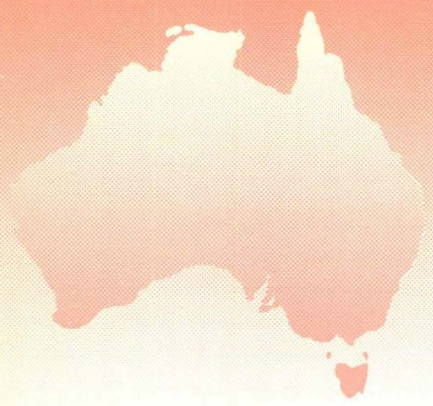

COMMUNICABLE DISEASES INTELLIGENCE

ISSN 0725-3141 VOLUME 19 NUMBER 21 16 October 1995

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COMMONWEALTH DEPARTMENT OF HUMAN SERVICES AND HEALTH

CDI is produced fortnightly by:
AIDS/Communicable Diseases Branch
Department of Human Services and Health
GPO Box 9848 Canberra ACT 2601
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COMMUNICABLE DISEASES NETWORK-AUSTRALIA
A National Network for Communicable Diseases Surveillance

A REVIEW OF NOTIFIED CASES OF LEGIONELLOSIS IN WESTERN AUSTRALIA, 1994

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Introduction

An apparent increase in notified cases of *Legionella* infection in Western Australia occurred in late 1994. Although an examination of data from previous years supported the likelihood that this was a seasonal effect, a review of all reported cases for 1994 was conducted in order to identify any clustering of cases or common risk factors.

Methods

Twenty-six notifications were identified from the Health Department of Western Australia (HDWA) infectious diseases database for 1994. Further information was compiled from *Legionella* data sheets, which were completed by both Communicable Diseases Control Unit medical staff and HDWA Environmental Health Officers, and were available for 24 of the cases.

Case definition

Notified patients had to fulfill at least one of the following criteria to be accepted as a case:

- isolation of *Legionella* from the patient
- a fourfold rise in antibody titre between acute and convalescent phase sera
- seroconversion to a titre of at least 256 and a compatible clinical illness
- a single titre of at least 512 and a compatible clinical illness.

Cases

Nineteen cases (79%) were male and five (21%) were female (Table). The ages ranged from 25 to 81 years (mean 59 years). Twenty-two cases resided in the Perth metropolitan area and one case came from each of the Kimberley, Central and Great Southern regions. There was no evidence of clustering by postcode. Eight cases (33%) were employed, 13 cases (54%) were retired, one case was an invalid pensioner, one was unemployed and the employment status of one case was unknown.

Seventeen patients (71%) presented between August and December, and the peak occurred in October when seven cases were notified (Figure).

Twenty patients presented with clinical features of acute lower respiratory tract infection and another had fever with chest X-ray evidence of pneumonia. Information on the presenting clinical syndrome of three patients was missing.

Twenty patients were hospitalised. Data were incomplete for four of these patients, however, for the other 16, the length of hospital stay was between six and 39 days (median 14.5 days, mean 16.9 days).

Three cases (cases 1, 11, 18) were admitted to intensive care units. Two required ventilation, case 11 because of a collapsed lung and case 1 for respiratory failure. All three cases survived.

There were two deaths, giving a case fatality rate of 8.3%. Case 4 died 19 days after admission for pneumonia. A single serum specimen taken on day 11 showed a titre of 512 to *L. longbeachae*. According to his wife he spent 'every spare moment in the garden'. *L. longbeachae* was subsequently isolated from peat mix at his home. Case 10 also died from pneumonia. He had an increase in titre to *L. longbeachae* from 512 to 32,768 over an 11 day period.

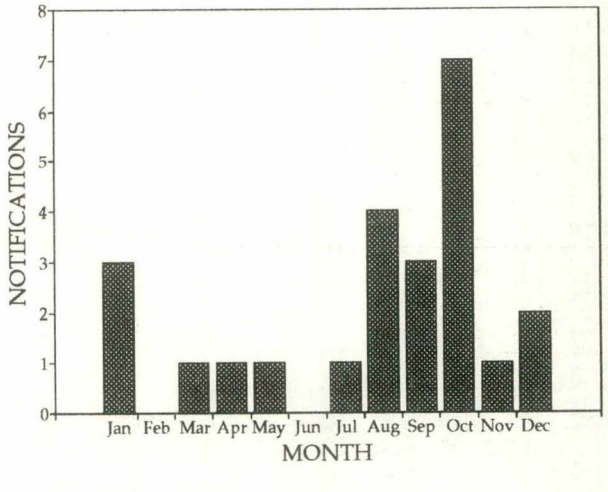
Laboratory diagnosis

The serological test used was the indirect fluorescent antibody test, conducted at the State Health Laboratory Services. Nineteen patients had a fourfold or greater rise in titre with paired sera and five others had a single titre of ≥512 and an illness compatible with *Legionella* pneumonia.

Eighteen cases had serological evidence of *L. longbeachae* alone (Table). Four cases had serological evidence of *L. pneumophila* alone and two cases had increased titres to both species.

In only two cases was *Legionella* isolated. Both isolates were *L. pneumophila* serogroup 1; one was cultured from bronchoalveolar lavage (case 9) and the other was from unspecified respiratory secretions (case 17).

Figure. Notified cases of legionellosis in Western Australia, 1994, by month of onset



Potential risk factors

Most patients had gardening exposure or one or more potential predisposing factors for legionellosis (Table).

Case 17 worked in an environment with opportunities for exposure to *Legionella* . He was a rural water authority worker who cleaned the inside of a waste water treatment tank with a high pressure hose 18 days before the onset of symptoms. Over seven days, he seroconverted to *L. pneumophila* serogroups 1 and 2 (titres were respectively 256 and 512), and additionally had a titre of 128 to *L. pneumophila* serogroup 4.

Thirteen cases (all with *L. longbeachae* infection) were keen gardeners and seven of them gave a precise history of gardening activities which involved the handling of potting soil or mulch between six and 10 days prior to their first symptoms. Samples of peat, soil or potting mix were collected from six of the gardener cases: in four instances no *Legionella* were grown; one sample which was collected five weeks after the patient's *L. longbeachae* infection grew *L. bozemanni*; *L. longbeachae* was cultured from peat mix and a *Legionella* species was isolated from soil conditioner at the home of case four.

Samples were taken from home water fixtures of six cases (two with *L. pneumophila* and four with *L. longbeachae*) but no *Legionella* were isolated from these sources.

Four of the cases had no potential predisposing factors for legionellosis and five cases had age 65 years or over only. Of this group, six were regular gardeners of whom four had used mulch or potting soil just before the illness onset. Two *L. longbeachae* cases, however, had no known risk factors.

Three cases were diagnosed with *Legionella* infection during hospital admissions for unrelated conditions. Cases 23 and 21 developed symptoms four and seven days respectively after admission for elective surgery. Although consistent with possible nosocomal infection, both cases were active gardeners with *L. longbeachae* infection, therefore it is more likely that their infections were community acquired. Case nine was an inpatient at a large teaching hospital for 47 days before the onset of legionellosis caused by *L. pneumophila*. She had occasional home leave during this time but was housebound by her illness. Testing

Table. Legionellosis cases, Western Australia, 1994

Case	Sex	Age (years)	*Legionella* species[1]	Gardening exposure[2]	Potential predisposing factors
1	M	67	L, P3	No	Asbestosis, smoker, alcohol, ischemic heart disease, steroids, past pulmonary melioidosis
2	M	29	P1	No	Alcohol, heavy smoker
3	F	68	L	Yes	Pulmonary fibrosis, ex-smoker
4	M	73	L	Yes	Chronic renal failure, congestive heart failure, CABG[3], cancer of prostate
5	M	31	P6	No	Alcohol, smoker, pulmonary embolus
6	M	25	L, P3, P4	No	Nil known
7	M	44	L	Yes (9 days)	Diabetes mellitus
8	F	59	L	Yes	Nil known
9	M	46	P1, P4	No	Alcohol cirrhosis, diabetes mellitus, steroids, smoker
10	M	77	L	No	Ischemic heart disease, CABG, smoker
11	M	62	L	Yes (7 days)	Nil known
12	M	34	L	No	Ischemic heart disease, ex-smoker
13	M	80	L	No	Chronic obstructive airways disease, ischemic heart disease
14	M	73	L	No	Nil known
15	M	70	L	Yes (7 days)	Ischemic heart disease, CABG
16	M	67	L	Yes (7 days)	Nil known
17	M	58	P1, P2	No	Asthma, ischemic heart disease, smoker, occupation
18	M	72	L	Yes (6 days)	Ischemic heart disease, CABG, smoker
19	M	46	L	Yes (9 days)	Nil known
20	M	69	L	Yes	Nil known
21	M	68	L	Yes	Post-trauma splenectomy
22	F	65	L	No	Nil known
23	F	51	L	Yes	Elective surgery
24	F	81	L	Yes (10 days)	Nil known

1. L - *L. longbeachae*; P - *L. pneumophila*; number represents serogroup.
2. Probable incubation period in brackets.
3. CABG - coronary artery bypass graft

of her home water fixtures did not reveal a source of *Legionella*.

Discussion

L. pneumophila is usually the commonest causative species in infections due to the Legionellaceae family [1,2,3]. The epidemic potential of *L. longbeachae* is well known but outbreaks contribute only a small proportion of all legionellosis, and there is little published on the epidemiology of sporadic *Legionella* disease[3].

The epidemiology of legionellosis in Western Australia is unusual because over the period 1989 to 1994, 60% of notified cases have been due to *L. longbeachae*, notwithstanding that *L. longbeachae* infection has been notifiable in Western Australia only since July 1991[4].

It is unlikely that the 1994 spring increase represented an outbreak as similar peaks have occurred in previous years and the total of cases for 1994 did not exceed the expected number. Both autumn and spring peaks of *L. longbeachae* infection are typical in Western Australia, and they coincide with the year's most intense gardening periods[4]. Several other factors support the interpretation that no point source was involved in our *L. longbeachae* case series including the diversity of potting soil brands used by the gardener cases, the lack of geographical clustering and the diagnosis of the infection in some non-gardeners.

Similarly, the *L. pneumophila* infections were unlikely to have had a common source; onset dates for these six infections were from January to October and several serogroups were identified.

Most of the predisposing factors previously reported to be associated with serious disease due to *Legionella* were found among the cases including advancing age, smoking, alcohol abuse, chronic heart, lung and renal disease, immunosuppression, diabetes mellitus, malignancy and male gender[1,2]. Smoking and alcohol consumption among our cases was, in fact, likely to have been under-reported as questions were only asked about current use. Splenectomy (case 23) is not a recognised risk factor[1,2] but is a biologically plausible one.

A range of natural and man-made aqueous habitats are known to be ecological niches for *Legionella*. They have also been found in soil[1,2] and *L. longbeachae* has been found commonly in ready-to-use potting soils in Australia[5]. During an investigation into an outbreak of *L. longbeachae* disease in South Australia in 1988–89, a point source was not identified, nor was soil proven to be a source, but gardening was found to be a common factor among most of the cases[6].

As *L. longbeachae* is the major cause of legionellosis in Western Australia, if exposure to man-made soil products is indeed a risk factor for disease, strategies to eliminate the organism from the soil, or to reduce exposure to the soil or soil products could prevent much of the disease in Western Australia. Further research into the ecology of *L. longbeachae* and its transmission to humans is required.

Acknowledgments

I would like to thank Richard Theobald and other officers of the Environmental Health Branch, and Communicable Diseases Control Unit staff of the Health Department of Western Australia for collecting the data and providing the reports on which this review is based.

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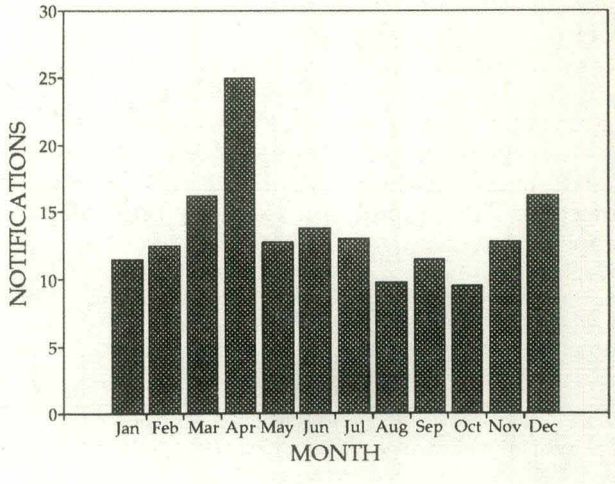
CDI editorial comment

In contrast to the spring seasonal peak apparent in Western Australia, legionellosis reports received by the National Notifiable Diseases Surveillance System (NNDSS) for Australia overall have peaked in autumn or early summer each year, and on average for the

Figure. Legionellosis notifications, Australia, average for 1991 to 1994, by month of onset



period 1991 to 1994 (Figure). Cases have, however, occurred throughout the year, probably reflecting different seasonal peaks in cases due to *L. pneumophila* (in summer and autumn) and *L. longbeachae* (for which spring peaks have been reported). The NNDSS will distinguish between cases caused by *L. pneumophila*, cases caused by *L. longbeachae* and cases due to other species, beginning in the near future.

There were 179 notifications of legionellosis in Australia in 1994, with a peak in onset in March-April. So far this year there have been 144, about the same as by this time last year, and equivalent to 193 for the whole year. One hundred and seven have been for males and 36 for females (the sex of one was not reported). Fifty-seven reports (40%) have been for persons over the age of 65 years and a further 44 cases (31%) have been reported for persons between 50 and 64 years.

A PROBABLE FOODBORNE OUTBREAK OF TOXOPLASMOSIS

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Introduction

Toxoplasma gondii is a ubiquitous intracellular protozoan parasite. Cats are the only known definitive hosts, but a wide range of animals and man are susceptible to infection as intermediate hosts[1,2].

Three life forms of *T. gondii* occur: the oocyst (which contains sporozoites and is the product of the sexual cycle in the small intestine of cats), the tachyzoite (the asexual invasive form), and the tissue cyst (which contains bradyzoites capable of persisting in tissues during the chronic or latent phase of the infection). Cats acquire infection by hunting wild mice and birds, from being fed raw meat or by the faecal-oral route from other cats. In other hosts, ingestion of oocyts results in initiation of an asexual cycle with the formation of tissue cysts. Ingestion of these tissue cysts by a wide range of mammals and birds leads to release of motile bodies (bradyzoites) by the action of digestive enzymes; thus initiating fresh asexual cycles and acute infection. Herbivores, including livestock, become infected by ingestion of grass and other foodstuffs that have been contaminated by cat faeces. Their tissue may remain infectious for life.

The two major routes of transmission to humans are oral and congenital. Ingestion of environmentally resistant oocysts shed from infected cats may contaminate hands, food or water and result in infection. Eating raw (uncooked or undercooked and unfrozen meat) which contains tissue cysts is the other common source of exposure. The incubation period was said to be 10 to 23 days in one outbreak associated with undercooked meat and 5 to 20 days in an outbreak associated with cats[3]. Person to person transmission is known only from mother to child *in utero*. Tachyzoites cause infection in the fetus during a primary maternal infection. These rapidly proliferating forms may also result in infection from contaminated blood transfusions.

It is not known what proportion of human *T. gondii* infection is due to eating raw or undercooked meat containing tissue cysts and what is due to oocysts on unwashed hands or vegetables. Estimates of the rate of infection of meat vary widely depending on the animal species and locality. The changes in food preparation and eating habits that have taken place throughout the world in the past few decades have increased the risk of human infection through consumption of undercooked meat. The impact of new game meats such as kangaroo is unknown.

T. gondii infection in children and adults is often asymptomatic or characterised by a mild influenza-like illness or lymphadenopathy, while congenital infection may lead to severe defects or death *in utero*. Infection is serious in immunocompromised hosts, such as transplant recipients and AIDS patients, in whom reactivation of latent infection may also occur.

This report details a probable foodborne outbreak of toxoplasmosis which involved 12 acute infections in adults and a case of congenital toxoplasmosis.

Index cases

The index case was a 31 year old woman diagnosed with acute toxoplasmosis after her baby was found to have chorioretinitis. At 34 weeks gestation an amniocentesis has been performed to investigate possible Rhesus incompatibility. One week later (in January 1995) she went into premature labour and delivered a live female infant who was jaundiced because of rhesus disease. Phototherapy was required for 10 days. No transfusions were administered.

At three months of age, delayed visual maturation was found but thought to be unrelated to an incidental finding of bilateral mature looking retinal scars clinically suggestive of congenital toxoplasmosis. Serological testing of the baby in May 1995 at 16.5 weeks revealed a positive IgG (index 8.902) and IgM (index 2.224) by EIA. The positive IgM was confirmed by an indirect immunofluoresence antibody technique (IFA) at a titre of 1:160. Serology on the mother at the same time was consistent with acute toxoplasmosis; both IgG (index 7.720) and IgM (index 2.136) were positive. IFA testing demonstrated a IgM titre of 1:160. Antenatal serum collected at 21 weeks was tested in parallel and showed no evidence of previous exposure to toxoplasmosis (IgG negative, index 0.075; IgM negative 0.779; IFA<20), confirming primary toxoplasmosis

Antenatal serum collected at 21 weeks was tested in parallel and showed no evidence of previous exposure to toxoplasmosis (IgG negative, index 0.075; IgM negative 0.779; IFA<20), confirming primary toxoplasmosis in the intervening period. The child was commenced on pyrimethamine and sulphamethoxazole together with twice weekly injections of folinic acid in June 1995 when she was five months of age. Treatment is anticipated to continue for one year.

Possible exposures were explored. The mother recalled eating undercooked kangaroo meat on 25 November 1994 when she was 28 weeks pregnant during a Christmas function at a suburban Brisbane restaurant. She was aware that another person who attended the restaurant had also been diagnosed with toxoplasmosis, in December 1994. In retrospect, she recalled lethargy and myalgias three weeks after the party. The index case worked as a preschool teacher but stopped work at 36 weeks gestation. The family did not own a cat but had a sandpit at home. In September 1994 they holidayed on an outback Queensland property but did not consume rare meat and drank boiled tank water. There were many feral cats about the property but direct contact was avoided.

The second case, of whom the first case had been aware, was a male aged 34 years who presented to his general practitioner nine days after the party with fever, nausea, myalgias and arthralgias. Liver function revealed a mild hepatic picture. Serological testing of paired sera on days 13 and 63 demonstrated seroconversion to toxoplasmosis. His wife who also attended the party was five weeks pregnant. Testing in a previous pregnancy had shown evidence of past exposure to toxoplasmosis.

Hypothesis

Related foodborne acquisition of toxoplasmosis for the two index cases was suspected. Its relative rareness suggested that even two cases acquired in the same time frame may have a possible epidemiological link. Hot foods served at the dinner party prior to their illness were spicy Thai chicken wantons, lamb satays, merguez sausage and marissa rolls, marinated eggplant, capsicum and fetta pizzas. Cold foods served were roasted eggplant dip with crudites and house made bread, smoked salmon and dill quiches, rare kangaroo medallions on crispy rissoto cakes and duck liver pate with naan bread.

The most likely 'risk food' was thought to have been the rare kangaroo medallions. Kangaroos are known to be an intermediate host for toxoplasmosis. Other meats (chicken, lamb and sausage) were thought to be less likely potential sources because they had been more thoroughly cooked. Whilst vegetables contaminated with oocysts may also have been a source, an environmental health inspection suggested hygiene practices in the restaurant were appropriate.

Methods

Approximately 60 people attended the function. Seven months afterwards a questionnaire was mailed to 46 attending persons asking about foods eaten and requesting a serum sample for *Toxoplasma* testing. Information was entered and collated using Epi Info v6.0. *Toxoplasma* IgG was tested using an in-house indirect EIA. IgM was tested using an indirect EIA (Gull Laboratories, Inc, Salt Lake City, Utah, USA). Various neighbours of the index case were also tested for serological evidence of acute toxoplasmosis.

Case definition

Evidence of acute infection was defined as:

a. Presence of *Toxoplasma* IgG and IgM on a single serum specimen, or

b. Evidence of seroconversion from negative to positive specific IgG for *Toxoplasma* on a retrieved and current serum specimen. Due to the timing of the blood collections in relation to the exposure time (up to seven months), the IgM may still be positive or have become negative. (Specific *Toxoplasma* IgM may remain detectable for many months after infection.)

Seroconversion provides the most reliable evidence for acute toxoplasmosis as there is the possibility of false positive IgM results with single specimens.

Those classified as not fulfilling the case definition included those with no previous exposure to *Toxoplasma* (*Toxoplasma* IgG and IgM negative) as well as those persons defined as having evidence of past exposure (*Toxoplasma* IgG positive without the presence of IgM, unless a stored serum specimen was available prior to the suspected exposure to enable seroconversion to be demonstrated).

This last group of patients may have been subject to misclassification because at the time of the study, those who experienced acute infection in December 1994 may have lost their IgM response. Alternatively, they may have acquired toxoplasmosis independently from alternative sources in the past. On a population basis one would expect approximately 30% of the cohort to be seropositive. A seroprevalence higher than this could have suggested that some patients have been misclassified as past rather than acute cases.

Results

Thirty-eight questionnaires were returned, a response rate of 83%, and all respondents also supplied a serum sample for testing. The mean age of the repondents was 38 years (range 24 to 61 years). Seventeen (45%) were female and 21 (55%) males.

Twelve adults in addition to the infant of the index case showed evidence of acute toxoplasmosis (Table 1). For six of these cases, acute seroconversion was demonstrated. (Retrospective sera were available for some subjects.) Of those that did not fulfill the case definition, 20 had no evidence of exposure. As a group,

Table 1. Serologically confirmed cases of acute toxoplasmosis

Case	Sex	Age (years)	Serology	Symptoms	Attended LMO[1]	Onset	Duration
1	M	35	Seroconversion	Asymptomatic	-	-	-
2	F	35	IgG+ IgM+	Asymptomatic	-	-	-
3	F	30	Seroconversion	Muscle pain, headache, lethargy, lymphadenopathy	-	16.12.94	3-4 weeks
3a[2]	F	0	IgG+ IgM+	Chorioretinitis			
4	M	26	IgG+ IgM+	Myalgia, headache, lethargy, sore throat, cervical lymphadenopathy, night sweats, fever	3.12.94 6.12.94	30.11.94	1 week
5	F	45	IgG+ IgM+	Myalgias, headache, tiredness, sore throat, cervical lymphadenopathy, fever, chills	-	3.12.94	4 weeks
6	M	52	Seroconversion	Myalgias, headache, lethargy, fever, chills, altered liver function	6.12.94	3.12.94	4 weeks
7	F	35	IgG+ IgM+	Asymptomatic	-	-	-
8	M	39	Seroconversion	Myalgias, lethargy, cervical and axillary lymphadenopathy, fever, night sweats	10.12.94	10.12.94	4 weeks
9	M	34	Seroconversion	Myalgias, cervical and axillary lymphadenopathy, fever, night sweats, altered liver function, atypical lymphocytes	4.12.94	4.12.94	2 weeks
10	M	25	IgG+ IgM+	Cervical lymphadenopathy	-	20.12.94	6-8 weeks
11	F	33	IgG+ IgM+	Asymptomatic	-	-	-
12	M	55	Seroconversion	Myalgias, headache, tiredness, rash, fever, night sweats, cough, altered liver function, atypical lymphocytes	1.12.94	28.11.94	2 weeks

1. LMO Local Medical Officer.
2. This patient was the congenitally affected five month old infant.

Table 2. Symptoms and signs documented for cases

	Cases	
	Number (n=12)	%
Fever	6	50
Night sweats	4	33
Myalgias	7	58
Headache	6	50
Lethargy	7	58
Sore throat	3	25
Rash	1	8
Lymphademopathy		
cervical	6	50
axillary	1	8
inguinal	1	8
Weight loss	1	8

therefore, at least 32 of 38 (84%) were susceptible to primary *Toxoplasma* infection prior to the party. The remaining six (16%) had detectable IgG only, suggesting past exposure. As at least seven months had elapsed prior to testing, those with evidence of past infection may also have acquired their infection in December 1994.

The incubation period was as short as three days with a mean of 11 days and range from three to 25 days.

Of the 12 cases that had evidence of acute toxoplasmosis, eight had associated symptoms and four had been asymptomatic. Five of the symptomatic patients had sought medical advice. The pregnant index case did not attend a doctor at the time, nor did the wife of one of the patients who sought medical attention who suffered symptoms very similiar to her husband. The remaining case noted persistent cervical lymphadenopathy without accompanying symptoms and was reassured by a medical colleague. The symptoms of the cases were varied and are summarised in Table 2.

Table 3. Attack rates for foods consumed at the cocktail party

	Persons who *ate* specified food				Persons who did *not* eat at specified food					Persons who were *uncertain* if they ate specified food		
Food item	Case	Non-case	Total	Attack rate (%)	Case	Non-case	Total	Attack rate (%)	*p* Value[1]	Case	Non-case	Total
Spicy chicken	10	17	27	37	1	0	1	100	0.39	1	9	10
Lamb satay	11	18	29	38	0	3	3	0	0.53	1	5	6
Sausage rolls	5	11	16	31	3	1	4	75	0.25	4	14	18
Eggplant pizza	9	19	28	32	1	0	1	100	0.34	2	7	9
Eggplant dip	5	10	15	33	2	5	7	29	1.00	5	11	16
Smoked salmon	9	16	25	36	1	4	5	20	0.64	2	6	8
Rare kangaroo	10	16	26	38	0	7	7	0	0.07	2	3	5
Duck liver	8	11	19	42	3	6	9	33	1.00	1	9	10

1. Fisher's exact test; 2-tailed.

Three patients had liver function tests performed at the time of acute illness. Alanine transaminase (ALT) and to a lesser extent, aspartate transaminase (AST) and lactate dehydrogenase (LDH) were elevated. Atypical lymphocytes (10–11%) were present in two patients. Duration of symptoms ranged from one to eight weeks.

Testing the other child of the index case as well as several neighbours revealed no previous exposure to *Toxoplasma*.

The attack rates for cases versus non-cases for the eight items on the menu are presented in Table 3. No statistically significant association could be demonstrated between the aquisition of toxoplasmosis and any of the foods ingested using the 2-tail Fisher's exact test.

Only one non-case described an illness with onset of symptoms three days after eating at the restaurant. He suffered fever, night sweats, myalgias, headache, tiredness and diarrhoea. The symptoms lasted two days and no further investigations were performed. Serological testing showed no exposure to *Toxoplasma*.

Confounding as a result of previous blood transfusions, ingestion of unpasteurized milk or raw eggs was not found. Three of the 12 cases (25%) were cat owners (no kittens) who fed their cat raw, canned and cooked meat. Seven of the 26 non-cases (27%) were cat owners. Nine of the cases were gardeners, five of whom wore gloves while gardening. None were vegetarian and all had consumed pork, lamb and beef in the previous 12 months. One or possibly two had consumed kangaroo in the preceding 12 months. Seven enjoyed their meat moderately to very well cooked, and five usually ate meat medium rare to very rare. All the cases, except one, usually washed their raw fruit and vegetables prior to ingestion and separated raw meat from other foods while cooking.

The majority of those that acquired toxoplasmosis did not usually mix socially or eat at common venues.

In addition to the index case and the woman who was five weeks pregnant, a third woman who was approximately 28 weeks pregnant attended the function and she also developed acute toxoplasmosis. Her infant was tested in May and July at two and 4.5 months and on both occasions had no demonstrable *Toxoplasma* IgG and IgM detected.

Discussion

The most important implication from this apparent outbreak was the risk posed to non-immune pregnant women and their unborn children. In a study from the Royal Women's Hospital (Melbourne) the seroprevalence of *Toxoplasma* antibodies in pregnant women was 45% on initial screening and the primary infection rate was 4 per 1000 births in the group studied[4]. Other data have shown that 60% of fetuses of infected mothers become infected[5]. The rate of congenital toxoplasmosis in the Melbourne population would therefore be 2-2.5 per 1000 births. If this is extrapolated to the whole of Australia (260,000 births per annum), there may be 520 to 650 infants born with congenital toxoplasmosis each year. Other studies have demonstrated lower rates of infection. In a recent Western Australian survey of pregnant women, 35% were seropositive on screening[6]. The rate of maternal infection in susceptible pregnancies was 1.6 per 1000 with a birth prevalence of congenital infection of 0.23 per 1000 births to non immune mothers. Studies in South Australia and Queensland have shown seroprevalences of 23 to 26% and in the Queensland study a congenital infection rate of 0.44 per 1000 non-immune pregnancies [7,8].

In Australia, neither serological screening nor patient education about *Toxoplasma* infectivity is routinely or systematically undertaken. The incidence of fetal infection relates to the stage of gestation at which a pregnant woman acquires the infection. Without treatment the incidence of congenital infection is approximately 10 to 15% for acquisition during the first trimester, 30% for the second trimester and 60% for the third trimester. The earlier in gestation transmission occurs, the greater the severity of infection in the fetus and newborn. Early maternal infection may result in death of the fetus *in utero* and spontaneous abortion. Almost all infected newborns of mothers who acquired the infection dur-

ing the third trimester are born without obvious signs of infection. Approximately 85% of infants with congenital infection appear normal at birth[5]. Without some form of screening, very few cases of congenital toxoplasmosis are recognized but, according to French studies, 85% will have long term sequelae including chorioretinitis or neurological damage[9].

In a recent study of 42 mothers of infants with congenital toxoplasmosis, 52% identified specific exposures to cat excrement; 52% had eaten raw or undercooked meat, and 16% had consumed raw eggs or unpasteurized milk. These data suggest that education can prevent or reduce the frequency of infection[5], however one study from Brussels reported a non-significant reduction of the rate of seroconversion in pregnant women routinely given a written list of recommendations[10].

Pregnant women should be advised on the modes of acquisition so they can take appropriate preventative measures. Ingestion of infected meat appears to be the main form of transmission in Australia. Rothe et al found that none of 115 stray cats examined shedded oocysts, agreeing with other studies that found a low prevalence of oocyst shedding in domestic cats[11]. These authors also reported one of 30 pork and none of 30 lamb chops contained viable cysts when the sensitivity of the detection method was one cyst per 100g of tissue[11]. As most pigs for domestic consumption in Australia are raised under clean conditions and without soil contact, infection should be unlikely. That none of the lamb chops showed detectable levels of cysts could have been due to the small sample size or to the fact that the samples were from animals probably less than nine months old which had not yet acquired infection. A survey on the prevalence of toxoplasmosis in Australian (Tasmanian) meat animals in 1975 showed the highest prevalence was in lambs (16.9%) and other sheep (61.7%), while the prevalence in vealers was 2.3% and in other cattle 0%. Pigs had an intermediate seroprevalence; cracker pigs 23.3% and other pigs 7.2%[12]. The prevalence of antibody to *Toxoplasma* in a more recent study of a sheep population of South Australia was 8%[13].

Whilst this study was unable to confirm the source of infection, the kangaroo meat would on theoretical grounds be the most likely source. A number of repondents paticularly recalled the kangaroo meat as being extremely rare and 'oozing with blood'.

Marsupials are highly susceptible to *T. gondii* infection because they evolved in the absence of this parasite until European settlement in Australia introduced the domestic cat, only 200 years ago[14]. Very severe clinical toxoplasmosis has been reported in wallabies and kangaroos[15], so many may die from the infection and leave only a small proportion of those surveyed as seropositive. In a study of 151 Bennett's wallabies (*Macropus rufogriseus rufogriseus*) and 85 Tasmanian pademelons (*Thylogayle billardierii*) which were tested to determine the prevalence of acute toxoplasmosis of macropods in the wild, 4% of the wallabies and 1.2% of the pademelons possessed *T. gondii* specific IgM in their sera[16].

The same workers demonstrated an overall seroprevalence of IgG to *Toxoplasma* in wild macropods of 8.5%[17]. There are probably significant differences in cat densities in various parts of the country, leading to differences in pasture contamination and exposure to infective oocysts.

Kangaroo meat served in restaurants, which is from field shot game, probably contains no more infective tissue cysts than other meats. The difference is that it is often presented for consumption very rare with the possibility that any tissue cysts may remain viable.

Whilst the disease is usually asymptomatic and without consequence in the immunocompetent adult, pregnant women should be alert to the risk to their unborn fetus if they ingest undercooked meat of any kind. Infected meat is rendered safe by adequate cooking (61°C for four minutes)[17]. Smoking and brine curing destroys bradyzoites in infected pork. Freezing at -21°C for 24 hours followed by thawing is reported to be effective; however conflicting results cast doubts on the reliability of this method. Other preventative measures should also be emphasized. Cutting boards and knives used for preparing meat and vegetables for human consumption should be thoroughly washed in hot water and detergent. Fruit and vegetables to be eaten raw should be thoroughly washed before consumption. Meats should not be eaten or tasted raw. Pet cats should not be fed uncooked meat scraps, especially pork. Cats should be fed on dry, canned or boiled food and discouraged from hunting (collar and bell) or scavenging; their faeces and litter should be disposed of daily before the development of infective sporozoites from oocysts, which takes up to five days depending on environmental conditions. Faeces should be disposed of carefully (burying or burning); litter pans should be disinfected daily by scalding with boiling water and gloves worn when handling faecal material. Outside sandpits should be covered when not in use. Hand washing is important after handling raw meat and possible contamination.

Acknowledgment

We wish to thank other diagnostic laboratories (Royal Brisbane Hospital and Queensland Medical Laboratory) for providing stored sera on request.

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RELATIONSHIP OF A DENGUE 2 ISOLATE FROM TOWNSVILLE, 1993, TO INTERNATIONAL ISOLATES

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Introduction

Between March 1992 and August 1993 there were over 700 cases of dengue fever notified from Townsville and the Charters Towers region of North Queensland[1,2]. Analysis of the epidemic pointed towards the suburb of South Townsville as the likely origin[3]. The next most recent epidemic occurred in 1981 and was caused by dengue 1[4], and there and have been small numbers of cases of this serotype reported as recently as 1991[5]. The 1992-93 epidemic was therefore most likely the result of the importation of a dengue 2 strain from overseas; it is of interest that a number of backpacker hostels are located in South Townsville. Importation and subsequent local transmission of dengue 2 has recently been demonstrated in Cairns[6] and highlights the risk of future epidemics from this source.

The molecular characterisation of dengue viral isolates provides a means to track more precisely the origins of epidemics and may provide insights that better enable control. Previous studies have compared sequences from 32[7] and 40[8] dengue 2 isolates creating an opportunity to relate dengue isolates to one another. We therefore undertook to sequence a dengue 2 isolate from the 1992-93 epidemic. The preliminary results are presented.

Methods

A dengue 2 viral isolate (TSV01) was obtained in April 1993 from a patient whose clinical illness was typical of dengue. The viral RNA genome was isolated and DNA encoding the 1485 base pair envelope (E) gene was obtained by reverse transcription and polymerase chain reaction (RT-PCR) using specific oligonucleotide primers. The PCR amplicon was cloned into a plasmid vector and the nucleotide sequence determined by the dideoxy chain termination method. Nucleotide sequences of two independent clones were obtained to identify any errors introduced by the PCR reaction.

The TSV01 sequence was compared with the sequence information on 35 other dengue 2 isolates from different geographical locations for which the complete E gene sequence is available and an additional 33 sequences where the last 111 bases of the E gene is known[7,8]. These data were obtained from the Division of Vectorborne Diseases at the Centers for Disease Control and Prevention in Atlanta, United States.

Results

The TSV01 gene sequence (data not shown) and the amino acid sequence deduced from it were compared with the nucleotide or aligned deduced amino acid sequences of the complete 35 E glycoprotein sequences.

Table. Nucleotide and deduced amino acid homology of the envelope gene of a 1993 Townsville isolate of dengue 2 and a selection of other dengue 2 isolates from elsewhere

Isolate	Nucleotide homology (%)	Amino acid homology (%)
Indonesia 1976	96	98
Seychelles 1977	95	98
Sri Lanka 1990	95	98
New Guinea 1944	94	97
Thailand 1980	93	97
Jamaica 1983	93	97
Brazil 1990	92	96
India 1957	91	96
Malaysia 1987	90	92

The nucleotide and amino acid sequence homology for nine isolates is shown in the Table. The most closely related isolate was from Indonesia in 1976 with 96% nucleotide homology. This compares with a more distantly related isolate from India, 1957 that has 91% homology.

Dengue 1, the next most closely related dengue serotype, has a nucleotide homology of 65% and a deduced amino acid homology of 68%.

A comparison of the last 111 bases of TSV01 with the shorter database which contained two Indonesian isolates from 1973 and 1978 also showed that these were the most closely related to TSV01.

On the basis of the phylogenetic study performed in 1993[7], five subgroups of dengue 2 have been described. The Townsville isolate belongs to subgroup IV and other isolates represented in this group include isolates from Somalia (1984), Seychelles (1977) and Sri Lanka (1982, 1985 and 1990) (data not shown).

Discussion

The exact origin of the dengue 2 strain imported into Australia in 1992 cannot be determined from this study. The comparison of the TSV01 nucleotide sequence and the amino acid sequence deduced from it indicates a close relationship with three Indonesian isolates from 1973, 1976 and 1978. This implies a possible origin of the TSV01 virus isolate, however a 14 year interval between viral isolations makes it possible that the strain may have arrived here via a third country. The nucleotide sequencing of a more recent Indonesian dengue 2 isolate would assist in the interpretation. A more rigorous statistical analysis of the sequence comparison of dengue 2 strains generated a phylogenic (genealogical) tree that confirms the relationships described here and this data will be presented elsewhere.

We believe that part of the investigation of a dengue epidemic should include the molecular characterisation of one or more representative isolates. The Division of Vectorborne Diseases at the Centers for Disease Control and Prevention have sequenced the E gene of a large number of flavivirus isolates (including dengue virus and Japanese encephalitis virus) and the databasee is available from them on request (GH Chang, personal communication).

There have been no reports of dengue infection in Townsville during the last two years. Further occurrences of dengue 2 infection in north Queensland will probably be caused by importation of new strains and comparison of new isolates with TSV01 will contribute to the basis upon which this will be established.

Acknowledgments

We wish to thank Townsville Mater Hospital and The Clive and Veera Ramaciotti Foundation for financial support for this project.

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OVERSEAS BRIEFS

In the last two weeks, the following information has been supplied by the World Health Organization (WHO), the South Pacific Epidemiological and Health Information Service and the Program for Monitoring Emerging Diseases.

Dengue in the Americas

This year, dengue activity has increased in several countries in the Americas and 16 countries have reported confirmed cases of dengue haemorrhagic fever (DHF)[1]. By 26 September 1995, Costa Rica, the Dominican Republic, El Salvador, Guatemala, Honduras, Nicaragua and Panama had reported 32,961 cases of dengue, of which 500 were DHF (10 deaths). In Mexico, the number of cases had reached 2586, compared with 373 in the same period last year, and there had been 45 cases of DHF (12 deaths). Brazil had reported 88,039 cases of dengue, including 105 cases of DHF, and Venezuela had reported 19,489 cases with 3568 cases of DHF and 18 deaths[2]. Two hundred and twenty-eight cases of dengue had also been reported from the English-speaking Caribbean.

Dengue 3 was recently detected in Nicaragua and Panama, and in early 1995, in Costa Rica, El Salvador and Honduras, representing the reappearance of this strain in the Americas after an absence of 16 years.

The resurgence of dengue and the emergence of DHF in the Americas is due in large part to setbacks in programs to eradicate the *Aedes aegypti* mosquito in the region; in 1995 its geographical distribution is similar that prior to the eradication campaigns of the 1950s and 1960s. Other contributors to the resurgence are dramatic shifts in populations, including increased urbanisation and settlement of new areas, population growth, more frequent international travel and increasing poverty.

Dengue in the Pacific update

The number of cases of dengue reported in the Pacific has declined recently, with recent activity reported only from New Caledonia and Palau. The South Pacific Epidemiological and Health Information Service warns, however, that the rainy season is already approaching in some of the Pacific Island countries and there is still risk of dengue in the region.

Meningococcal meningitis in Mozambique

The outbreak of meningococcal meningitis in Mozambique had included 1416 suspected cases and 118 deaths by 30 September, by which time the outbreak was waning. Vaccination had been carried out in prisons and military centres as well as in schools where cases have been reported, and rifampicin chemoprophylaxis has been used for contacts of the patients.

Plague in Madagascar

The outbreak of bubonic plague in Mahajanga Province in Madagascar had included 320 cases (23 confirmed) and nine deaths by 30 September. The number of cases occurring had stabilised and the situation appeared to be under control. Measures such as the management of patients, chemoprophylaxis of patients, control of vectors through the use of insecticide and the destruction of rats will continue in order to prevent further spread.

Venezuelan equine encephalitis in Colombia and Venezuela

Colombia and Venezuela have reported have reported an epidemic of Venezuelan equine encephalitis (VEE) in the border area between the two countries which began in early September[3]. As of 21 September 1995, 8825 human cases had been reported (54 laboratory confirmed) and there had been four deaths, mainly in State of Zulia in Venezuela. In Colombia, more than 250 cases had been reported in the Department of La Guajira.

The VEE virus in the most severe of the several viruses causing disease in horses, and can be transmitted to humans by mosquito bites. Most infections are relatively mild, and symptoms include abrupt onset of severe headache, chills, fever, muscular pain, nausea and vomiting. Clinical cases occur in 11% to 20% of the exposed population and death occurs in less than 1% of those cases.

The Pan American Health Organization is working closely with the two countries to support their efforts to control the outbreak. The efforts include collecting data, mass immunisation of horses, laboratory confirmation of clinical diagnoses, provision of essential medicines and vaccines for laboratory workers, and community education on prevention and control measures (such as using mosquito repellents and bednets and avoiding forested areas at dawn and twilight). These efforts have resulted in a sharp decline in the number of human cases which are being registered daily.

Large scale epidemics of VEE occurred in Colombia, Peru, Trinidad and Venezuela in the 1950s and Central America and Mexico in the late 1960s, reaching Texas in the United States in 1971. Outbreaks can be prevented by the regular immunisation of horses as part of public health programs.

Cholera update

Mexico has reported 8189 cholera cases and 70 deaths for the period 19 May to 8 September. In **Cote D'Ivoire**, Guiglo District in the Department de l'Ouest has been declared infected. An outbreak began there

on 18 September and had included 174 cases and seven deaths by 6 October.

Cholera cases have been reported since May from Afghanistan, Burkino Faso, Burundi, Cameroon, Cape Verde, China, Costa Rica, Cote d'Ivoire, Ecuador, El Salvador, Ghana, Guinea, India, Japan, Laos, Liberia, Mali, Mexico, Moldova, Nigeria, the Russian Federation, Romania, Sierra Leone, Singapore, Tanzania, Togo, Uganda, Ukraine, Vietnam and Zaire.

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COMMUNICABLE DISEASES SURVEILLANCE

Virology and Serology Reporting Scheme

There were 1295 reports received in the *CDI* Virology and Serology Reporting Scheme this fortnight (Tables 8, 9 and 10).

- Four reports of **measles** were received this period. The number of reports received remains low compared to the same period last year (Figure 1).
- **Rubella** was reported for 12 patients this period including 2 females of childbearing age. To the end of August the number of reports received remained below that received for a similar period over the last 2 years (Figure 2).
- **Hepatitis A** was reported for 9 patients this period including 3 males and 6 females.
- Positive **hepatitis B** serology was reported for 60 patients this fortnight, including 25 males and 33 females. A total of 53 were in the 15 to 44 year age range. Included were 3 injecting drug users, 9 pregnant females and 2 prisoners.
- Two hundred and four reports of positive **hepatitis C** serology were received this period. Included were 16 injecting drug users, 3 pregnant females and 2 index cases involved in needlestick injuries.
- No reports of **Ross river virus** were received this period.
- **Kunjin virus** was reported for a 42 year old female from Western Australia.
- **Untyped dengue** was reported for a 32 year old Western Australian male.
- Thirty-six reports of **adenovirus** were received this period diagnosed by virus isolation (29), antigen detection (6) and single high titre (one).
- **Herpes simplex virus type 1** was reported for 191 patients this fortnight. Diagnosis was by virus isolation (188) and antigen detection (3).
- **Untyped herpes simplex virus** was isolated from the aqueous humour of a 48 year old female with recurrent uveitis.
- One hundred and eighty eight reports of **herpes simplex virus type 2** were received, 187 diagnosed by virus isolation and one by antigen detection.

Figure 1. Measles laboratory reports, 1994 and 1995, by month of specimen collection

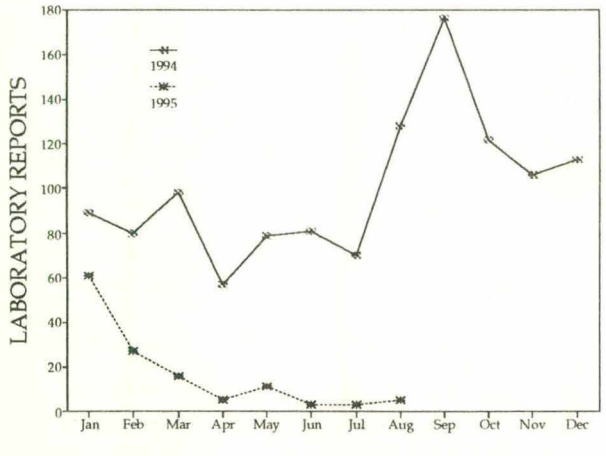


Figure 2. Rubella laboratory reports, 1993 to 1995, by month of specimen collection

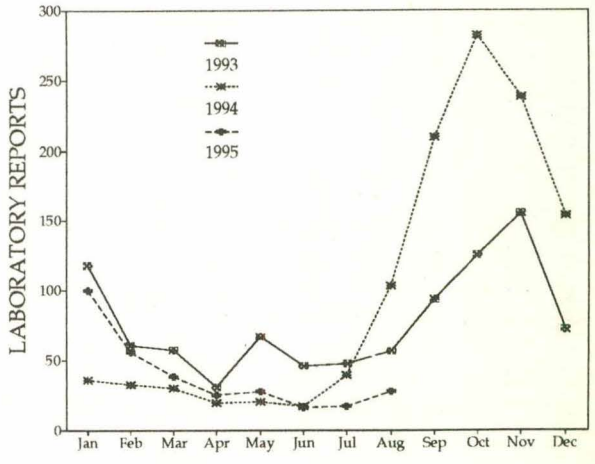


Figure 3. Influenza A laboratory reports, 1994 and 1995, by month of specimen collection

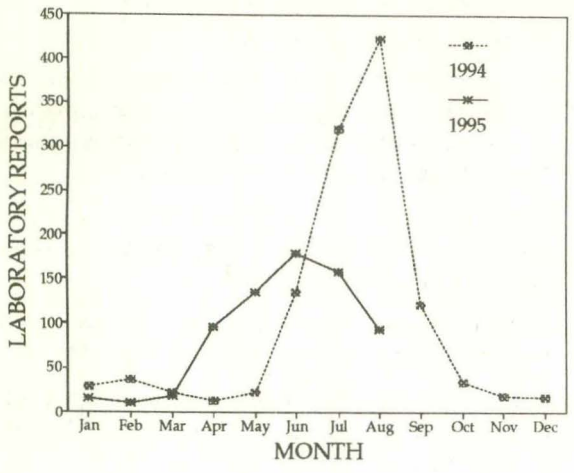


Figure 4. Parainfluenza virus type 3 laboratory reports, 1994 to 1995, by month of specimen collection

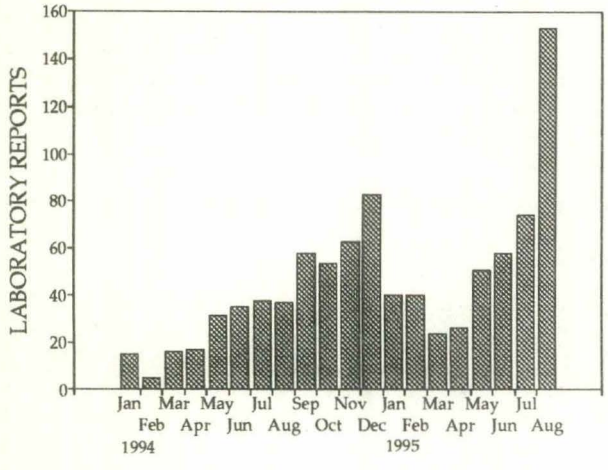
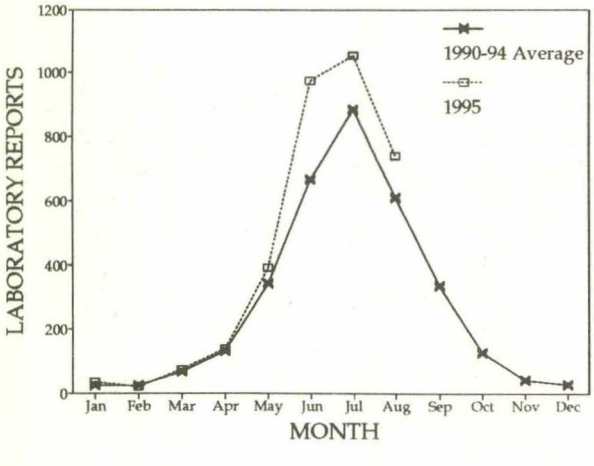


Figure 5. Respiratory syncytial virus laboratory reports, 1990 to 1994 average and 1995, by month of specimen collection



- Fifty two reports of **cytomegalovirus** were received this period. Diagnosis was by virus isolation (45), antigen detection (2) and serology (5). Included was one transplant recipient and one patient with a malignancy.
- **Varicella-zoster virus** was reported for 24 patients this period. Diagnosis was by virus isolation (14), antigen detection (7) and IgM detection (3).
- Nine **echovirus** reports were received this period including 2 reports of **echovirus type 9**, one of which was in association with meningitis (virus isolation from CSF). Five reports of echovirus type 9 were received for the month of August, more than for any month since March 1993.
- **Untyped enterovirus** was reported in association with myocarditis for a 2 month old male.
- **Rhinovirus** was reported for 28 patients this period, 21 of whom were under the age of 5 years.
- **Influenza A** was reported for 10 patients this fortnight. Diagnosis was by virus isolation (4), antigen detection (one) and single high titre (5). Reports were received from the Northern Territory (3) and Victoria (7). Included was a 73 year old male with a diagnosis of atypical pneumonia who died. A total of 718 reports has been received for the year to date. Ninety two isolates were identified as being H_1N_1 subtypes and 9 as H_3N_2 subtypes. The number of reports received fell markedly in August (Figure 3)
- Twenty reports of **influenza B** were received this fortnight. Diagnosis was by virus isolation (13), antigen detection (3), single high titre (3) and IgM detection (one). Reports were received from New South Wales (4), Queensland (5), Victoria (10) and Western Australia (one). A total of 297 reports has been received so far this year for 152 males and 142 females. The number of reports received remained high in August.
- **Parainfluenza virus type 3** was reported for 45 patients this fortnight, 33 of whom were under the age of one year and a total of 41 under 5 years of age. Diagnosis was by virus isolation (33) and antigen detection (12). The number of reports continues to rise (Figure 4).
- One hundred and twenty-two reports of **respiratory syncytial virus (RSV)** were received this fortnight. Method of diagnosis included virus isolation (56), antigen detection (62), single high titre (3) and fourfold rise in titre (one). The number of reports has continued to rise in recent weeks and is above average for the time of year (Figure 5).
- **Rotavirus** was reported for 78 patients this period including 50 males and 28 females. Seventy cases (90%) were 4 years of age or under. The number of reports is average for the time of year (Figure 6).
- ***Chlamydia trachomatis*** was reported for 72 patients this period diagnosed by isolation (17), antigen detection (9) and nucleic acid detection

Figure 6. Rotavirus laboratory reports, 1990 to 1994 average and 1995, by month of specimen collection

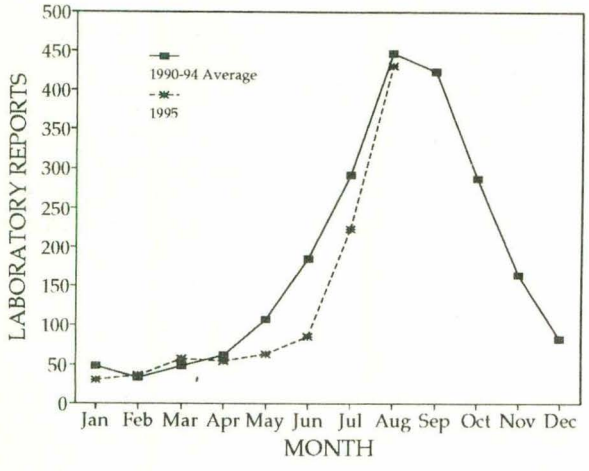
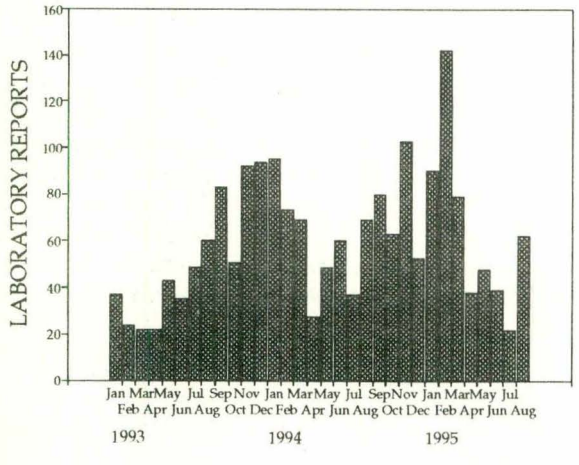


Figure 7. *Bordetella* laboratory reports, 1993 to 1995, by month of specimen collection



(46). Included were 22 males and 50 females 65 of whom were aged 15 to 44 years.

- Seventeen reports of ***Bordetella pertussis*** were received this period. The number of reports has risen in recent weeks (Figure 7).
- Four reports of ***Mycoplasma pneumoniae*** were received this period for 3 males and one female all in the one to 44 year age group. The number of reports has remained stable in recent months.
- ***Rickettsia australis*** was reported for a 43 year old female from the Northern Territory with a diagnosis of chronic fatigue syndrome.
- Positive ***Brucella*** serology was reported for a 59 year old male who worked in a slaughterhouse.
- Nine reports of ***Schistosoma*** species were received this period including four males with a documented history of travel to Africa.

Australian Sentinel Practice Research Network

Data for week 38 (ending 24 September) and week 39 (ending 1 October) are included in this issue of *CDI* (Table 1). There were 7956 consultations reported for week 38 and 6121 for week 39. The influenza reporting rate fell markedly this fortnight. The rate of reporting of rubella was higher than it has been in recent months and the gastroenteritis reporting rates continued to be slightly higher than usual.

HIV and AIDS Surveillance

Methodological note

National surveillance for HIV disease is coordinated by the National Centre in HIV Epidemiology and Clinical Research (NCHECR), in collaboration with State and Territory health authorities and the Commonwealth of Australia. Cases of HIV infection are notified to the National HIV Database on the first occasion of diagnosis in Australia, by either the diagnosing laboratory (ACT, New South Wales, Tasmania, Victoria) or by a combination of laboratory and doctor sources (Northern Territory, Queensland, South Australia, Western Australia). Cases of AIDS are notified through the State

Table 1. Australian Sentinel Practice Research Network, weeks 38 and 39, 1995

Condition	Week 38, to 24 September 1995		Week 39, to 1 October 1995	
	Reports	Rate per 1000 encounters	Reports	Rate per 1000 encounters
Influenza	70	8.8	88	14.4
Rubella	11	1.4	4	0.7
Measles	1	0.1	0	0
Chickenpox	4	0.5	18	2.9
Pertussis	7	0.9	4	0.7
Gastroenteritis	104	13.1	172	28.1

Table 2. New diagnoses of HIV infection, new diagnoses of AIDS and deaths following AIDS occurring in the period 1 to 30 April 1995, by sex and State or Territory of diagnosis

										TOTALS FOR AUSTRALIA			
		ACT	NSW	NT	Qld	SA	Tas	Vic	WA	This period 1995	This period 1994	Year to date 1995	Year to date 1994
HIV diagnoses	Female	0	1	0	0	0	0	1	3	5	7	26	30
	Male	1	34	1	12	1	0	14	4	67	70	286	305
	Sex not reported	0	2	0	0	0	0	0	0	2	2	8	5
	Total[1]	1	37	1	12	1	0	15	7	74	79	321	340
AIDS diagnoses	Female	0	1	0	0	0	0	0	0	1	4	6	10
	Male	0	11	0	1	1	0	14	2	29	70	138	272
	Total[1]	0	12	0	1	1	0	14	2	30	74	145	284
AIDS deaths	Female	0	2	0	0	0	0	0	0	2	5	11	12
	Male	0	15	1	6	1	0	11	1	35	63	171	226
	Total[1]	0	17	1	6	1	0	11	1	37	68	182	239

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

Table 3. Cumulative diagnoses of HIV infection, AIDS and deaths following AIDS since the introduction of HIV antibody testing to 30 April 1995, by sex and State or Territory of diagnosis

		ACT	NSW	NT	Qld	SA	Tas	Vic	WA	AUSTRALIA
HIV diagnoses	Female	13	539	4	88	44	4	155	63	910
	Male	155	9747	79	1494	541	69	3230	709	16024
	Sex not reported	0	2052	0	0	0	0	43	0	2095
	Total[1]	168	12346	83	1586	585	73	3435	773	19049
AIDS diagnoses	Female	3	121	0	24	15	2	40	13	218
	Male	68	3329	24	540	247	32	1207	244	5691
	Total[1]	71	3460	24	566	262	34	1254	258	5929
AIDS deaths	Female	2	84	0	18	11	2	21	8	146
	Male	46	2351	18	383	156	21	930	174	4079
	Total[1]	48	2441	18	403	167	23	957	182	4239

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

and Territory health authorities to the National AIDS Registry. Diagnoses of both HIV infection and AIDS are notified with the person's date of birth and name code, to minimise duplicate notifications while maintaining confidentiality.

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

HIV and AIDS diagnoses and deaths following AIDS reported for April 1995, and cumulative to 30 April 1995, as reported to 31 July 1995, are included in this issue of *CDI* (Tables 2 and 3).

National Influenza Surveillance 1995

Australian Capital Territory Department of Health and Community Care; Australian Sentinel Practice Research Network; Communicable Diseases Intelligence Virology and Serology Reporting Scheme Contributing Laboratories; New South Wales Department of Health; Australia Post; Victorian Department of Health and Community Services; South Australian Health Commision; World Health Organization (WHO) Collaborating Centre for Influenza Reference and Research, Melbourne

This is the last National Influenza Surveillance report for 1995. The Scheme will re-commence for winter 1996.

Overall the rate of influenza reporting has continued to decline to low levels this fortnight.

Sentinel general practitioner surveillance (Figure 8)

- The **Australian Sentinel Practice Research Network** has reported an overall reduction in the rate of reporting of influenza like illness over the past month. For the weeks ending 24 September and 1 October consultation rates of 9 and 14 per 1000 encounters were reported respectively.

- **New South Wales** sentinel general practitioners reported rates of 14 and 13 per 1000 consultations for the weeks ending 17 and 24 September respectively, similar to the rates reported in previous weeks.

Absenteeism surveillance (Figure 9)

- **Australia Post** reported a national absenteeism rate of 2.4% and 2.2% for the weeks ending 1 and 8 October respectively, a reduction on the rates reported in previous weeks. The absenteeism rate fell in all States and Territories other than South Australia where it remained stable.
- **New South Wales Schools Absenteeism Surveillance** reported rates of 8.3% and 4.5% for the weeks ending 17 and 24 September respectively.

Laboratory surveillance

- **Influenza A** was reported for 10 patients this fortnight. Diagnosis was by virus isolation (4), antigen detection (one) and single high titre (5). Reports were received from the Northern Territory (3) and Victoria (7). Included was a 73 year old male with a diagnosis of atypical pneumonia who died. A total of 718 reports has been received for the year to date. Ninety-two isolates were identified as being H_1N_1 subtypes and 9 as H_3N_2 subtypes. The number of reports received fell markedly in August (Figure 10).
- Twenty reports of **influenza B** were received this fortnight. Diagnosis was by virus isolation (13), antigen detection (3), single high titre (3) and IgM detection (one). Reports were received from New

Figure 8. Sentinel general practitioner influenza reports per 1000 encounters, 1995, by week

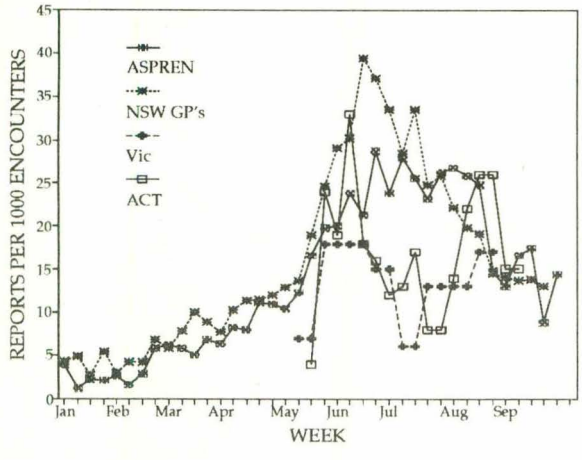


Figure 9. Absenteeism reports, 1995, by week and scheme

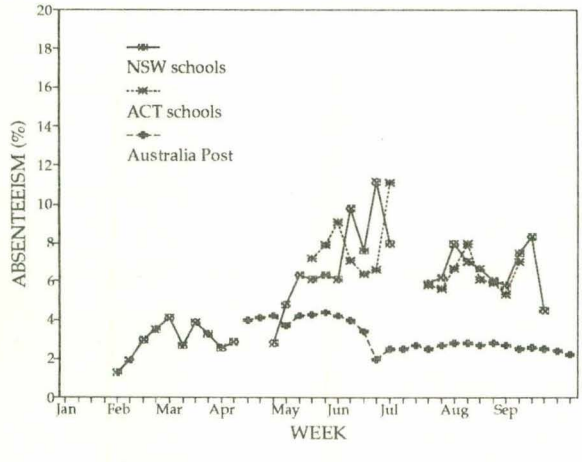


Figure 10. Influenza A laboratory reports, 1995, by method of diagnosis and week of specimen collection

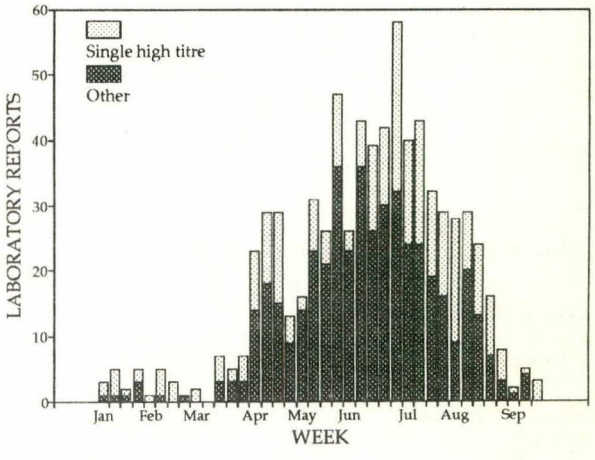
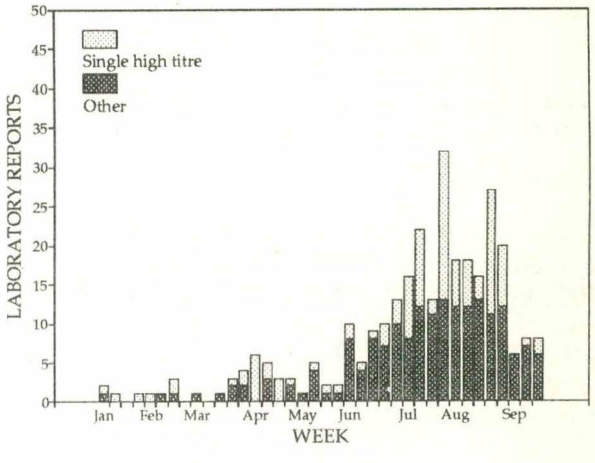


Figure 11. Influenza B laboratory reports, 1995, by method of diagnosis and week of specimen collection



South Wales (4), Queensland (5), Victoria (10) and Western Australia (one). A total of 297 reports has been received so far this year for 152 males and 142 females. The number of reports received remained high in August (Figure 11).

Deaths surveillance

- **South Australian Deaths Surveillance** reported death rates of 1.7, 1.7 and 1.2 per 10,000 population for the weeks ending 22 and 29 September and 6 October respectively. The death rate has remained stable in recent weeks.

Surveillance of Serious Adverse Events Followng Vaccination

The Serious Adverse Events Following Vaccination Surveillance Scheme is a national surveillance scheme which monitors the serious adverse events which occur rarely following vaccination. More details on the Scheme were published in *CDI* 1995; **19**: 273-274.

Acceptance of a report does not imply a causal relationship between the 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 to Australian children under the age of 6 years every month.

Results for the reporting period 3 to 30 September 1995

There were 14 reports of serious adverse events following vaccination for the reporting period 3 September to 30 September 1995. Reports were for episodes which occurred between April and September 1995. They were received from the Northern Territory (2), Queensland (2), South Australia (4) and Western Australia (6).

Of the 14 reports, 5 cases were of persistent screaming, 4 of hypotonic/hyporesponsive episodes, one of convulsions, one of encephalopathy and 3 were other events temporally associated with vaccination (Table 4). Parainfluenza virus type 3 was isolated from the child with encephalopathy. Of the 3 'other' cases, one had an apnoea following DTP, OPV and Hib vaccination, one had hives, diarrhoea and screaming following DTP vaccination and one was a child who developed lymphadenopathy 10 days after receiving MMR vaccine.

Events associated with DTP alone or DTP in combination with other vaccines were associated with the first (4), second (5), fourth (3) or fifth (one) dose. Five children were hospitalised: one with convulsions, two with hypotonic/hyporesponsive episodes, one with encephalopathy and one following an apnoea. Twelve children were fully recovered at the time the initial report was sent in, the child who had an encephalopathy had not fully recovered and the status of one child was unknown.

National Notifiable Diseases Surveillance System, 17 to 30 September 1995

There were 1328 notifications received for the period (Tables 5, 6 and 7, and Figure 13). No notifications were received from Queensland and the notifications received from Tasmania are for only a part of the current reporting period.

- There were 5 notifications of **Ross River virus infection**; 3 cases were male and 2 were female. Cases were in age groups from 25-29 to 65-69 years. Two cases were reported from each of New South Wales and Western Australia, and one case from the Northern Territory. Dates of onset were reported as August (2 cases) and September (3 cases).
- There were 311 notifications of **campylobacteriosis**; 180 cases were male and 131 cases were female. Cases were reported from all age groups, with 23% of cases being aged less than 5 years.
- There were 42 notifications of **gonococcal infection** received; 36 cases were male and 6 cases were female. Of the total, 9 cases were reported from Victoria, 13 cases from Western Australia, 6 from the Northern Territory and 7 from New South Wales. Recorded ages were from most age groups in the range from 15-19 years to 50-54 years, with one case in the 65-69 years age group; 55% of the cases were aged between 15 and 29 years.
- One case of ***Haemophilus influenzae* type b infection** was reported during the period. The patient was a male aged one year reported from New South Wales.

Table 4. Adverse events following vaccination for the period 3 to 30 September 1995

Event	Vaccine				Reporting States or Territories	Total reports for this period
	DTP	DTP/OPV	DTP/OPV/Hib	MMR		
Persistent screaming	5				NT, Qld, SA, WA	5
Hypotonic/ hyporesponsive episode	1	1	2		Qld, SA, WA	4
Convulsions	1				WA	1
Encephalopathy			1		WA	1
Other			2	1	NT, WA	3
Total	7	1	5	1		14

- Twenty cases of **hepatitis A** were reported; 12 cases were male and 8 cases were female. The cases were from most of the age groups 5-9 years to 40-44 years, with one case in the 80-84 years age group.
- Nine cases of **hepatitis B** were reported; 3 were males and 6 were females. All age-groups from 15-19 years to 40-44 years were represented.
- Two notifications of **legionellosis** were received. Both cases were male, one from New South Wales and one from Western Australia. Their ages were recorded as being from the 55-59 and 75-79 years age groups.
- One case of **leprosy** was reported from New South Wales.
- Two cases of **listeriosis** were reported, one male in the age group 70-74 years, and one female in the age group 55-59 years. Both cases were reported from Victoria.
- There were 5 notifications of **malaria** received; 3 cases were male and 2 cases were female. Recorded ages were between 10 and 49 years. Reported onset dates were in August (3 cases) and September (2 cases). The ages of cases ranged fromless than one to 60 years.
- Twenty-four cases of **measles** were reported; 10 cases were male and 14 cases were female. All cases were aged between 0 and 25 years, with 9 cases reported for children aged less than two years. There was one apparent cluster of 4 cases in the same postcode area in New South Wales.
- There were 10 cases of **meningococcal infection** reported; 4 cases were male and 6 cases were female. The cases were aged up to 55 years, with 5 cases in the age group 0-4 years and 2 in the age group 15-19 years. There were 3 apparent clusters of 2 cases each in the same postcode areas, 2 in New South Wales and one in South Australia.
- There were 103 notifications of **pertussis**; 47 cases were male and 56 cases were female. All age-groups between 0-4 years and 65-69 years were represented, with one case from the 75-79 years age group. Twelve cases were aged less than one year, and 7 were in the 1-4 year age group. There were 17 apparent clusters of between 2 and 12 cases each in the same postcode area. Apparent clusters were in New South Wales (9), Victoria (3), South Australia (4) and Western Australia (one).
- Nine notifications of **Q fever** were received; all cases but one were male. Recorded age groups of cases were from 15-19 to 50-54 years.
- There were 165 cases of **rubella** reported; 111 cases were male, 52 cases were female, and the sex of 2 cases was not recorded. Recorded ages of cases were from all age groups between 0-4 and 50-54 years. Fourteen cases were reported for females in the age range from 15 to 44 years. Half of the cases (82) were reported in males 10-24 years of age.
- There were 79 cases of **salmonellosis** reported; 38 cases were male, 37 cases were female, and the sex of 4 cases was not recorded. The cases were from all age groups 0-4 years to 55-59 years, with 2 cases in older age groups; 48% of the cases were aged less than 5 years.
- Thirty-seven cases of **syphilis** were reported; 17 cases were male and 20 cases were female. One case was aged under one year; the other cases were from all age groups between 15-19 and 80-84 years. The highest notification rates have been for persons between 15 and 29 years over the last 5 years (Figure 12).
- There were 29 cases of **tuberculosis** reported; 15 cases were male and 14 cases were female. One case was reported in a female from the age group 0-4 years. All other age groups betwen 15-19 years and 75-79 years were represented. The dates of onset were reported as being in February (one), June (one), July (4), August (6) and September (17).
- Two cases of **yersiniosis** were reported, one male and one female. The age groups of the cases were reported as 20-24 and 50-54 years.

Figure 12. Average annual syphilis notification rate per 100,000 population, 1991 to 1995, by age age group and sex

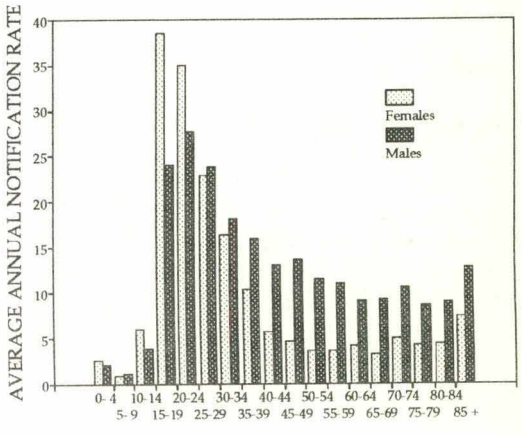
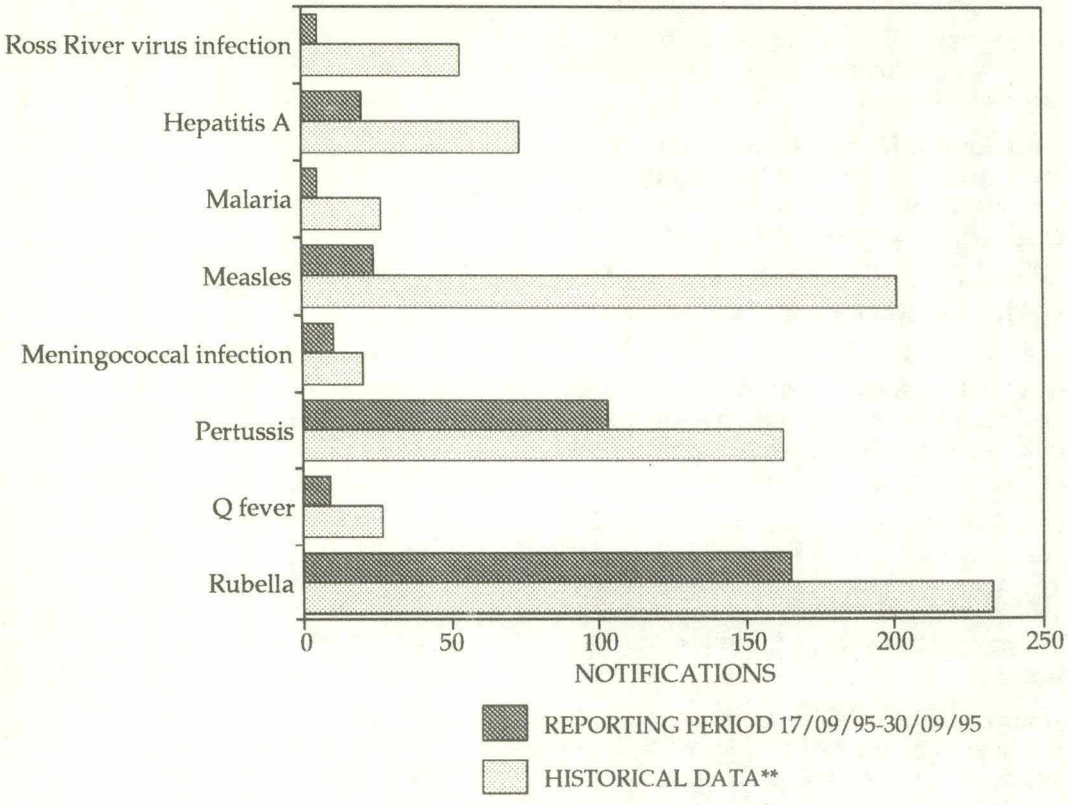


Figure 13. Selected National Notifiable Diseases Surveillance System reports, and historical data[1]



1. The historical data are the averages of the number of notifications in 9 previous 2-week reporting periods: the corresponding periods of the last 3 years and the periods immediately preceding and following those.

Table 5. Notifications of diseases preventable by vaccines recommended by the NHMRC for routine childhood immunisation, received by State and Territory health authorities in the period 17 to 30 September 1995

DISEASES	ACT	NSW	NT	Qld	SA	Tas	Vic	WA	TOTALS FOR AUSTRALIA[1] This period 1995	This period 1994	Year to date 1995	Year to date 1994
Diphtheria	0	0	0		0	0	0	0	0	0	0	0
Haemophilus influenzae b infection	0	1	0		0	0	0	0	1	3	55	142
Measles	0	12	0		0	1	8	3	24	262	1055	3242
Mumps	1	1	1	NN	0	0	0	0	3	1	50	19
Pertussis	0	56	1		27	0	15	4	103	278	3043	3998
Poliomyelitis	0	0	0		0	0	0	0	0	0	0	0
Rubella	14	35	0		4	4	75	33	165	213	1927	1469
Tetanus	0	0	0		0	0	0	0	0	0	3	10

1. Totals comprise data from all States and Territories. Cumulative figures are subject to retrospective revision, so there may be discrepancies between the number of new notifications and the increment in the cumulative figure from the previous period.

NN Not Notifiable.

Table 6. Notifications of other diseases[1] received by State and Territory health authorities in the period 17 to 30 September 1995

DISEASES	ACT	NSW	NT	Qld	SA	Tas	Vic	WA	TOTALS FOR AUSTRALIA[2] This period 1995	This period 1994	Year to date 1995	Year to date 1994
Arbovirus infection												
Ross River virus infection	0	2	1		0	-	0	2	5	25	2290	3762
Dengue	0	0	0		0	-	0	0	0	0	21	15
NEC[3]	0	3	0		0	0	1	0	4	13	736	467
Campylobacteriosis[4]	13	-	7		126	8	73	84	311	356	7726	7052
Chlamydial infection (NEC)[5]	5	NN	1		11	2	45	27	91	230	4441	5592
Donovanosis	0	NN	1		NN	0	0	0	1	7	59	79
Gonococcal infection[6]	0	7	6		7	0	9	13	42	106	2259	2281
Hepatitis A	0	5	1		1	0	9	4	20	58	1066	1484
Hepatitis B	0	2	0		1	0	6	0	9	11	258	254
Hepatitis C incident	-	0	0		0	-	-	-	0	2	81	25
Hepatitis C unspecified	25		0			1	233	71	330	310	6927	6780
Hepatitis (NEC)	0	0	0		0	0	0	NN	0	1	31	33
Legionellosis	0	1	0		0	0	0	1	2	7	144	140
Leptospirosis	0	0	0		0	0	0	0	0	1	96	91
Listeriosis	0	0	0		0	0	2	0	2	0	49	20
Malaria	0	1	2		0	0	2	0	5	38	470	573
Meningococcal infection	0	0	0		2	0	6	2	10	12	278	284
Ornithosis	0	NN	0		0	0	4	0	4	2	98	62
Q fever	0	8	0		0	0	1	0	9	16	344	510
Salmonellosis (NEC)	3	33	5		8	2	17	11	79	152	4677	4086
Shigellosis[4]	1	-	1		3	0	4	1	10	30	600	563
Syphilis	1	20	3		1	0	10	2	37	132	1535	1981
Tuberculosis	1	11	0		2	0	15	0	29	39	813	761
Typhoid[7]	0	0	0		0	0	0	0	0	3	32	35
Yersiniosis (NEC)[4]	0	-	0		2	0	0	0	2	17	248	321

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

NN Not Notifiable.

NEC Not Elsewhere Classified.

\- Elsewhere Classified.

Table 7. Notifications of rare[1] diseases received by State and Territory health authorities in the period 17 to 30 September 1995

DISEASES	Total this period	Reporting States or Territories	Year to date 1995
Botulism	0		0
Brucellosis	0		20
Chancroid	0		2
Cholera	0		5
Hydatid infection	0		28
Leprosy	1	NSW	6
Lymphogranuloma venereum	0		1
Plague	0		0
Rabies	0		0
Yellow fever	0		0
Other viral haemorrhagic fevers	0		0

1. Fewer than 60 cases of each of these diseases were notified each year during the period 1988 to 1994.

Table 8. Virology and serology laboratory reports by State or Territory[1] for the reporting period from 21 September to 4 October 1995, historical data[2], and total reports for the year, continued

	State or Territory[1]							Total this fortnight	Historical data[2]	Total reported this year
	ACT	NSW	NT	Qld	Tas	Vic	WA			
MEASLES, MUMPS, RUBELLA										
Measles virus						4		4	41.8	268
Mumps virus						3	1	4	3.3	60
Rubella virus						6	6	12	59.5	581
HEPATITIS VIRUSES										
Hepatitis A virus			1				8	9	12.8	350
Hepatitis B virus		15	1	4		19	21	60	86.8	1,861
Hepatitis C virus		19	14		11	11	149	204	223.3	4,580
Hepatitis D virus				1				1	1.2	14
ARBOVIRUSES										
Barmah Forest virus			1				1	2	4.7	201
Dengue not typed							1	1	.8	15
Kunjin virus							1	1	.2	4
Flavivirus (unspecified)						1		1	2.2	33
ADENOVIRUSES										
Adenovirus type 1						1		1	2.8	31
Adenovirus type 2						1		1	2.0	23
Adenovirus type 3						1		1	5.2	49
Adenovirus type 7		1						1	.5	18
Adenovirus not typed/pending		4		16	1	6	5	32	52.8	703
HERPES VIRUSES										
Herpes simplex virus type 1		10	6	26	3	72	74	191	152.5	3,833
Herpes simplex virus type 2		30	6	17	1	40	94	188	184.7	4,008
Herpes simplex not typed/pending						1	4	5	22.5	367
Cytomegalovirus		5		20	1	13	13	52	62.7	1,172
Varicella-zoster virus		2		1		10	11	24	36.7	835
Epstein-Barr virus		2				7	8	17	50.7	1,460
OTHER DNA VIRUSES										
Papovavirus group						3		3	.2	10
Parvovirus							2	2	3.3	96
PICORNA VIRUS FAMILY										
Echovirus type 3						1		1	.0	22
Echovirus type 9						2		2	.2	14
Echovirus type 11						1		1	3.2	4
Echovirus type 14		1						1	.2	5
Echovirus type 22		1						1	.0	7
Echovirus type 25		1						1	.0	1
Echovirus not typed/pending							2	2	.0	5
Poliovirus type 2 (uncharacterised)		2						2	.5	6
Rhinovirus (all types)		2		8		18		28	45.0	532
Enterovirus not typed/pending		4		8		3	13	28	45.7	722

Table 8. Virology and serology laboratory reports by State or Territory[1] for the reporting period from 21 September to 4 October 1995, historical data[2], and total reports for the year, continued

	State or Territory[1]							Total this fortnight	Historical data[2]	Total reported this year
	ACT	NSW	NT	Qld	Tas	Vic	WA			
ORTHO/PARAMYXOVIRUSES										
Influenza A virus				3		6		9	59.0	643
Influenza A virus H_3N_2						1		1	2.8	9
Influenza B virus		4		5		10	1	20	36.7	301
Parainfluenza virus type 2		1		1				2	2.2	172
Parainfluenza virus type 3		7		24		11	3	45	28.3	631
Parainfluenza virus type 4				1				1	.0	1
Parainfluenza virus typing pending					3			3	1.7	35
Respiratory syncytial virus		14		33	21	17	37	122	150.5	3,659
OTHER RNA VIRUSES										
HTLV-1							1	1	.2	3
Rotavirus	1	21			5	27	24	78	128.0	1,379
Norwalk agent						3		3	.7	23
Small virus (like) particle						1		1	2.8	11
OTHER										
Chlamydia trachomatis not typed		12	13		3	4	40	72	80.5	1,968
Chlamydia psittaci						4		4	2.0	119
Mycoplasma pneumoniae							4	4	51.2	237
Rickettsia australis			1					1	.0	12
Streptococcus group A						1		1	16.7	433
Brucella species						1		1	.2	10
Bordetella pertussis						10	7	17	26.2	496
Legionella longbeachae							1	1	.0	14
Treponema pallidum		8					3	11	16.2	437
Entamoeba histolytica						1		1	.2	14
Toxoplasma gondii						1		1	2.0	106
Schistosoma species					1	8		9	.0	97
Strongyloides stercoralis			1			1		2	.0	14
TOTAL	1	166	44	168	50	331	535	1,295	1,715.7	32,714

1. State or Territory of postcode, if reported, otherwise State or Territory of reporting laboratory.
2. The historical data are the averages of the numbers of reports in 6 previous 2 week reporting periods: the corresponding periods of the last 2 years and the periods immediately preceding and following those.

Table 9. Virology and serology laboratory reports by clinical information for the reporting period 21 September to 4 October 1995

	Meningitis	Other CNS	Respiratory	Gastrointestinal	Hepatic	Skin	Eye	Muscle/joint	Genital	Other/unknown	Total
MEASLES, MUMPS, RUBELLA											
Measles virus						2				2	4
Mumps virus										4	4
Rubella virus						3		1		8	12
HEPATITIS VIRUSES											
Hepatitis A virus					7					2	9
Hepatitis B virus					10					50	60
Hepatitis C virus			1		78				1	124	204
Hepatitis D virus					1						1
ARBOVIRUSES											
Barmah Forest virus								1		1	2
Dengue not typed										1	1
Kunjin virus								1			1
Flavivirus (unspecified)										1	1
ADENOVIRUSES											
Adenovirus type 1			1								1
Adenovirus type 2			1								1
Adenovirus type 3			1								1
Adenovirus type 7			1								1
Adenovirus not typed/pending			21	7			1			2	33
HERPES VIRUSES											
Herpes simplex virus type 1		1	10			118	9		34	19	191
Herpes simplex virus type 2						93			86	9	188
Herpes simplex not typed/pending			1			3				1	5
Cytomegalovirus			33		3	2	2			12	52
Varicella-zoster virus						21			1	2	24
Epstein-Barr virus										17	17
OTHER DNA VIRUSES											
Papovavirus group										3	3
Parvovirus										2	2
PICORNA VIRUS FAMILY											
Echovirus type 3						1					1
Echovirus type 9	1					1					2
Echovirus type 11										1	1
Echovirus type 14			1								1
Echovirus type 22			1								1
Echovirus type 25						1					1
Echovirus not typed/pending				1						1	2
Poliovirus type 2 (uncharacterised)			2								2
Rhinovirus (all types)			24			1				3	28
Enterovirus not typed/pending			17	2		5				4	28

Table 9. Virology and serology laboratory reports by clinical information for the reporting period 21 September to 4 October 1995, continued

	Meningitis	Other CNS	Respiratory	Gastrointestinal	Hepatic	Skin	Eye	Muscle/joint	Genital	Other/unknown	Total
ORTHO/PARAMYXOVIRUSES											
Influenza A virus			6							3	9
Influenza A virus H_3N_2			1								1
Influenza B virus			13							7	20
Parainfluenza virus type 2			1							1	2
Parainfluenza virus type 3			41							4	45
Parainfluenza virus type 4			1								1
Parainfluenza virus typing pending			3								3
Respiratory syncytial virus			115			1				6	122
OTHER RNA VIRUSES											
HTLV-1										1	1
Rotavirus				76						2	78
Norwalk agent				3							3
Small virus (like) particle				1							1
OTHER											
Chlamydia trachomatis not typed						1	2		49	20	72
Chlamydia psittaci			3							1	4
Mycoplasma pneumoniae			4								4
Rickettsia australis										1	1
Streptococcus group A										1	1
Brucella species										1	1
Bordetella pertussis			14							3	17
Legionella longbeachae			1								1
Treponema pallidum									3	8	11
Entamoeba histolytica										1	1
Toxoplasma gondii										1	1
Schistosoma species										9	9
Strongyloides stercoralis										2	2
TOTAL	1	1	318	90	99	253	14	3	174	341	1295

Table 10. Virology and serology laboratory reports by contributing laboratories for the reporting period 21 September to 4 October 1995

STATE OR TERRITORY	LABORATORY	REPORTS
New South Wales	Prince Henry/Prince of Wales Hospitals, Sydney	56
	Royal Alexandra Hospital for Children, Camperdown	24
	South West Area Pathology Service, Liverpool	85
Queensland	State Health Laboratory, Brisbane	168
Tasmania	Northern Tasmanian Pathology Service, Launceston	18
	Royal Hobart Hospital, Hobart	30
Victoria	Monash Medical Centre, Melbourne	41
	Royal Children's Hospital, Melbourne	76
	Unipath Laboratories	24
	Victorian Infectious Diseases Reference Laboratory, Fairfield Hospital	194
Western Australia	PathCentre Virology, Perth	331
	Princess Margaret Hospital, Perth	66
	Western Diagnostic Pathology	182
TOTAL		1295