Infections with *Corynebacterium diphtheriae* - changing epidemiology and clinical manifestations

Report of the third international meeting of the European Laboratory Working Group on Diphtheria (ELWGD), Institut Pasteur, Paris 7 - 8 June 1996

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Abstract

A widespread epidemic of diphtheria began in 1990 in the former Soviet Union, in the context of falling immunisation rates and social disruption. Control was impeded by limited diagnostic resources in affected countries (mainly Russia) and there was a risk of spread to neighbouring countries. The European Laboratory Working Group on Diphtheria (ELWGD) was formed to assist in control of the epidemic. The ELWGD is convened by the Public Health Laboratory Service in the United Kingdom and includes 15 laboratories in Europe, one in North America (Centers for Disease Control and Prevention) and the Institute of Clinical Pathology and Medical Research, Sydney. At the group's last annual meeting in Paris, reports were presented on the progress of the epidemic, control strategies and improvements in laboratory diagnosis. The group discussed the increased carriage of, and infection with, nontoxigenic *Corynebacterium diphtheriae* in countries with high immunisation rates, including the United Kingdom and Australia. They also considered the possible relationship between this increase and the continued diphtheria outbreak in eastern Europe. Preliminary results of molecular typing of toxigenic and nontoxigenic isolates from many parts of the world were presented. It was agreed that further epidemiological investigation is required, using a standardised ribotyping system. *Comm Dis Intell* 1997;21:161-164

Introduction

In most developed countries, classical respiratory diphtheria has become rare over the past 40 years due to effective immunisation. However, the disease remains endemic in many countries, including Turkey, Bangladesh, Vietnam, Africa and some parts of South America. Cutaneous diphtheria occurs in many tropical areas, usually without causing systemic complications. Small outbreaks of potentially fatal respiratory diphtheria occur occasionally in Europe and North America, usually introduced by an imported case of either respiratory or cutaneous diphtheria.

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Table. Cases of diphtheria reported in the WHO European region, 1990 to 1995

Year	Total cases (rate per 100,000 population)	Russia (rate per 100,000 population)	Other NIS[1] (rate range)
1990	1483 (0.002)	1211 (0.82)	225 (0-0.22)
1991	3216 (0.004)	1876 (1.27)	1119 (0-2.13)
1992	5814 (0.007)	3897 (2.63)	1850 (0-3.0)
1993	19608 (0.023)	15209 (10.3)	4295 (0.03-11.8)
1994	47707 (0.055)	39582 (26.9)	8046 (0.45-32.2)
1995	50445 (0.058)	35652 (24.3)	14438 (0.81-73.0)

1. NIS - Newly independent states of the former Soviet Union

Recent significant outbreaks have occurred among skid row alcoholics in Seattle (1972-82) and in Scandinavia (1984-86) despite very high infant immunisation rates[1,2].There had previously been no indigenous cases for more than 20 years in these locations .

The largest and most widespread epidemic of diphtheria in recent times began in 1990 in countries of the former Soviet Union. More than one hundred and thirty thousand cases have been reported (Table) and the epidemic is continuing[3].

In 1993, the ELWGD was formed to:

- develop guidelines and outline future study needs and directions for laboratories;
- strengthen laboratory collaboration and support, particularly to those in greatest need;
- increase current knowledge and develop new technology relating to the laboratory diagnosis and epidemiological surveillance of *Corynebacterium diphtheriae*;
- and, importantly, to form an international network of Diphtheria Reference Centres[4].

The group includes 18 laboratories, 15 in Europe and three outside Europe including the Department of Clinical Microbiology, Institute of Clinical Pathology and Medical Research, New South Wales.

At the third international meeting of the group, at the Institut Pasteur, Paris in June 1996, the current epidemiology of diphtheria in Eastern Europe was reviewed. The group also discussed methods of laboratory diagnosis, antibiotic susceptibility, typing methods and molecular epidemiology of toxigenic and nontoxigenic *C. diphtheriae.*

Incidence of diphtheria in Europe

Dr Colette Roure, from the World Health Organization's Regional Office for Europe, reported that between 1980 and 1989 the average number of cases of diphtheria reported in Europe was 1100 (average annual rate, 0.0013 per 100,000 population). Of these more than 90% were from the USSR (average annual rate, 0.38 per 100,000 population per annum). In 1990, there was a dramatic increase. This was attributed to decreasing childhood immunisation rates, waning immunity in adults and major population movements since the dissolution of the former Soviet Union. Although all age groups were affected, the highest incidence was in adolescents and young adults. The case fatality rate was generally 5-10% but rates up to 22% were reported when there was a delay in diagnosis and treatment.

Cases of diphtheria imported from eastern Europe have been reported in a number of western European countries.

Control strategies

Control strategies recommended by WHO include:

- mass immunisation in countries where the rate is 3.5 per 100,000 population or more;
- improvement in routine immunisation rates to achieve an uptake of 95% in children and 90% in adults;
- confirmation of the diagnosis in suspected cases;
- appropriate treatment of infected individuals; and
- rapid investigation of contacts.

Implementation of these strategies, to varying degrees, has been reflected by a decrease in the number of new cases or a slowing in the rates of increase. For example following mass immunisation in Russia there was a 10% fall in cases between 1994 and 1995 which was sustained in the first few months of 1996. This compares with 2-3 fold increases in the number of cases each year for the previous three years.

Control programs have been frustrated in some countries by vaccine shortages and inadequate laboratory diagnostic facilities.

Laboratory diagnosis

Laboratory diagnosis of diphtheria involves isolation of the causative organism on selective medium (blood tellurite agar), biochemical testing to confirm the species and biovar, and tests for toxigenicity.

In some areas of the region basic laboratory skills required for the diagnosis of diphtheria had been lost. This loss of expertise was largely due to the occurrence of few cases and hence lack of experience. Basic culture media and biochemical reagents were not available in many countries affected by the epidemic. To address these problems, training workshops were held at the WHO Collaborating Center, Central Public Health Laboratory (CPHL), Colindale, United Kingdom. In addition laboratory kits were developed for distribution by WHO to laboratories. These kits contain all the basic requirements for confirmation of the diagnosis in 100 suspected cases and investigation of 1,000 contacts.

The traditional methods of detection of toxin production by *C. diphtheriae* are either the Elek test or guinea pig inoculation. Both these methods are relatively slow. Animal inoculation is expensive and, increasingly, ethically unacceptable. The traditional Elek test involves the use of a filter paper strip impregnated with diphtheria antitoxin. This is incorporated into clear agar on which the test organism(s) and

appropriate controls are inoculated at right angles to the strip. Following 48 hours incubation toxin produced by the test organism is shown by a line of precipitation which forms a line of identity with that produced by a positive control strain. In practice both false negative results (due to reduced sensitivity) and false positive results (due to nonspecific lines of precipitation) are not uncommon. A modification of this method was described by Dr Kate Engler of the CPHL, United Kingdom. This involves the use of a very thin layer of agar in small agar plates and an antitoxin-impregnated disc, around which heavy spot-inocula of test and control organisms are placed. Lines of precipitation are visible after only 16-24 hours incubation, before any nonspecific precipitation occurs. This method requires further evaluation but is potentially more rapid, accurate and economical than the conventional Elek test. It will be more accessible to laboratories in high prevalence areas, where resources are extremely limited, than newer molecular methods which are being used increasingly in Western countries.

Polymerase chain reaction (PCR) has been developed to detect phage-encoded toxin genes of *C. diphtheriae*[5-7]. Potential targets for amplification include *tox*A and *tox*B which encode toxin fragments A and B respectively and *dtx*R, which encodes an iron-dependent toxin regulatory protein. For isolates which have been identified as *C. diphtheriae,* there is generally an excellent correlation between the presence of *tox*A (the most commonly used target) and biological toxigenicity[6]. *C. ulcerans* and *C. pseudotuberculosis* are also potentially toxigenic and occasionally clinically significant. However, some other *Corynebacterium* species, without the ability to produce toxin, possess the *tox*A gene, and can give false positive PCR results. Moreover, rare biologically nontoxigenic *C. diphtheriae* strains possess the toxin gene(s) which are either repressed or defective. Although the potential clinical significance of these strains is unknown, PCR results should be interpreted with caution and only in association with the results of conventional methods of identification and toxigenicity testing.

PCR also has the potential for the detection of toxigenic *C. diphtheriae* directly in clinical specimens such as throat swabs. Dr Tanja Popovic of the Centers for Disease Control and Prevention, Atlanta described a direct PCR which is quite sensitive and can detect 150 organisms. However this method is dependent on optimisation of a number of factors, including the type of swab used, transport and storage conditions and DNA extraction method.

Antibiotic susceptibility of C. diphtheriae

The antibiotic susceptibility of 38 nontoxigenic strains of *C. diphtheriae* isolated in France between 1987 and 1993 was reported by Dr O Patey. All were susceptible to penicillin, most other commonly used β-lactams, vancomycin and perfloxacin. Two strains were resistant to lincomycin; of these one was resistant to erythromycin and the susceptibility of the other was reduced. Seven isolates (18%) were resistant to rifampicin and of these one was also resistant to erythromycin and lincomycin. Penicillin or erythromycin are the agents of choice for treatment of diphtheria or nontoxigenic *C. diphtheriae* infections. Carriers are usually treated with erythromycin. However, Dr Patey reported that eradication of carriage was more likely after treatment with rifampicin (91% after five days; 97% after seven days treatment) than with erythromycin (64% after five days, 89% after seven or ten days treatment). The value of rifampicin for treatment of carriers will depend on the degree of resistance.

Nontoxigenic C. diphtheriae infections

There have been an increasing number of reports of disease due to nontoxigenic *C. diphtheriae* in recent years, mainly in children and young adults. Outbreaks of pharyngitis have occurred among homosexual men and in educational and military establishments. Invasive infections, mainly endocarditis and septic arthritis, have been reported[1,8,9]. Most cases have been due to *C. diphtheriae* biovar *gravis.* Invasive disease is associated with significant morbidity and some mortality. Most affected individuals had been previously immunised against diphtheria, although their antitoxin levels at the time of infection were not recorded. There are no recent data available on carriage rates, but nontoxigenic *C. diphtheriae* were isolated from throat or nose swabs of 12 of 359 contacts (3%) of patients with invasive nontoxigenic *C. diphtheriae* infections in Victoria in 1994. Seven isolates were *C. diphtheriae* var *belfanti* and 5 (1.4%) were var *gravis*[9].

In the United Kingdom, 50-60% of laboratories routinely culture throat swabs for *C. diphtheriae.* Dr Androulla Efstratiou reported that the number of isolates referred to the CPHL, London, for toxigenicity testing increased from 17 in 1990 to 140 in 1995. Seventy-five per cent of these were *C. diphtheriae* var *gravis*. To determine the clinical significance of these isolates, a questionnaire was sent to referring doctors and laboratories. Most isolates were from throat swabs of children and young adults with severe pharyngitis which had not responded to, or recurred after treatment with, penicillin. Most responded to therapy with erythromycin. The greatest proportion of isolates were from general practice or genitourinary medicine clinics and, in most cases, no other potential pathogen had been isolated although viral cultures had rarely been done. There have been few cases of invasive infection in the United Kingdom.

The significance of these findings is not clear. Mechanisms of pathogenicity of nontoxigenic *C. diphtheriae* are poorly understood. The organism clearly is potentially invasive in a minority of individuals, many of whom have underlying risk factors, such as intravenous drug use or cardiac valvular disease[8]. It is known that nontoxigenic *C. diphtheriae* can regain toxigenicity by lysogeny with the phage carrying the toxin gene and it is postulated that this can occur in vivo[10]. Dr G Tseneva of the Pasteur Institute, St Petersburg reported that some *C. diphtheriae* isolates from long-term carriers, which were nontoxigenic by Elek test and rabbit inoculation, were shown by PCR to contain *tox*A. After repeated passaging on Elek medium, which contains a low iron content to inhibit the toxin repressor protein (DtxR), 50% of these isolates produced toxin. Thus, nontoxigenicity is apparently sometimes due to reversible toxin gene repression, rather than loss of the gene or the carrier phage.

There have been a relatively large number of cases of invasive nontoxigenic *C. diphtheriae* infections in Australia recently. This includes at least seven in New South Wales, one each in Queensland and Western Australia and three in Victoria.[8, 9, 11] It is therefore likely that throat carriage or infection is not uncommon but remains undetected because most laboratories do not culture throat swabs from patients with sore throats for *C. diphtheriae*.

Molecular typing of C. diphtheriae

A variety of methods for the epidemiological typing of *C. diphtheriae* isolates have been described. These include ribotyping and pulsed field gel electrophoresis (PFGE)[11,12]. They have been used to demonstrate predominant ribotypes among toxigenic isolates of both *C. diphtheriae* var *gravis* and var *mitis* from Russia and surrounding countries. They have also been used to trace the origin of imported cases in western Europe[12].

Multiple clones of nontoxigenic *C. diphtheriae* var *gravis,* with one predominating (six of seven isolates from cases in New South Wales), were shown to have caused invasive infections in Australia[11]. PFGE was used to demonstrate similarity between the New South Wales isolates and those from three patients with endocarditis and five of their contacts in Victoria[12]. Dr Aruni DeZoysa (CPHL, London) reported that, among 118 nontoxigenic *C. diphtheriae* var *gravis* isolates referred to the CPHL in 1995, there were 23 different ribotypes. However, 75% belonged to a single ribotype which, on the basis of preliminary results, appears to be very similar to a ribotype found among isolates from Eastern Europe.

Unfortunately, because different endonucleases, probes and ribotype nomenclature are used, the results of one study cannot be compared with those of another. It was therefore proposed by Professor Patrick Grimont of the Institut Pasteur, Paris that a standard ribotyping method and common nomenclature be adopted. This would enable the establishment of a database of ribotypes, validated using appropriate computer software. It would also assist in the international surveillance of outbreaks of diphtheria and nontoxigenic *C. diphtheriae* infections, contribute to a better understanding of the epidemiology of this disease and improve disease control worldwide.

Ribotyping and PFGE of *C. diphtheriae* are being performed at the ICPMR, Westmead. Ribotyping will be standardised with the international method once this has been established. However, in a recent comparison of the two methods using 100 toxigenic and nontoxigenic isolates of *C. diphtheriae*, we found that PFGE was significantly more discriminatory than ribotyping (K Cheung and L Gilbert, unpublished data).

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Editorial: Diphtheria - the Australian perspective

Henry Kilham[1] and Richard Benn[2]

Diphtheria has been a major cause of morbidity and mortality in Australian history. A decline in the incidence of this disease began with the implementation of public heatlh measures before the infective nature of the disease was understood. The death rate was greatly reduced when antiserum became available a century ago. Active immunisation began in the 1920s. This was in widespread use by the 1940s and led to the almost complete elimination of the disease by the 1960s. However sporadic cases have continued to occur in unimmunised individuals. In 1984 the National Health and Medcal Research Council recommended the use of ADT (adult diphtheria-tetanus toxoid) in place of tetanus toxoid for adult booster immunisation.

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Death from diphtheria in Australia is now rare. However notifications of bacteriologically proven diphtheria-related conditions continue to occur. The National Notifiable Diseases Surveillance System recorded eight cases in 1991, 14 in 1992 and one in 1993[1]. Toxigenic as well as non-toxigenic strains of *Corynebacterium diphtheriae* remain endemic in parts of Australia[2].

The most important lesson to be learnt from recent outbreaks in the former Soviet Union, is that diphtheria recurs when community susceptibility increases and toxigenic organisms recirculate. In Australia many adults will now be susceptible, even if previously immunised, because of lack of natural boosting. This is a direct result of previous success in eradicating the organism from the community. We now have a large population of individuals whose only protection came from childhood immunisation. They have had no subsequent boosting either from further vaccine or from occasional contact with the organism. In the former Soviet Union there was social disruption and a considerable reduction in childhood immunisation. This provided conditions which enabled imported diphtheria to spread more easily resulting in a high incidence of disease, particularly in adults whose immunity had lapsed.

There have been no diphtheria serosurveillance studies carried out in Australia recently. However, using the international standard of susceptibility (antitoxin <0.01 IU/mL), 35% of United Kingdom-born blood donors aged 40 to 49 years are susceptible, and 53% of those aged 50 to 59[3]. It can be assumed that similar rates would apply in Australia, the United States of America and the former Soviet Union up until 1990. Spread of toxigenic *C. diphtheriae* into a susceptible Australian population could be expected to produce outbreaks and deaths. It should not be forgotten that there is no effective treatment for diphtheritic myocardiopathy, which is commonly fatal.

Some countries close to the former Soviet Union such as Finland and Poland have commenced adult immunisation against diphtheria. In Australia there appears to be no cause for alarm at present. However the possibility of a resurgence of diphtheria must be acknowledged. The following measures are pertinent:

- improving the uptake of childhood immunisation, to reduce the number of susceptible children and hence spread of any imported organism in the community;
- maintaining adequate surveillance, which must include maintaining skills in bacteriological diagnosis, even if only at selected laboratories. The European experience detailed by Dr Gilbert, in this issue, should assist in choosing the best approaches;
- achieving better adult immunisation, especially in migrants arriving without evidence of adequate childhood immunisation. Also for Australians intending to travel to areas where diphtheria is endemic;
- being prepared to embark upon localised mass immunisation of susceptible populations, for both adults and children, should an outbreak occur, the use of other public health interventions such as active case-finding and isolation for such outbreaks.

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National Health and Medical Research Council recommendations on diphtheria vaccination

The National Health and Medical Research Council recommends diphtheria vaccination as part of the standard childhood vaccination schedule[1]. Primary vaccination is achieved with three doses of a diphtheria toxoid-containing vaccine at one to two monthly intervals, with boosters at 18 months and four to five years.

Prior to the eighth birthday DTP (diphtheria, tetanus, pertussis vaccine) should be given. If there is a genuine contraindication to pertussis vaccine DT (adsorbed diphtheria, tetanus vaccine, CDT paediatric formulation) should be used. After the eighth birthday, the adult formualtion (Td, ADT) should be given. The change to Td (ADT) (low dose diphtheria toxoid) after the eighth birthday is related to the reduced tolerance of older children and adults to diphtheria toxoid.

Older children who have not received diphtheria vaccination are also likely to have missed tetanus vaccination. Those who have not reached their eighth birthday should receive three injections of DTP (or DT, CDT) at intervals of one to two months, and those individuals who have passed their eighth birthday should receive three doses of Td (ADT) at intervals of two months.

The need for booster injections in adult life is unclear. However, as protective antibody levels wane with age, it is considered prudent for adults to have booster injections, which may be given as Td (ADT) vaccine, at 10 year intervals. Diphtheria can be a significant risk for travellers to some countries, so all international travellers should ensure that their Td (ADT) vaccination is current.

Reference

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Communicable Diseases Surveillance

Shigellosis

Shigellosis is a highly communicable acute bacterial disease involving the large and distal small intestine. Studies have shown that infection may occur following ingestion of only 10 - 100 organisms. Symptoms include diarrhoea, accompanied by fever, nausea and sometimes toxaemia, vomiting and cramps. Mild and asymptomatic infections also occur. Illness is usually self-limited, lasting an average of 4 - 7 days. The severity of the disease is a function of both the host and the particular serotype. The disease is more severe in children, the elderly, the debilitated and the malnourished.

There are four species or serogroups of *Shigella*: Group A, *S. dysenteriae*; Group B, *S. flexneri*; Group C, *S. boydii*; and Group D, *S. sonnei*. The groups are further divided into a number of different serotypes and subtypes. More than one serotype commonly occurs in a particular community. In general, *S. sonnei* is the most common and *S. dysenteriae* the least common in developed countries. *S. flexneri*, *S. boydii* and *S. dysenteriae* generally account for the majority of isolates in developing countries. In Australia, *S. sonnei* appears to be the most common isolate. However, *S. flexneri* is also relatively common, being predominantly reported in Aboriginal communities. *Shigella* is also frequently isolated from recent overseas travellers.

Shigellosis is predominantly spread by personal contact, with the principal route of transmission being the faecal-oral route. The only reservoir is humans. Over-crowding and poor personal hygiene are risk factors for acquiring infection. Less frequently, food vehicles have been implicated in the transmission of the disease. Outbreaks have also been linked to the consumption of contaminated fresh water when swimming. Prevention of infection includes the provision of appropriate sanitary conditions and education regarding personal hygiene.

Although *Shigella* is endemic in Australia, notifications usually peak in the summer months. Flies may contribute to higher transmission rates during these times. National Notifiable Diseases Surveillance System data from 1994 to 1997 show the highest number of notifications for shigellosis usually occur in January, February and March (Figure 1). Notification rates vary throughout Australia and are highest in warmer climates. In 1996, Queensland had the highest number of notifications (239), followed by the Northern Territory (145) and Western Australia (140). The highest number of notifications were for children under 10 years of age, accounting for 47% of reports for that year (Figure 2).

National Notifiable Diseases Surveillance System

The NNDSS is conducted under the auspices of the Communicable Diseases Network Australia New Zealand. The system coordinates the national surveillance of more than 40 communicable diseases or disease groups

Figure 1. Shigellosis notifications, 1994 to April 1997, by month of onset

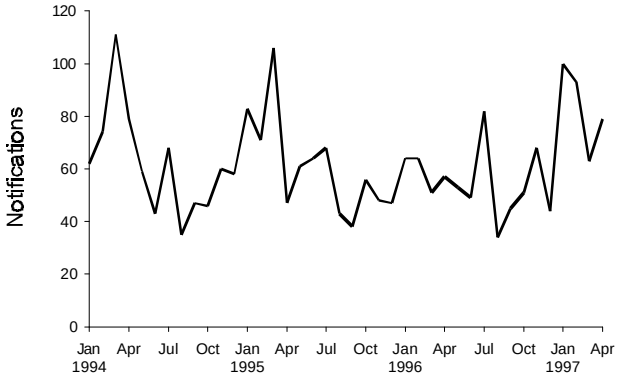


Figure 2. Shigellosis notifications, 1996, by age group

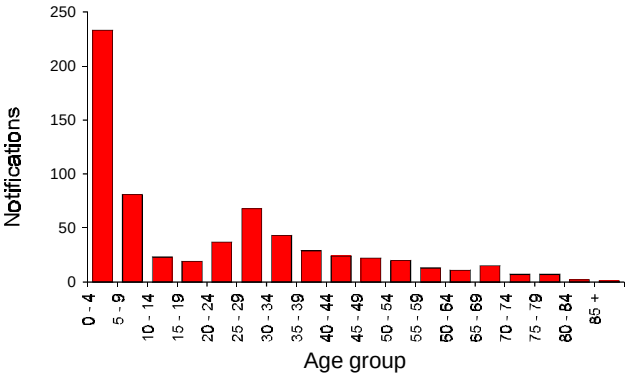


Figure 3. Q fever notifications, 1993 to 1997, by month of onset

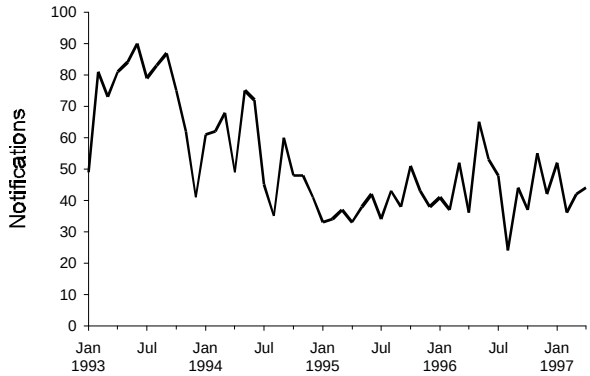


Table 1. Notifications of diseases preventable by vaccines recommended by the NHMRC for routine childhood immunisation, received by State and Territory health authorities in the period 14 to 27 May 1997

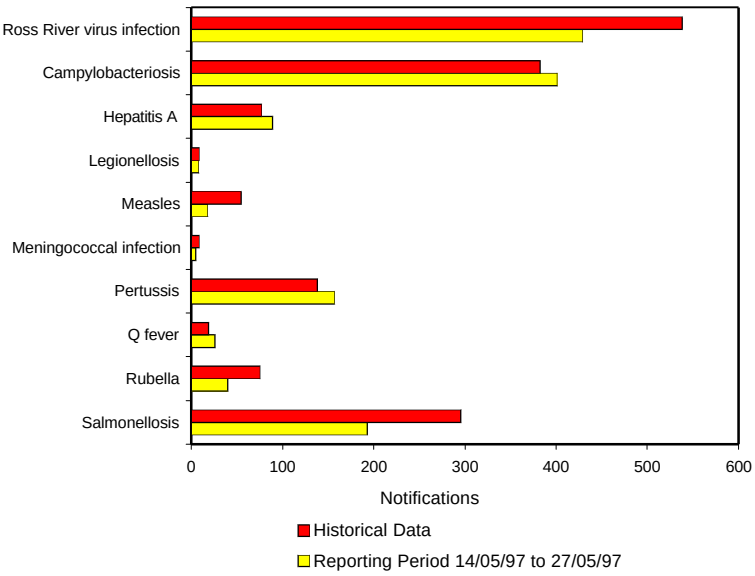
Disease[1,2]	ACT	NSW	NT	Qld	SA	Tas	Vic	WA	This period 1997	This period 1996	Year to date 1997	Year to date 1996
Diphtheria	0	0	0	0	0	0	0	0	0	0	0	0
Haemophilus influenzae type B	0	2	1	0	0	0	0	0	3	3	22	23
Measles	1	7	1	2	0	0	6	1	18	22	188	199
Mumps	2	1	1	NN	2	1	4	0	11	3	81	47
Pertussis	2	44	2	23	39	2	35	10	157	110	3074	1300
Rubella	1	1	0	14	10	2	6	6	40	79	600	1161
Tetanus	0	0	0	0	0	0	0	0	0	0	3	1

NN. Not Notifiable

1. No notifications of poliomyelitis have been reported since 1986.
2.

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

Figure 4. 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.

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 1997;21:5.

Reporting period 14 to 27 May 1997

There were 2,545 notifications received for this two week period (Tables 1, 2 and 3). The numbers of reports for selected diseases have been compared with historical data for corresponding periods in the previous three years (Figure 4).

There were 26 notifications of Q fever this period. Of the notifications for the year to date (233), most have been from Queensland (117) and New South Wales (102). The male:female ratio was 5.6:1 and the highest number of notifications were for the 35 - 39 years age group. There has been a general downward trend in Q fever notifications since a peak reached in mid-1993 (Figure 3).

Reports of salmonellosis remain at a high level with 193 reports received in this period (Figure 5). The majority of reports were from Queensland (54) and New South Wales (45). The number of notifiications so far received for 1997 is 3,860. The majority were from Victoria (1,073, 28%) and Queensland (1,032, 27%). The male:female ratio was 1:1 and most cases (1,321, 34%) were in the 0 - 4 years age group.

There were 8 notifications of legionellosis in this period, bringing the total to 70 for the year to date. Legionellosis is most commonly reported for males in the 60 - 69 years age range (Figure 6). Overall, males are more commonly notified than females, the male:female ratio being 2.2:1.

Figure 5. Salmonellosis notifications, 1995 to 1997, by month of onset

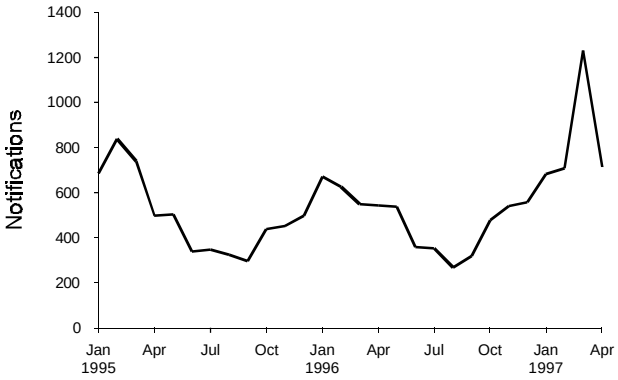


Figure 6. Legionellosis notifications, 1991 to 1996, by age group and sex

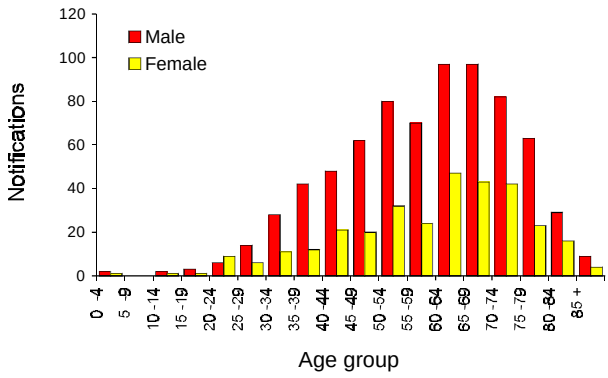


Table 2. Notifications of other diseases received by State and Territory health authorities in the period 14 to 27 May 1997

Disease[1,2]	ACT	NSW	NT	Qld	SA	Tas	Vic	WA	This period 1997	This period 1996	Year to date 1997	Year to date 1996
Arbovirus Infection (NEC)[3]	0	1	4	0	0	0	4	0	9	4	93	67
Barmah Forest virus infection	0	19	0	19	0	0	2	-	40	29	405	506
Campylobacteriosis[4]	17	-	12	112	75	13	130	42	401	453	4741	4770
Chlamydial infection (NEC)[5]	7	NN	31	142	0	8	0	39	227	313	3251	2929
Dengue	0	0	0	0	0	-	0	0	0	3	190	23
Donovanosis	0	NN	1	0	NN	0	0	0	1	4	12	23
Gonococcal infection[6]	0	13	94	38	0	1	0	26	172	184	1787	1534
Hepatitis A	0	36	14	24	5	0	7	3	89	93	1534	1069
Hepatitis B incident	0	3	6	3	0	0	3	4	19	7	154	95
Hepatitis C incident	0	1	0	-	0	0	-	-	1	1	6	14
Hepatitis C unspecified	12	NN	19	146	NN	5	128	18	328	391	3504	3748
Hepatitis (NEC)	0	0	0	0	0	0	1	NN	1	0	9	10
Legionellosis	0	2	0	0	3	0	2	1	8	9	70	83
Leptospirosis	0	2	0	0	1	0	1	0	4	9	53	104
Listeriosis	0	1	0	0	1	0	0	1	3	1	40	23
Malaria	1	9	0	45	0	0	4	1	60	17	346	319
Meningococcal infection	0	0	0	2	0	0	3	0	5	12	128	107
Ornithosis	0	NN	0	0	1	0	3	0	4	9	32	39
Q Fever	0	12	0	13	0	0	1	0	26	15	233	190
Ross River virus infection	0	145	24	190	19	1	37	13	429	286	5342	6727
Salmonellosis (NEC)	1	45	18	54	20	2	37	16	193	273	3860	2898
Shigellosis[4]	2	-	10	8	5	0	1	3	29	32	392	282
Syphilis	2	22	17	10	0	0	0	0	51	73	493	610
Tuberculosis	1	8	4	7	1	0	13	2	36	43	397	474
Typhoid[7]	0	1	1	0	0	0	1	0	3	1	37	47
Yersiniosis (NEC)[4]	0	-	0	2	1	0	2	0	5	6	134	111

1. For HIV and AIDS, see *CDI* 1997;21:154. For rarely notified diseases, see Table 3.
2. Totals comprise data from all States and Territories. Cumulative figures are subject to retrospective revision so there may be discrepancies between the number of new notifications and the increment in the cumulative figure from the previous period.
3. NT and WA: includes Barmah Forest virus.
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.

NECNot Elsewhere Classified

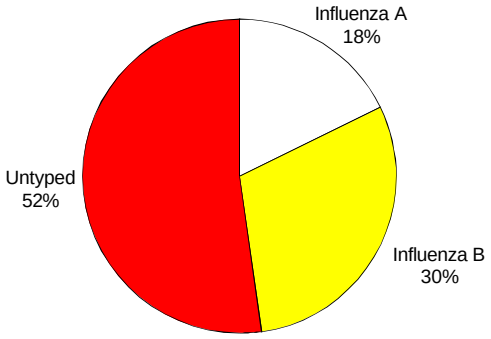
\- Elsewhere Classified.

Table 3. Notifications of rare[1] diseases received by State and Territory health authorities in the period 14 to 27 May 1997

Disease[2]	Total this period	Reporting States or Territories	Total notifications 1997
Brucellosis	2	Qld	16
Chancroid			1
Cholera			1
Hydatid infection	3	Qld, Vic	14
Leprosy			7

1. Fewer than 60 cases of each of these diseases were notified each year during the period 1988 to 1996.
2. No notifications have been received during 1997 for the following rare diseases: botulism, lymphogranuloma venereum, plague, rabies, yellow fever, or other viral haemorrhagic fevers.

Figure 8. Laboratory reports of influenza, 1997, by type



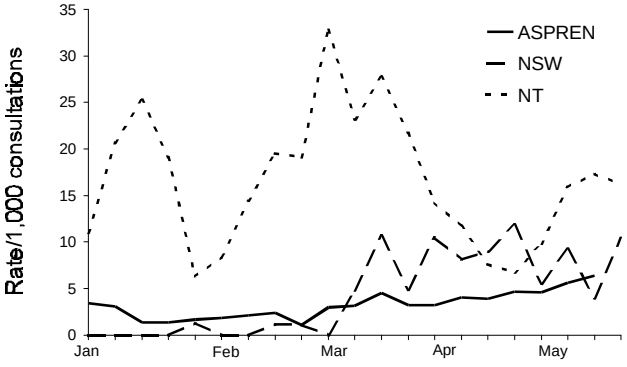
National Influenza Surveillance, 1997

Three types of data are included in National Influenza Surveillance, 1997. 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 Department of Health and Community Services, 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 1997; 21:126.

Sentinel general practitioner surveillance

Consultation rates for influenza-like illness rose to 10.4 per 1,000 encounters in New South Wales over the last fortnight and remained steady at 16.2 per 1,000 encounters in the Northern Territory (Figure 7). The consultation rate recorded by ASPREN rose slightly this period to 6.4 per 1,000 encounters.

Figure 7. Sentinel general practitioner influenza consultation rates, 1997, by week and scheme



Laboratory surveillance

Forty-three reports of influenza virus were recorded by the LabVISE scheme this fortnight, 11 influenza A, 8 influenza B and 24 untyped. For the year to date, 293 reports of influenza have been received. More than half of these are untyped and, of those that have been typed, the majority are influenza B (Figure 8). If there is the same proportion of influenza A and B among the untyped, 63% of all reports would be influenza B. Overall the male:female ratio was 1:1 and 20% of patients were over 65 years of age. The number of laboratory reports remains average for the time of year.

Absenteeism surveillance

Australia Post recorded a national absenteesim rate of 2.6% and 2.7% over the last fortnight, similar to previous weeks.

Australian Sentinel Practice Research Network

The Australian Sentinel Practice Research Network (ASPREN) comprises 99 sentinel general practitioners from throughout the country. Approximately 9,000 consultations are recorded each week for 12 conditions. Of

Table 4. Australian Sentinel Practice Research Network reports, week 20, to 18 May 1997

	Week 20 , to 18 May 1997	
Condition	Reports	Rate per 1,000 encounters
Chickenpox	19	2.5
Gastroenteritis	84	10.9
HIV testing (doctor initiated)	3	0.4
HIV testing (patient initiated)	16	2.1
Influenza	49	6.4
Measles	0	0.0
Pertussis	2	0.3
Ross River virus infection	3	0.4
Rubella	2	0.3

these, CDI reports the consultation rates for chickenpox, gastroenteritis, HIV testing (doctor initiated), HIV testing (patient initiated), influenza, measles, pertussis, Ross River virus infection and rubella. For further information including case definitions see CDI 1997;21:6.

Data for week 20 ending 18 May is included in this issue of *CDI* (Table 4). The consultation rate for chickenpox is slightly higher than in recent weeks. The consultation rate for gastroenteritis has continued at relatively low levels since mid-January 1997. Consultation rates for HIV testing have remained slightly higher than the rates experienced during April. Consultation rates for Ross River virus infection, measles, rubella and pertussis remain low.

LabVISE

The Virology and Serology Laboratory Reporting Scheme, LabVISE, is a sentinel reporting scheme. Twenty-one laboratories contribute data on the laboratory identification of viruses and other organisms. Data are collated and published in Communicable Diseases Intelligence each fortnight. These data should be interpreted with caution as the number and type of reports received is subject to a number of biases. For further information, see CDI 1997;21:8-9.

There were 708 reports received in the *CDI* Virology and Serology Laboratory Reporting Scheme this period (Tables 5 and 6).

Fifty-one reports of Ross River virus were received this fortnight for 26 males and 25 females. Fewer reports have been received for the year to date than for the same period in 1996 (Figure 9).

A total of 36 reports of parainfluenza virus were received this period. Included were parainfluenza virus type 2 (15), type 3 (16) and untyped (5). The number of reports of parainfluenza virus type 2 has risen recently (Figure 10). As the last epidemic year for this virus was 1995 and outbreaks tend to occur in alternate years in Australia, we can expect more reports in the coming months. By contrast peaks in parainfluenza virus type 3 activity are recorded each year during late winter and early autumn.

Figure 9. Ross River virus laboratory reports, 1995 to 1997, by month of specimen collection

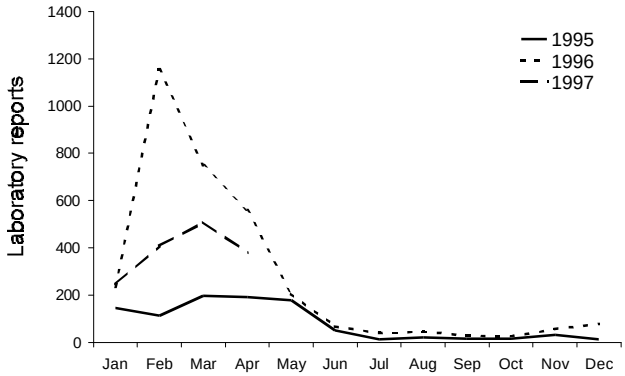
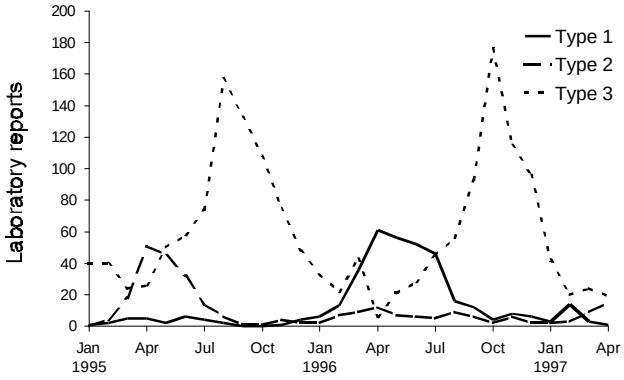


Figure 10. Parainfluenza virus laboratory reports, 1995 to 1997, by month of specimen collection



Ten reports of Q fever were reported this fortnight. Included were 8 males aged 17 - 52 years and 2 females in the 33 - 36 years age range.

Reports of respiratory syncytial virus rose to 169 in April which is average for the time of year. Two hundred and four reports were received this fortnight. Of these, 63% were for patients under one year of age and 93% were for children under the age of 5 years.

Table 5. Virology and serology laboratory reports by State or Territory[1] for the reporting period 8 to 21 May 1997, historical data[2], and total reports for the year

	State or Territory[1]						Total this fortnight	Historical data[2]	Total reported in *CDI* in 1997
	NSW	Qld	SA	Tas	Vic	WA			
Measles, mumps, rubella									
Rubella virus			4				4	14.2	371
Hepatitis viruses									
Hepatitis A virus			4				4	18.2	399
Hepatitis D virus		3					3	1	13
Arboviruses									
Ross River virus		37	11		3		51	182.7	1,699
Barmah Forest virus		2					2	16.8	157
Dengue type 2		3					3	0.2	47
Dengue not typed		1					1	0.3	39
Flavivirus (unspecified)		1					1	1.5	22

Table 5. Virology and serology laboratory reports by State or Territory[1] for the reporting period 8 to 21 May 1997, historical data[2], and total reports for the year, continued

	State or Territory[1]						Total this fortnight	Historical data[2]	Total reported in *CDI* in 1997
	NSW	Qld	SA	Tas	Vic	WA			
Adenoviruses									
Adenovirus type 2					2		2	0.3	21
Adenovirus type 3					2		2	2.2	16
Adenovirus type 4					1		1	0.2	4
Adenovirus type 8					1		1	1.7	6
Adenovirus not typed/pending	9	14	2		17	1	43	32.5	437
Herpes viruses									
Cytomegalovirus	6	10	1	1	7	10	35	59.8	569
Varicella-zoster virus	3	3	10		21	1	38	37	662
Epstein-Barr virus	4		29		5		38	71.5	1,320
Other DNA viruses									
Poxvirus group not typed					1		1	0	2
Parvovirus	1				8		9	4.8	185
Picornavirus family									
Coxsackievirus B2	1				1		2	0.2	10
Coxsackievirus B3	2						2	0.2	5
Echovirus type 5	1						1	0	4
Echovirus type 30					2		2	0.5	2
Poliovirus type 2 (uncharacterised)	1				1		2	0	8
Poliovirus type 3 (uncharacterised)	1						1	0.3	2
Rhinovirus (all types)	5	10			2		17	26.7	280
Enterovirus not typed/pending		8					8	26	290
Ortho/paramyxoviruses									
Influenza A virus		6			5		11	24	162
Influenza B virus	2	3			3		8	5.3	130
Influenza virus - typing pending			24				24	0.5	169
Parainfluenza virus type 2	2	1	1		11		15	9.2	48
Parainfluenza virus type 3	3	2	1		10		16	10.5	366
Parainfluenza virus typing pending			5				5	1	176
Respiratory syncytial virus	83	16	3	1	101		204	163.2	641
Other RNA viruses									
Rotavirus	2		16		21		39	48	417
Norwalk agent					1		1	1.2	55
Other									
Chlamydia trachomatis not typed	13	14	19	3	2	3	54	142.2	2,231
Chlamydia psittaci					1		1	3.2	40
Chlamydia species	1						1	2	17
Mycoplasma pneumoniae	15	2	3		6		26	17.2	808
Coxiella burnetii (Q fever)	7	2			1		10	8	151
Rickettsia australis		1					1	1.2	11
Bordetella pertussis				1	14		15	13.8	966
Cryptococcus species						1	1	0.8	11
Leptospira hardjo		2					2	1.8	12
TOTAL	162	141	133	6	250	16	708	951.7	12,981

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 6. Virology and serology laboratory reports by contributing laboratories for the reporting period 8 to 21 May 1997

State of Territory	Laboratory	Reports
New South Wales	Institute of Clinical Pathology & Medical Research, Westmead	31
	The New Children's Hospital, Westmead	35
	Royal Prince Alfred Hospital, Camperdown	9
	South West Area Pathology Service, Liverpool	86
Queensland	State Health Laboratory, Brisbane	139
South Australia	Institute of Medical and Veterinary Science, Adelaide	133
Tasmania	Northern Tasmanian Pathology Service, Launceston	6
Victoria	Microbiological Diagnostic Unit, University of Melbourne	2
	Monash Medical Centre, Melbourne	28
	Royal Children's Hospital, Melbourne	144
	Victorian Infectious Diseases Reference Laboratory, Fairfield	79
Western Australia	Royal Perth Hospital	16
TOTAL		708

Overseas briefs

Source: World Health Organization (WHO)

Anthrax, Ghana

An outbreak of human anthrax has been reported from a village in Bolgatanga District in the Upper East Region. The outbreak began in mid-April when district health services identified 26 active cases and 14 deaths.

The outbreak was rapidly brought under control and appears to have been related to the consumption of carcasses of cattle which had died of a sudden illness. A total of 185 cases occurred, of whom 26 died. Widespread health education has been important in the control of the outbreak. The District Veterinary Service has organised mass immunisation of cattle. The slaughter and sale of cattle in the area is temporarily banned.

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Contributions covering any aspects of communicable diseases are invited. Instructions to authors can be found in *CDI* 1997;21:9.

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