1	14
Adenoviruses										
Adenovirus type 1							1		1	17
Adenovirus type 2							3		3	11
Adenovirus type 4							1		1	8
Adenovirus type 37							1		1	13
Adenovirus type 40								5	5	43
Adenovirus not typed/pending		8	1	3	29		25	7	73	716
Herpes viruses										
Cytomegalovirus		17	1	9	20	1	22	1	71	713
Varicella-zoster virus		6	1	26	29		24	12	98	1,031
Epstein-Barr virus		4	2	54	107	1	18	8	194	1,573
Other DNA viruses										
Papovavirus group							1	1	2	12
Molluscum contagiosum								1	1	11
Parvovirus				11	2	3	17	5	38	265
Picorna virus family										
Coxsackievirus A9							1		1	4
Coxsackievirus A16							2		2	4
Coxsackievirus B5							1		1	3
Echovirus type 11		14					1		15	58
Poliovirus type 3 (uncharacterised)							1		1	4
Rhinovirus (all types)		9			3		5	13	30	231
Enterovirus not typed/pending			1			1	3	47	52	492
Ortho/Paramyxoviruses										
Influenza A virus		94		23	74		57	2	250	649
Influenza A virus H3N2							9	1	10	17
Influenza B virus		7		1	5		1	1	15	86
Parainfluenza virus type 1				1	4				5	29
Parainfluenza virus type 2					10		9	2	21	76
Parainfluenza virus type 3		8		4	8		7	4	31	373
Respiratory syncytial virus		198		52	45	4	58	11	368	1,169
Other RNA viruses										
HTLV-1			1						1	9
Rotavirus		87			38	9	25	9	168	720
Astrovirus							2		2	4
Norwalk agent							6		6	55

Table 3. Virology and serology laboratory reports by State or Territory[1] for the reporting period 17 June to 14 July 1999, and total reports for the year (continued)

	State or Territory[1]									
	ACT	NSW	NT	Qld	SA	Tas	Vic	WA	Total this period	Total reported in *CDI* in 1999
Other										
Chlamydia trachomatis not typed		10	15	74	53	1	19	39	211	1,757
Chlamydia psittaci							11	1	12	59
Chlamydia species				1					1	9
Mycoplasma pneumoniae		11		28	5		28	4	76	735
Coxiella burnetii (Q fever)		1		8			5	1	15	106
Bordetella pertussis		1		46			22	4	73	428
Legionella pneumophila								1	1	9
Legionella longbeachae					2				2	27
TOTAL	1	479	26	378	446	21	394	201	1,946	13,406

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 17 June to 14 July 1999

State or Territory	Laboratory	Reports[1]
New South Wales	Institute of Clinical Pathology & Medical Research, Westmead	132
	New Children's Hospital, Westmead	122
	South West Area Pathology Service, Liverpool	202
Queensland	Queensland Medical Laboratory, West End	415
	Townsville General Hospital	12
South Australia	Institute of Medical and Veterinary Science, Adelaide	445
Tasmania	Northern Tasmanian Pathology Service, Launceston	16
Victoria	Monash Medical Centre, Melbourne	105
	Royal Children's Hospital, Melbourne	152
	Victorian Infectious Diseases Reference Laboratory, Fairfield	136
Western Australia	PathCentre Virology, Perth	209
TOTAL		1,946

1. Due to computer processing problems figures from the Institute of Clinical Pathology & Medical Research, Westmead have been under reported since February 1999. Reports of influenza from Westmead have been manually corrected in this report; numbers for other organisms remain under reported. It is anticipated that the reporting system will be corrected for the next issue.

Table 5. Australian Sentinel Practice Research Network reports, weeks 25 to 28, 1999

Week number	25		26		27		28	
Week ending on	27 June 1999		4 July 1999		11 July 1999		18 July 1999	
Doctors reporting	49		47		49		48	
Total encounters	6,457		6,087		6,496		6,289	
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	67	10.4	78	12.8	91	14.0	87	13.8
Rubella	0	0.0	1	0.2	2	0.3	0	0.0
Measles	1	0.2	0	0.0	0	0.0	1	0.2
Chickenpox	10	1.5	4	0.7	7	1.1	13	2.1
New diagnosis of asthma	14	2.2	9	1.5	13	2.0	12	1.9
Post operative wound sepsis	3	0.5	10	1.6	11	1.7	10	1.6
Gastroenteritis	67	10.4	40	6.6	52	8.0	60	9.5

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

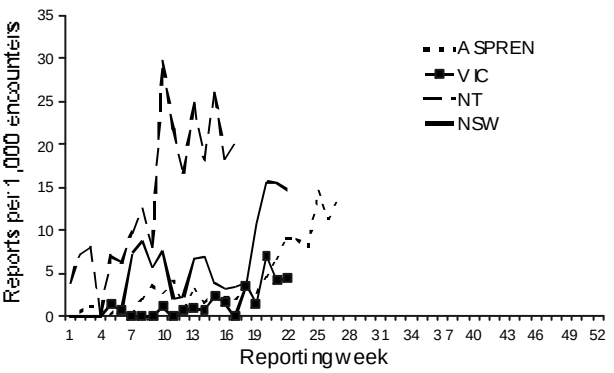
National Influenza Surveillance, 1999

Three types of data are included in National Influenza Surveillance, 1999. These are sentinel general practitioner surveillance conducted by the Australian Sentinel Practice Research Network, Department of Human Services (Victoria), Department of Health (New South Wales) and the Tropical Influenza Surveillance Scheme, Territory Health (Northern Territory); laboratory surveillance data from the Communicable Diseases Intelligence Virology and Serology Laboratory Reporting Scheme, LabVISE, and the World Health Organization Collaborating Centre for Influenza Reference and Research; and absenteeism surveillance conducted by Australia Post. For further information about these schemes, see CDI 1999; 23:56.

Sentinel general practitioner surveillance

An increase in consultation rates for influenza-like illness reported by the ASPREN, NSW and Victorian schemes was apparent in April. Rates for influenza-like illness recorded by ASPREN were lower this year than for the same period in 1998. In contrast, the consultation rates for influenza activity reported by the Tropical Influenza

Figure 4. Sentinel general practitioner influenza consultation rates, 1999, by scheme and week

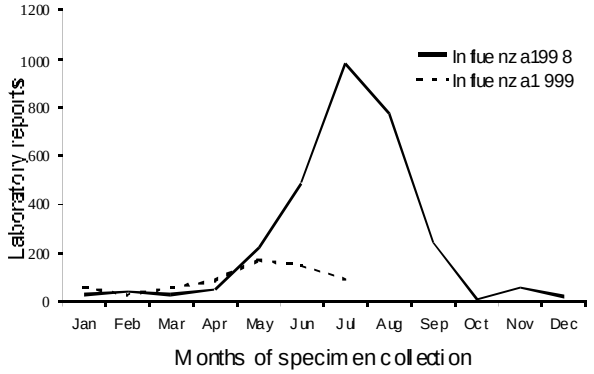


Surveillance Scheme showed higher rates from March to June than for the same period in 1998. Victorian rates were similar to those recorded for the corresponding period in 1998.

Laboratory surveillance

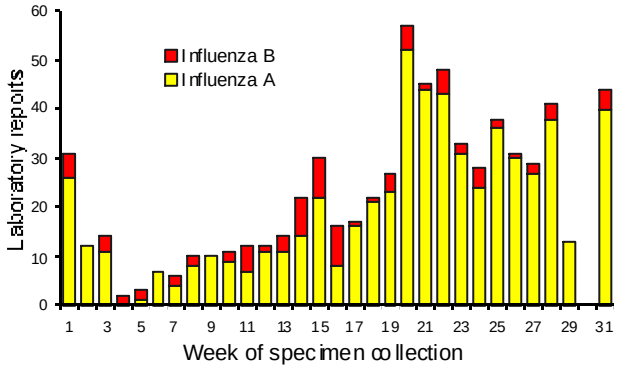
Figure 5 shows the number of laboratory reports for 1998 and 1999. Data for 1999 is provided only for January to July. For the year to date there have been 735 laboratory reports of influenza.

Figure 5. Laboratory reports of influenza, 1998-99, by month of specimen collection



To July 1999, there have been 649 (88.3%) reports of influenza A of which 17 were H3N2 and 2 were H1N1 (Figure 6). There were 86 (11.7%) reports of influenza B. To date there has been no lodging of influenza reports in week 30.

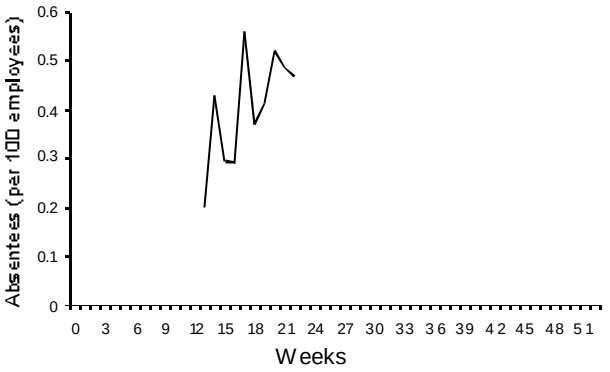
Figure 6. Laboratory reports of influenza, 1999, by type and by week of specimen collection



Absenteeism surveillance

Australia Post reports employees absent if they are not at work for three or more consecutive days in one week. The average rates for May were 0.45% which is higher than for May 1998 (0.28%) (Figure 7). There are no changes in the reports of absenteeism since the previous report.

Figure 7. Absenteeism rates in Australia Post, 1999



Sentinel Chicken Surveillance Programme

Sentinel chicken flocks are used to monitor flavivirus activity in Australia. The main viruses of concern are Murray Valley encephalitis (MVE) and Kunjin which cause the potentially fatal disease Australian encephalitis in humans. Currently 26 flocks are maintained in the north of Western Australia, seven in the Northern Territory, nine in New South Wales and ten in Victoria. The flocks in Western Australia and the Northern Territory are tested year round but those in New South Wales and Victoria are tested only from November to March, during the main risk season.

Results are coordinated by the Arbovirus Laboratory in Perth and reported bimonthly. For more information see CDI 1999;23:57-58.

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May/June 1999

Sentinel chicken serology was carried out for 24 of the 27 flocks in Western Australia in May and June 1999. There were again a large number of seroconversions to flaviviruses in the Kimberley, Pilbara and Gascoyne flocks during this period. Twenty-five of the total 34 seroconversions occurred in May 1999. The number of chickens positive for flavivirus antibodies by ELISA and the virus (or viruses) they were infected with is shown in Table 6.

Serum samples from six of the seven Northern Territory sentinel chicken flocks were tested in our laboratory in May and June 1999. There were seroconversions to flaviviruses at Howard Springs and Leanyer (Darwin area) and from Beatrice Hill Farm and Tennant Creek. The number of

Table 6. Flavivirus seroconversions in Western Australian sentinel chicken flocks in May and June, 1999

Location	May 1999			June 1999
	MVE	KUN	MVE/KUN	MVE
Kimberley				
Kalumburu		1	1	
Wyndham	1			
Kununurra	1			
Fitzroy Crossing	4			
Lombadina	1	1		
Derby*	2			
Broome*	2			
Pilbara				
Port Hedland				1
Karratha		1		1
Harding Dam*				3
Tom Price	1			
Paraburdoo	2			1
Newman*			1	
Exmouth	3			3
Gascoyne				
Carnarvon	3			

* 2 flocks of 12 chickens at these sites
MVE Antibodies to Murray Valley encephalitis virus detected by ELISA
KUN Antibodies to Kunjin virus detected by ELISA
MVE/KUN Antibodies to both MVE and KUN viruses detected by ELISA
FLAVI Antibodies to a flavivirus only (not MVE or KUN) detected by ELISA

chickens positive for flavivirus antibodies by ELISA and the virus (or viruses) they were infected with is shown in Table 7. Seroconversions to MVE virus from Tennant Creek in May have not yet been confirmed.

Details of the locations of all chicken flocks are given in *CDI* 1999;23:57-58.

Table 7. Flavivirus seroconversions in the Northern Territory sentinel chicken flocks in May and June, 1999

Location	May 1999		June 1999			
	MVE	KUN	MVE	KUN	MVE/KUN	FLAVI
Howard Springs	1					
Leanyer		1		1	1	1
Beatrice Hill	3		2			
Tennant Creek	2					

MVE Antibodies to Murray Valley encephalitis virus detected by ELISA
KUN Antibodies to Kunjin virus detected by ELISA
MVE/KUN Antibodies to both MVE and KUN viruses detected by ELISA
FLAVI Antibodies to a flavivirus only (not MVE or KUN) detected by ELISA

HIV and AIDS Surveillance

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

Tabulations of diagnoses of HIV infection and AIDS are based on data available three months after the end of the reporting interval 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 March 1999, as reported to 30 June 1999, are included in this issue of CDI (Tables 8 and 9).

Table 8. New diagnoses of HIV infection, new diagnoses of AIDS and deaths following AIDS occurring in the period 1 to 31 March 1999, 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	3	0	2	0	0	1	2	8	10	18	20
	Male	0	35	1	9	1	0	10	3	59	66	138	184
	Sex not reported	0	0	0	0	0	0	1	0	1	0	2	2
	Total[1]	0	38	1	11	1	0	12	5	68	76	158	206
AIDS diagnoses	Female	0	1	0	0	0	0	0	0	1	1	2	3
	Male	0	5	0	1	0	0	0	0	6	20	20	72
	Total[1]	0	6	0	1	0	0	0	0	7	21	22	75
AIDS deaths	Female	0	0	0	0	0	0	0	0	0	2	2	3
	Male	0	0	0	0	0	0	4	0	4	8	25	34
	Total[1]	0	0	0	0	0	0	4	0	4	10	26	37

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

Table 9. Cumulative diagnoses of HIV infection, AIDS and deaths following AIDS since the introduction of HIV antibody testing to 30 June 1999, by sex and State or Territory

		State or Territory								
		ACT	NSW	NT	Qld	SA	Tas	Vic	WA	Australia
HIV diagnoses	Female	23	587	8	134	57	5	202	107	1,123
	Male	188	10,569	105	1,888	652	77	3,775	878	18,132
	Sex not reported	0	258	0	0	0	0	26	0	284
	Total [1]	211	11,433	113	2,029	709	82	4,016	988	19,581
AIDS diagnoses	Female	8	171	0	46	21	3	67	26	342
	Male	85	4,526	34	792	327	44	1,586	344	7,738
	Total [1]	93	4,709	34	840	348	47	1,660	372	8,103
AIDS deaths	Female	2	113	0	30	15	2	47	16	225
	Male	63	3,126	24	554	225	28	1,246	245	5,511
	Total [1]	65	3,247	24	586	240	30	1,299	262	5,753

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

Childhood Immunisation Coverage

Tables 10 and 11 provide the latest quarterly report on childhood immunisation coverage from the Australian Childhood Immunisation Register (ACIR).

The data show the percentage of children fully immunised at age 12 months for the cohort born between 1 January and 31 March 1998 and at 24 months of age for the cohort born between 1 January and 31 March 1997, according to the Australian Standard Vaccination Schedule.

A full description of the methodology used can be found in CDI 1998;22:36-37.

Table 10. Percentage of children immunised at 1 year of age, preliminary results by disease and State for the birth cohort 1 January to 31 March 1998; assessment date 30 June 1999

	State or Territory								
Vaccine	ACT	NSW	NT	Qld	SA	Tas	Vic	WA	Australia
Total number of children	1,070	21,453	982	12,233	4,697	1,541	14,848	6,195	63,019
Diphtheria, Tetanus, Pertussis (%)	89.5	85.3	79.7	89.4	89.7	88.3	89.1	87.4	87.6
Poliomyelitis (%)	89.3	85.0	79.1	88.8	89.6	88.3	89.3	87.2	87.3
Haemophilus influenzae type b (%)	89.2	84.7	84.9	89.7	89.1	88.0	88.8	87.1	87.4
Fully Immunised (%)	88.7	83.5	77.3	88.0	88.6	87.7	87.9	85.9	86.1
Change in fully immunised since last quarter (%)	+1.0	+0.8	+2.7	+1.5	+1.1	+0.5	+1.4	+1.5	+1.2

Table 11. Proportion of children immunised at 2 years of age, preliminary results by disease and State for the birth cohort 1 January to 31 March 1997; assessment date 30 June 1999[1]

	State or Territory								
Vaccine	ACT	NSW	NT[1]	Qld	SA	Tas	Vic	WA	Australia
Total number of children	1051	22,006	946	11,888	4,628	1,558	15,454	6,475	64,006
Diphtheria, Tetanus, Pertussis (%)	86.2	81.3	67.7	85.5	83.2	83.6	83.6	82.0	82.8
Poliomyelitis (%)	88.9	85.6	79.9	91.0	88.4	89.5	89.3	85.3	87.7
Haemophilus influenzae type b (%)	85.4	81.3	77.2	85.8	81.3	83.3	83.6	82.2	82.8
Measles, Mumps, Rubella (%)	89.7	85.9	81.8	90.9	88.0	89.0	88.6	86.7	87.8
Fully Immunised (%)[2]	81.4	70.4	57.8	80.3	71.6	74.8	74.7	70.5	73.5
Change in fully immunised since last quarter (%)	+3.7	+3.5	+3.2	+2.8	+3.5	+3.2	+2.7	+4.5	+3.2

1. The 12 months age data for this cohort was published in *CDI* 1998;22:233.
2. These data relating to 2 year old children should be considered as preliminary. The proportions shown as "fully immunised" appear low when compared with the proportions for individual vaccines. This is at least partly due to poor identification of children on immunisation encounter forms.

Acknowledgment: These figures were provided by the Health Insurance Commission (HIC), to specifications provided by the Commonwealth Department of Health and Aged Care. For further information on these figures or data on the Australian Childhood Immunisation Register please contact the Immunisation Section of the HIC: Telephone 02 6124 6607.

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Bulletin Board

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

The Queensland Institute of Medical Research
Symposium on Q Fever
13-14 October 1999
Brisbane, Queensland
Phone: 07 3844 1138
Fax: 07 3844 0909
Email: qfever@icms.com.au

Institute of Nanotechnology
The Surgery Room of the 21st Century
1-2 November 1999
The Diagnostic Centre of the 21st Century
3-4 November 1999
Glasgow, Scotland
Phone: 44 1786 447520
Fax: 44 1786 447530
Email: julie@nano.org.uk

The theme of the conference is taken from the Royal Academy of Engineering's publication *Medical Engineering - A Field With Potential.* The keynote address: *Lab-on-a-Chip Technologies - The Future* will be given by Professor Andreas Manz, Imperial College of Science, Technology and Medicine.

Some of the topics are of interest to communicable diseases. For details see contacts above.

Australasian Society for HIV Medicine Inc
11th Annual Conference
9-11 December 1999
Perth, Western Australia
Contact: ASHM Conference Secretariat
C/- ICMS Australasia Pty Ltd, GPO Box 2609,
Sydney, NSW, 2001
Phone: 02 9241 1478
Fax: 02 9251 3552

Advance notice

The First Pacific Rim Biomedical Seminar
Transportation of Infectious and Diagnostic Substances
3 March 2000
Sheraton on the Park
Sydney, NSW
Contact: Christine Sherwood
Phone: 1800 023 560; or
Sydney: 9693 2988
Email: sherwood@worldcourier.com.au

International Society of Travel Medicine/WHO/CDC
2nd European Conference of Travel Medicine
29-31 March 2000
Venice, Italy
Contact: Dr Walter Pasini, Italy
Phone: 390-541-24301
Fax: 390-541-25748
Email: wpasini@rimini.com

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
Email: Tom@micr.crg.cs.nsw.gov.au

Australian Infection Control Association
First Biennial Conference
Infection Control Beyond 2000
3-5 May 2000
Hilton Adelaide International, South Australia
Contact: AICA 2000 Secretariat
PO Box 1280 , Milton, Queensland 4064
Phone: 07 3369 0477
Fax: 07 3369 1512
Email: aica2000@im.com.au
Website: http://www.aica.org.au/aica2000.htm

Australian School of Environmental Studies
Arbovirus Research in Australia
3-7 July 2000
Couran Cove Nature Resort, Gold Coast, Queensland
Contact Dr Michael Brown, Queensland Institute of
Medical Research, PO Box Royal Brisbane Hospital,
Herston, Queensland, 4029
Website: http://www.mcaa.org.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.

Malaria in Kenya

Malaria epidemics in Kenya have become periodic since the 1980s. They have been characterized by transmission upsurges in the highlands in the western part of the country, and more recently in the semiarid north-eastern area, after the 1997 El Nino rains. These outbreaks were generally contained by case management, but the strategy began to fail with increasing chloroquine resistance. This year the epidemic districts were supplied with sulfa-pyrimethamine (SP). As a result, the outbreak was kept in check during the early stages, but as the intensity of transmission increased at the beginning of May, the number of patients outstripped the capacity of the health facilities, leading to a severe management crisis at all levels including in drug procurement systems.

Although a general upsurge of malaria cases has been reported in most of the districts which usually experience epidemics, the following 9 districts have been more severely affected: Buret, Gucha, Kisii, Mount Elgon, Narok, Nyamira, Trans Mara, Trans Nzoia and West Pokot.

Stocks of antimalarial drugs have now been exhausted in most districts owing to the unusually severe outbreak this year.

An integrated approach to malaria control is needed. Mortality rates are still at emergency level, and they are not expected to decrease, as the number of new cases continues to rise. Environmental conditions are still suitable for mosquito breeding.

Shigella in Guinea

Following reports of an outbreak of diarrhoeal disease, a rapid-response team has visited the district of Dabola located 450km from Conakry. Out of 7 patients examined, 5 were suffering from bloody diarrhoea. Of 7 samples sent to the laboratory at Donka hospital, 1 was identified as *Shigella dysenteriae* type 1 resistant to ampicillin, chloramphenicol and erythromycin; it was however sensitive to nalidixic acid. All 7 patients have been treated with norfloxacin and have recovered. No new case has been reported.

Cholera in Afghanistan

A total of 14,402 cases of severe diarrhoea, including cholera cases, has been reported between 29 May and 12 July. The most affected area is Kabul province, Central region where nearly 7,000 cases have occurred. Out of 9 samples tested, 5 were laboratory-confirmed as cholera. A significant increase in the number of suspect cholera cases was noted in Kunduz province, North-eastern region and various provinces in Southern region during the week 3 to 9 July. Drug supplies have been distributed and implementation of control activities is underway.

Crimean-Congo haemorrhagic fever, Russian Federation

On 26 July, the Ministry of Health reported that Crimean-Congo haemorrhagic fever had been confirmed by laboratory tests. A total of 65 cases has been reported, with 6 deaths (3 of which were children). Of those admitted to hospital, 44 have been discharged and 21 are still undergoing treatment. There have been no new cases since 22 July, and the epidemic is localized. Transmission appears to have occurred mainly through reservoirs in the environment (ticks).

Plague in Malawi

Since 18 June a number of sporadic suspect cases of plague have occurred in Nsanje district, Southern Region. Up to 21 July, 74 suspect cases had been reported from a total of 22 villages. Six villages along the Mozambican border were the most affected reporting around 3 to 4 suspect cases each. Some of the other villages reported only one suspect case each. The treatment of patients is under way as well as environmental control measures but drug supplies have been depleted and more may be needed.

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Website

http://www.health.gov.au/pubhlth/cdi/cdihtml.htm

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.

Contacts other than subscriptions

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