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Communicable Diseases Intelligence

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Contents:

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OVERSEAS BRIEFS

1. PERU CHOLERA EPIDEMIC

The number of cholera cases in Peru is continuing to rise. A recent report from the WHO/PAHO mentions that as of 18 February there were 22,477 confirmed cases (including 5,122 hospitalised) and 114 fatalities.

An unconfirmed report indicates that 40,000 have been infected and 198 deaths have occurred.

The most affected areas are the Departments of Ancash, Piura, La Libertad and Metropolitan Lima.

Case details for these and other areas are as follows:

	CASES	HOSPITALISED	DEATHS
Lima and Callao	7,878	1,515	41
Ancash	8,393	2,399	29
Piura	2,792	452	22
La Libertad	2,274	708	16
Apurimac, Arequipa, Cajamarca, Huanuco, Ica, Junin and Lambayeque	1,140	48	6
TOTAL	22,477	5,122	114

Health Ministers from 7 countries in the region have agreed to take coordinated measures to try and prevent the spread of the disease to neighbouring countries.

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INFLUENZA UPDATE No. 1/91, 7 FEBRUARY 1991, WHO NATIONAL INFLUENZA CENTRE, CSL

In contrast to 1989/90 which produced the most extensive and severe influenza seen in the Northern Hemisphere for some time, there has been relatively little influenza activity for the current winter to date. Influenza Type B has been predominant however Type A H1 or H3 viruses have been reported in some regions.

Most reports indicate only sporadic influenza activity but a few countries have reported limited outbreaks. Exceptions at this stage are Sweden which experienced high levels of H1N1 virus in Stockholm and the central part of the country during December and the USA where Influenza B activity has been increasing during January with widespread activity reported in New York State.

EUROPE

With the exception of Sweden, European countries have been reporting only sporadic influenza activity which appears to be almost exclusively due to influenza Type B.

NORTH AMERICA

Both the USA and Canada have reported sporadic cases and localised outbreaks of influenza commencing during November and December, however, activity appears to be becoming more widespread during January. Most of the infections are due to influenza Type B, however, there are some reports of Type A influenza - H1N1 in the USA and H3N2 in Canada.

ASIA

Both Korea and Japan have reported localised outbreaks of Type A H3N2 virus during December and January. Thailand reported sporadic cases of both B and H3N2 viruses during November and December. There currently appears to be little influenza activity in China or Hong Kong.

MIDDLE EAST

There has been a single report of a few cases involving H3N2 during November - December.

ANALYSIS OF VIRUS STRAINS

Type B virus isolates from Europe and the USA are reported to be most closely related to the B/Hong Kong/22/89 strain - a variant of the B/Yamagata/16/88 virus.

Information to date suggests that current H3N2 isolates mainly resemble the A/Beijing/352/89 strain as did the majority of isolates from Oceania and South Africa in the last southern winter, and H1N1 isolates are still closely related to the A/Victoria/36/87 and A/Taiwan/1/86 strains.

WHO VACCINE FORMULATION - RECOMMENDATIONS FOR 1991

The WHO recommendation for influenza vaccine formulation for the 1991/92 northern winter will be decided at a meeting in Geneva on 18 - 19 February and should be announced to the National Control Authorities and manufacturers on 20 February.

HEPATITIS C

(Reproduced with acknowledgement from Monthly Infectious Diseases Report, Royal Alexander Hospital for Children, January 1991 (15),editor D Isaacs)

In the 1970's, following the availability of serological tests for hepatitis A and B, it became apparent that most cases of post-transfusion hepatitis were not due to hepatitis A or B infection. Since no virus had been identified the disease could not be given the logical name hepatitis C; instead the highly imaginative name non-A, non-B hepatitis (NANBH) was coined. Apart from the ugliness of the name, the term NANBH came to be applied to two different clinical syndromes, a post-transfusion hepatitis and an epidemic, waterborne hepatitis. It is now clear that most cases of the former but probably not the latter are due to hepatitis C virus.

Post-transfusion non-A, non-B hepatitis has an incubation period of about 30 to 60 days. The hepatitis is mild, and may often be subclinical: three-quarters of patients have no jaundice. The diagnosis is then made on the basis of a rise in serum alanine transaminase to at least twice the upper limit of normal on at least two occasions at least one week apart. A high proportion, approximately half to two thirds, of these episodes of hepatitis result in chronic infection which is characterised by irregular rises in liver enzymes, with or without symptoms.

Studies in which blood from healthy human blood donors was given to chimpanzees and transmitted to other chimpanzees clearly showed the presence of a transmissible agent in blood and serum from some human blood donors[1]. This was felt to be the agent responsible for non-A, non-B hepatitis. Non-A, non-B hepatitis could also be transmitted by human blood products such as clotting factors and by some preparations of intravenous gammaglobulins[2]. The agent responsible was presumed to be a virus and studies suggested a small RNA virus, although there were no consistent reports of visualisation of the agent in serum or liver, the agent could not be cultured and there were no reliable serological tests.

Various groups attempted to isolate the virus responsible for post-transfusion hepatitis and develop serological tests to identify infected individuals. However, the US National Institutes of Health (NIH) had meanwhile collected a panel of sera from individuals with classic post-transfusion hepatitis who were negative for other viruses including hepatitis A, B, CMV and EBV. All candidate serological tests were submitted to the NIH panel, and many a promising test came to grief when confronted with this final arbiter.

A group working at the Chiron Corporation in California used a different approach from most other groups. They used plasma from an infected chimpanzee as a source of virus, which they pelleted by ultra-centrifugation. The nucleic acid was extracted and complementary DNA (cDNA) synthesised by reverse transcriptase. The cDNA was cloned into a bacteriophage to give a cDNA library which could be screened for expression of an antigen which would be recognised by serum from a chronically-infected subject. About a million clones were screened before one phage was found to be producing a protein that reacted with the serum. The cDNA from this clone was then used to probe the chimpanzees' serum (and its liver). Further molecular biology techniques were used to express large amounts of the protein. It was then subjected to the NIH test : sera from 26 of 34 NIH patients with post-transfusion NANBH but no control sera reacted with this protein. Interestingly, 34 (58%) of 59 sera from cases of sporadic hepatitis also reacted with the protein. The protein is called C-100 and has been used as the basis for an ELISA test to screen for hepatitis C virus (HCV) antibodies. Hepatitis C virus appears to be a flavivirus-like RNA virus, probably related to yellow fever virus.

The ELISA tests employing the C-100 antigen have been used to test sera from healthy blood donors and from high-risk groups. These are shown in Table 1 below.

Table 1. Sero-prevalence of anti-HCV antibodies as measured by C-100 ELISA

	HCV Ab positive
Healthy blood donors	0.2 - 2%
Post-transfusion non A, non B hepatitis	75 - 85%
Cryptogenic cirrhosis	50 - 70%
Intravenous drug users (IVDU)	50 - 70%
Haemophiliac (factor VIII dependent)	60 - 85%
Haemodialysis patients	5 - 20%
Homosexual men (excluding IVDU)	4 - 8%
Prostitutes (excluding IVDU)	4 - 6%

Thus HCV infection is clearly transmitted in blood and blood products and probably not sexually transmitted to any great extent. How reliable is the ELISA test for HCV antibodies? Sero-conversion after exposure takes around 6 months (range 10-52 weeks) but liver enzyme rises occur within 8 weeks. Nevertheless sero-conversion does usually occur: about 80% of patients with post-transfusion non-A non-B hepatitis sero-convert to hepatitis C[3]. Antibody persists in patients with chronic disease but may disappear after a few years in patients with acute hepatitis which resolves[4]. The main worry about the ELISA test seems to be its specificity rather than its sensitivity, i.e. if the ELISA test is used to screen donated blood then non-infected blood will be discarded rather than infected blood being included[3]. One problem is that there is no confirmatory test for HCV antibodies, although a recombinant immunoblot assay (RIBA)[3] and an assay using the polymerase chain reaction (PCR) to amplify and detect a segment of HCV genome[5] have both been suggested as possible candidates.

In some countries, including Australia, donors of blood and blood products are now being routinely screened for hepatitis C virus antibodies using the ELISA test. This will certainly miss some cases of non-A, non-B hepatitis and possibly even some people with early hepatitis C infection who have not yet sero-converted. In addition some units of blood will be falsely discarded due to false positive anti-HCV tests. In America it has been argued that discarding units from antibody positive donors may just be discarding protective antibody without substantially lowering viral load. It may certainly be that other tests such as PCR will prove more specific and more predictive of infectivity[6]. However, PCR is a technique that is so susceptible to cross contamination that false positives may easily arise and it is therefore unlikely to be useful as a universal screening test until problems due to contamination are solved. At present the wisest course is probably to use the ELISA test to screen blood donors, as is done in Australia, until further confirmatory tests have been evaluated.

Since HCV infection often becomes chronic and may progress to cirrhosis, treatment options have been explored. Treatment with interferon-alpha leads to remission in about 40% of cases, although they may relapse when treatment is stopped[7]. Ribavirin treatment is also being tried and early reports suggest a similar response rate to interferon[7].

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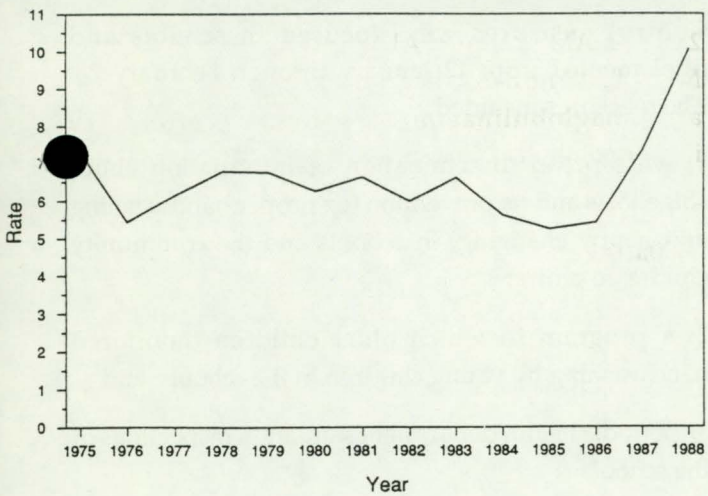
COMMUNITY OUTBREAKS OF SHIGELLOSIS - USA

(Based on MMWR 1990;39[30]:509-19)

From 1986 to 1988 (the most recent year for which national surveillance data are available) the reported isolation rate of *Shigella* in the United States increased from 5.4 to 10.1 isolates per 100,000 persons (Figure 1). In 1988, state health departments reported 22,796 isolates of *Shigella* to the Centers for Disease Control, Atlanta, the highest number since national surveillance began in 1965. In addition to the recent increase in *Shigella* isolation rates, many community-wide shigellosis outbreaks that have been difficult to control have been reported. This report describes four community outbreaks of shigellosis during 1986-1989 in which innovative public health control measures were used.

Figure 1. Shigella isolation rates*, by year - United States, 1975-1988+



* Per 100,000 population.

\+ Data from the National Shigella Surveillance System.

Kankakee County, Illinois. From October 1986 through February 1987, an outbreak of shigellosis caused by *S sonnei* occurred in Kankakee County, Illinois (population 97,800). Of 191 persons with culture-confirmed shigellosis, 70% were black and 61% were aged 1-10 years. Thirty-one percent of patients were hospitalised. Cases were clustered in low-income areas. An epidemiologic investigation did not identify common sources of exposure in the community; many patients reported having had contact with persons with culture-confirmed shigellosis or symptoms compatible with shigellosis.

To control this outbreak from 12 December to 10 January the following measures were implemented:

1) information about shigellosis and its prevention was provided to parents of all children in the school district where most of the cases occurred, to child-care centres and preschools, and through schools, churches, and the news media;

2) teachers monitored handwashing by students before lunch;

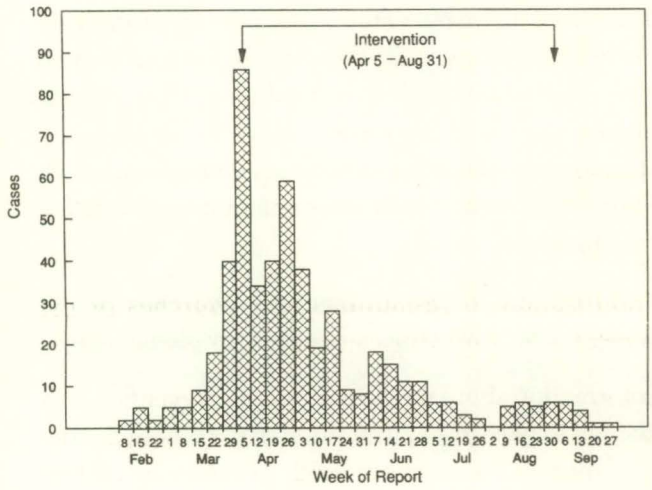
3) parents assisted in monitoring handwashing in schools in the most severely affected areas; and

4) home-prepared foods were not permitted at any school or child-care events.

Although the number of reported cases subsequently decreased, the outbreak did not end until March.

Peoria County, Illinois. From February through September 1987, a shigellosis outbreak caused by *S sonnei* occurred in Peoria County, Illinois (Figure 2) (population 181,500). Of the 513 culture-confirmed cases, 75% were in blacks and 69% were in children aged 1-10 years. Most patients resided in low-income areas. Seven percent of patients were hospitalised. Investigation did not identify a common source of exposure; most patients had a history of contact with a person who had culture-confirmed shigellosis or symptoms compatible with shigellosis.

Figure 2. Reported cases of culture-confirmed shigellosis - Peoria County, Illinois; Feburary-September 1987



During April, the following interventions were implemented:

1) child-care centre and nursery school employees were informed about shigellosis prevention;

2) school officials in the affected area ensured that warm water, soap, and disposable towels for handwashing were always avaiable for students;

3) in schools, parent and teachers instructed students on proper handwashing and monitored children for symptomss of shigellosis;

4) printed educational material about shigellosis was provided to all persons attending Women, Infants, and Children (WIC) clinics, immunisation clinics, community clinics, and hospital emergency rooms;

5) volunteers from the local Urban League and housing authority made door-to-door visits in affected neighbourhoods to identify cases and provide printed educational material;

6) religious leaders discussed the *Shigella* outbreak with their congregations, and church publications included information on shigellosis prevention; and

7) parents taught neighbourhood children how to wash their hands and monitored them for symptoms of shigellosis.

Although the number of reported cases decreased concurrently with the intervention, the outbreak continued at a lower level until September.

Orange County, New York. From 29 November 1986 to 28 February 1987, 110 culture-confirmed cases of *S sonnei* gastroenteritis were reported in residents of a religious community (population 5200) in Orange County, New York (Figure 3). Cases occurred primarily among school children 2 1/2-9 years of age; cases were evenly distributed by sex. An epidemiologic investigation did not identify a point source of exposure; spread of disease was consistent with person-to-person transmission.

Control measures were focused in schools and implemented from 12 January through February 28. The measures included:

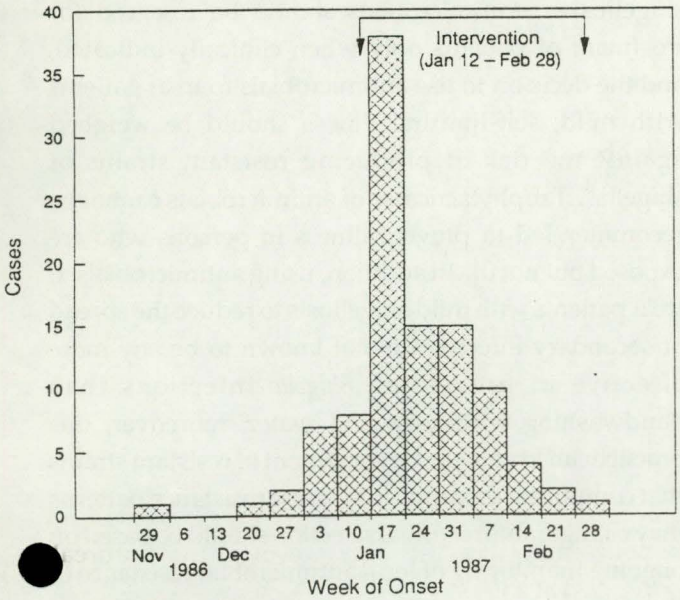
1) widespread dissemination of information about shigellosis and its prevention (eg proper handwashing and nappy changing) in schools and the community child-care centre;

2) a program in which older children monitored handwashing by young children in the schools; and

3) periodic health department sanitation inspections of the schools.

The number of reported cases of shigellosis declined concurrently with the intervention efforts.

Figure 3. Reported cases of culture-confirmed shigellosis* - Orange County, New York, November 1986 - February 1987



* Six reports did not specify date of onset and are not included here.

Caddo County, Oklahoma. From August through October 1989, 34 persons with gastroenteritis caused by *S sonnei* were identified in Caddo County, Oklahoma (Figure 4) (population approximately 32,100, including 18% Native Americans). Ninety-one percent of cases were in Native Americans. Seventy-one percent were in children and teenagers. An epidemiologic investigation did not identify a common source of infection but did suggest person-to-person transmission - 37 persons with symptoms compatible with shigellosis became ill after being exposed to a person (usually in their household) with a culture-confirmed *Shigella* infection. Clusters of cases occurred in persons residing in two Native American housing developments where children regularly played and ate snacks together.

Initial interventions implemented from 29 August to 13 September included:

1) efforts to contact families of patients to identify potential exposures and secondary cases and to provide information on hygiene and handwashing;

2) education at child-care centres and other institutions on the importance of hygiene and sanitation in preventing transmission; and

3) encouragement of physicians, hospitals, and clinical laboratories in the area to assist in identifying and reporting new cases.

The number of new cases reported initially declined, however, when new cases began to increase again, additional measures were implemented from 26 September to 4 October, including dissemination of information on shigellosis and its prevention through:

1) assistance of tribal leaders in providing information in tribal newsletters and at informal gatherings;

2) presentations at tribal senior citizen lunches;

3) house-to-house visits by public health officials and other persons in areas where clusters of cases wee identified;

4) distribution of take-home handouts to students in child-care centres and schools;

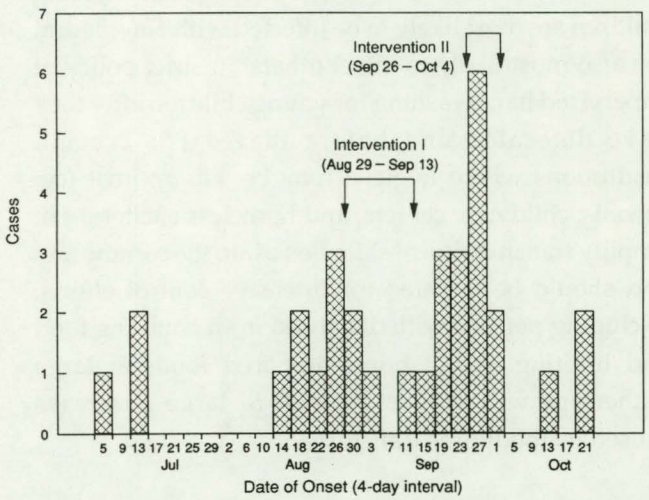
5) press releases to local newspapers and radio stations;

6) puppet shows on handwashing performed at all child-care centres, where informational posters were distributed to attendees; and

7) notification to restaurants and churches of the importance of excluding symptomatic persons from food handling duties.

The last confirmed case occurred on 21 October.

Figure 4. Reported cases of culture-confirmed shigellosis* - Caddo County, Oklahoma, July-October 1989



* Six reports did not specify date of onset and are not included here.

MMWR Editorial Note: Since 1986, the incidence of shigellosis in the United States has increased in all regions of the country. The highest isolation rates were reported among residents of counties with large proportions of low-income minority residents, among young children, and among women of childbearing age.

Communitywide outbreaks of shigellosis can be difficult to control because of the ease of person-to-person transmission among young children, high secondary attack rates, the frequently extended duration of these outbreaks, and multiple points of exposure. The impact of community interventions can be difficult to measure, however, the outbreaks described in this report suggest that effective control efforts should include the following:

1) communitywide recognition of the problem and participation in the intervention;

2) diversified and culture-specific educational efforts to promote handwashing and hygiene; and

3) supervised handwashing for children.

Because community leaders can play a key role in developing interventions and ensuring that these interventions are accepted in the community, they should be actively involved in all control efforts.

Handwashing with soap and running water may be the single most important preventive measure to interrupt transmission of shigellosis[1]. Soap and running water should be readily accessible to all persons during community outbreaks of shigellosis. Because young children are most likely to be infected with *Shigella* and are also most likely to infect others[2], a strict policy of supervised handwashing for young children after they have defecated and before they eat is crucial. Institutions where hygiene may be sub-optimal (eg, schools, child-care centers, and homeless shelters) can amplify transmission of shigellosis into the community and should be targeted for intensive control efforts. Excluding persons with diarrhoea from handling food and limiting use of home-prepared foods at large gatherings will reduce the risk of large outbreaks caused by foodborne transmission.

Antimicrobials have a limited role in the control of epidemic shigellosis and are not a substitute for hygienic measures in reducing the secondary spread of shigellosis. Antimicrobials should be reserved for treatment of patients only when clinically indicated, and the decision to use antimicrobials to treat patients with mild, self-limiting illness should be weighed against the risk of producing resistant strains of shigella[3]. Prophylactic use of antimicrobials cannot be recommended to prevent illness in persons who are exposed but not ill. In addition, using antimicrobials to treat patients with mild shigellosis to reduce the spread of secondary infections is not known to be any more effective in preventing *Shigella* infections than handwashing with soap and water, moreover, this practice can lead to the development of resistant strains that complicate therapy[4,5]. Because resistance patterns may change, antimicrobial selection should be based on ongoing monitoring of local antimicrobial resistance of *Shigella* strains.

Shigellosis outbreaks can occur at any time of the year but are most common in the summertime[6]. *Shigella* infections should be suspected in communitywide epidemics of diarrhoeal illness that disproportionately affect young children. Stool specimens should be obtained and state and local health departments informed promptly of culture-confirmed cases so that outbreaks of shigellosis can be recognized and appropriate control measures instituted.

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BACTERAEMIA - AN INTERESTING CASE

(Dr David Mitchell, Westmead Hospital, NSW)

Case report

A 43 year old man presented with an infected dog bite. He was febrile and a faint erythematous rash was noted. He quickly responded to empirical treatment with erythromicin. Swab of the bite site grew no pathogens but a gram negative fusiform rod was isolated from blood cultures after 5 days incubation. The organism produced flat, rather mucoid colonies without haemolysis on horse blood agar after 48 hrs incubation in a CO_2 enriched atmosphere. There was no growth on MacConkey agar. Positive biochemical reactions included catalase, oxidase, ONPG and alkaline phosphatase. Serum supplementation was required for fermentation tests and the organism produced acid from glucose, lactose and maltose. Gliding motility was demonstrated. These characteristics and the patients history suggested that the organism was a DF-2.

Comment

The term dysgonic fermenter-2 (DF-2) was used by the Centers for Disease Control to describe a group of previously unknown bacteria first isolated in 1961. Since the mid 1970's increasing reports of infection with this organism have been described, most in association with dog bites or other contact with dogs or other animals (including cats). The organism appears to be part of the normal oral flora of dogs and of general low pathogenicity for humans but may cause overwhelming septic shock in splenectomised or alcoholic patients. Many cases of infection go unreported because of the difficulty in isolating the organism.

In 1989, DF-2 was renamed *Capnocytophagia canimorsus*. It differs from other *Capnocytophagia* species by its positive oxidase and catalase reaction but otherwise resembles this group in morphology, microaerophilic requirements, fermentation capabilities, gliding motility and cell wall fatty acids.

Penicillin is regarded as the treatment of choice for DF-2 infection but the organism demonstrates *in vitro* susceptibility to cephalosporins, erythromicin, clindamicin and tetracyclines.

In general, significant animal bites should be treated emperically with antibiotics and this is vital if the patient is splenectomised or otherwise immunocompromised. The laboratory should be informed that the patient has a history of animal bite so that appropriate culture methods (for fastidious organisms including DF-2) can be used on the specimen.

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CDI REPORTING SCHEME

VIRUSES, CHLAMYDIAS, COXIELLAS, RICKETTSIAS AND MYCOPLASMAS REPORTS

Kokobera virus infection was serologically confirmed in a 46 year old female from Rockhampton who had not travelled outside the city. The patient presented with fever, arthralgia and a rash.

There were 21 reports of **Q fever** for the period, including 1 from November and 5 from December 1990. The majority of reports were from Queensland (11) and Victoria (8). Ages ranged from 17 to 71 years and occupational exposure details were provided for 6; 3 abattoir workers, 1 meat inspector, 1 sheep worker and 1 cattle worker.

There were 135 reports of **Ross River virus** infection for the period, with the majority (78) from Victoria (Fairfield Hospital). The Victorian cases appear to have been distributed throughout the State and it is notable that few were from Gippsland (there were about 280 reported cases of epidemic polyarthritis from the Gippsland Lakes region for the 1988/89 summer). As in previous years a significant number of cases were from the Murray Valley (about 20) and about 10 were from Melbourne. Tracebacks to determine the geographic places of infection would provide a more complete epidemioligical description of this disease.

Twenty-two (22) of the reports were from South Australia, 17 from Queensland, 10 from Western Australia and 8 from New South Wales.

A separate report from the Northern Territory Department of Health and Community Services mentions that they have identified 190 cases of epidemic polyarthritis so far this year. Tracebacks on 108 cases revealed that the majority were from the greater Darwin area (urban and rural). Small numbers of cases have been recorded from most inland centres. The 'top end' cases are probably due to the above average wet season rains this year.

Measles in Darwin

The Northern Territory Department of Health and Community Services has reported a further 35 cases of **measles** in Darwin (see CDI Vol 15/No. 4 pg 65).

A second high school, with 7 of the total of 62 cases (as of 4 March), has now become involved in the outbreak. Each of the 7 cases were found to have been in contact with students from the high school originally affected (where 41 cases have now been detected).

The Department has advised health centres and general practitioners to lower the immunisation age to 6 months and to ensure that these infants receive booster vaccinations at age 15 months.

The Department is also actively promoting vaccination for students likely to be at risk in the two high schools.

An attempt to gauge the immunisation status of students in the high school originally affected revealed that satisfactory proof of measles vaccination could only be supplied for 15% of approximately 1200 students. Measles vaccination is being offered to all students that are either unimmunised or whose immunisation status is uncertain. Offers of immunisation have been made through the media and letters to parents. Acceptance rates at the high school after two rounds, have been 50% (first round) and 30% (of total target, second round). The Department is currently planning a third round which will be based primarily on telephone calls to parents.

Similar offers to 'at risk' students in the second high school have had a 32% initial acceptance rate (total of about 1400 students).

CDI Editorial Comment: In the 20 years from 1966-1985, measles caused more deaths in Australia (179) than diphtheria (13), tetanus (113), pertussis (38) and poliomyelitis (4) combined[1].

Vaccination with live attenuated measles virus is the primary preventive measure indicated for all individuals susceptible to measles, unless specifically contraindicated. The NH&MRC recommends that the vaccine should be offered routinely at the age of 12 months when a seroconversion rate of 95 percent is expected[1]. Despite this recommendation, the uptake of measles vaccine in Australia has been poor.

This most recent epidemic in Darwin follows a similar outbreak reported in the Port Stephens area of NSW last August[2]. In both epidemics a significant number of previously vaccinated subjects developed clinical measles. A similar trend has been noted in the United States, where an increasing proportion of recent cases has occurred in previously vaccinated school students[3].

This paradox may be explained in at least two ways. It is possible that measles is so highly contagious that measles elimination will require that both vaccine efficacy and vaccination coverage be higher than previously estimated[3]. There is also the possibility of secondary measles vaccine failure, suggesting that measles vaccine efficacy may be sufficiently low to allow sustained outbreaks to occur in highly vaccinated school populations[4].

The spread of measles can be contained by vaccinating susceptible children who have been in contact with an infected case within 72 hours. If there is doubt about a child's measles immunity the vaccine should be given, since there are no ill-effects from vaccinating those already seropositive. Normal immunoglobulin (human) is available for individuals for whom the live vaccine is contraindicated[1].

If a young infant is exposed or is likely to be exposed to measles, normal immunoglobulin can be used either to abort or modify the attack. When this is done, it is necessary that vaccination be given at 12-15 months of age (but not within three months of the immunoglobulin) to ensure active immunity.

An alternative approach, followed in Darwin, has been to lower the age of vaccination to 6 months. However, if a child has been vaccinated before the age of 12 months, he/she should be followed up and revaccinated at the age of 15 months[1].

In Darwin offers of immunisation have been made throulgh the media and by letters to parents. The acceptance rates have been strikingly low and they highlight a fundamental problem with measles control in Australia - that of unsatisfactory vaccine coverage rates.

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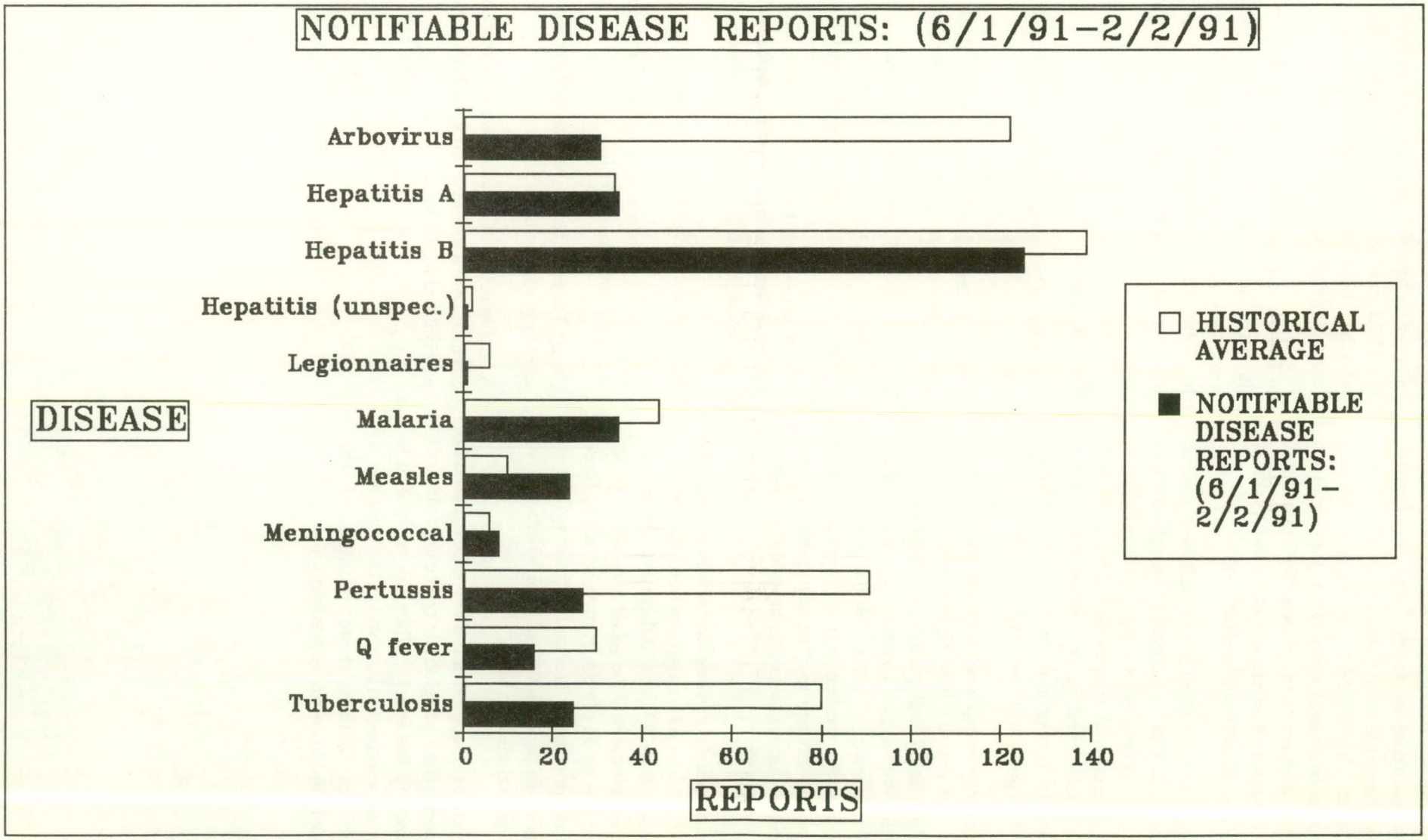
NON-VIRAL PATHOGEN REPORTS

Group B streptococcus was isolated from the placenta of a child that died at birth.

Group B streptococcus was isolated in blood culture of an elderly man with cellulitis.

A fatal infection of *Pseudemonas aeruginosa* in a 29 year old man with a history of cholecyshitis. The organism was isolated from blood culture.


NOTIFIABLE DISEASE REPORTS: (6/1/91-2/2/91)
Arbovirus
Hepatitis A
Hepatitis B
Hepatitis (unspec.)
Legionnaires
Malaria
Measles
Meningococcal
Pertussis
Q fever
Tuberculosis
DISEASE
0
20
40
60
80
100
120
140
REPORTS
HISTORICAL AVERAGE
NOTIFIABLE DISEASE REPORTS: (6/1/91-2/2/91)

NOTIFIABLE DISEASE REPORTS: REPORTING PERIOD 1 (6/1/91 - 19 /1/91) AND 2 (20 /1/91 - 2/2/91)

DISEASES	ACT	NSW*	NT	QLD	SA	TAS	VIC	WA	TOTAL
Arbovirus unsec.	0	0	0	0	31	0	0	0	31
Ross River	0	11	64	128	0	0	14	0	217
Dengue	0	NN	0	0	0	0	0	0	0
Brucellosis	0	0	0	0	0	0	0	0	0
Campylobacter	1	5	21	180	67	0	11	0	285
Chancroid	0	0	0	0	NN	NN	NN	0	0
Chlamydia	3	0	20	196	37	0	NN	0	256
Cholera	0	0	0	0	0	0	0	0	0
Diphtheria	0	0	0	0	0	0	0	0	0
Donovanosis	0	0	0	0	NN	0	NN	0	0
Gonococcal	1	0	24	33	10	0	0	0	68
HIB	0	NN	0	6	0	0	8	0	14
HIV	1	0	0	0	1	0	0	0	2
Hydatid	0	0	0	0	0	0	0	0	0
Legionnaires	NN	0	0	0	1	0	0	0	1
Leprosy	0	0	0	0	0	0	0	0	0
Leptospirosis	0	0	0	0	0	0	1	0	1
Listeriosis	0	NN	0	1	0	0	2	0	3
LGV	0	0	NN	0	NN	NN	NN	NN	0
Malaria	4	0	2	21	5	0	3	0	35
Measles	0	0	0	10	0	0	14	0	24
Meningococcal	0	0	2	2	1	NN	3	0	8
Ornithosis	0	0	0	0	0	0	0	0	0
Pertussis	0	1	0	15	0	0	11	0	27
Plague	0	0	0	0	0	0	0	0	0
Poliomyelitis	0	0	0	0	0	0	0	0	0

NOTIFIABLE DISEASE REPORTS: REPORTING PERIOD 1 (6/1/91 - 19/1/91) AND 2 (20/1/91 - 2/2/91) CONTINUED

DISEASES	ACT	NSW*	NT	QLD	SA	TAS	VIC	WA	TOTAL
Q fever	NN	0	0	16	0	NN	0	0	16
Rabies	0	0	0	0	0	0	0	0	0
Rubella	0	0	1	15	5	0	1	0	22
Salmonella	0	6	23	121	44	0	9	0	203
Shigella	1	1	14	9	8	0	0	0	33
Syphilis	0	0	20	34	1	0	0	0	55
Tetanus	0	0	0	0	0	0	0	0	0
TB	1	0	0	1	10	0	13	0	25
Typhoid	0	1	0	0	0	0	1	0	2
VHF	0	0	0	0	0	0	0	0	0
Viral Hep. unspec.	0	0	0	0	0	NN	1	NN	1
Hep. A	0	1	1	22	11	0	0	0	35
Hep. B	0	3	0	111	3	0	8	0	125
Hep. C	0	0	0	68	0	0	39	0	107
Yellow Fever	0	0	0	0	0	0	0	0	0

* Data for January 1991

NN Not notifiable

AUSTRALIA - COMMUNICABLE DISEASES INTELLIGENCE

VIRAL IDENTIFICATIONS FROM CONTRIBUTING LABORATORIES - FIRST PART
BASED ON DATE OF REPORTING

PERIOD 13/02/91 TO 26/02/91

CODE 018 - MICROBIOLOGICAL DIAGNOSTIC UNIT, UNIVERSITY OF MELBOURNE (VIC)
CODE 019 - FAIRFIELD HOSPITAL, MELBOURNE (VIC)
CODE 065 - STATE HEALTH LABORATORY SERVICES, PERTH (WA)
CODE 066 - PRINCESS MARGARET HOSPITAL, PERTH (WA)
CODE 110 - INSTITUTE OF MEDICAL & VETERINARY SCIENCE, ADELAIDE (SA)
CODE 111 - ROYAL CHIDLDRENS HOSPITAL, MELBOURNE (VIC)
CODE 112 - INSTITUTE OF CLINICAL PATHOLOGY & MEDICAL RESEARCH, WESTMEAD (NSW)
CODE 113 - PRINCE HENRY/PRINCE OF WALES HOSPITALS, SYDNEY (NSW)
CODE 114 - ROYAL ALEXANDRA HOSPITAL FOR CHILDREN, CAMPERDOWN (NSW)
CODE 115 - STATE HEALTH LABORATORY, BRISBANE (QLD)
CODE 116 - WODEN VALLEY HOSPITAL, GARRAN (ACT)
CODE 400 - DR TB LYNCH, PATHOLOGIST, ROCKHAMPTON (QLD)
CODE DSH - DIAGNOSTIC SERVICES LTD, HOBART (TAS)
CODE RHH - ROYAL HOBART HOSPITAL (TAS)
CODE TPL - TOOWOOMBA PATHOLOGY LABORATORY (QLD)

		018	019	065	066	110	111	112	113	TOTAL
0100	ADENOVIRUS NOT TYPED	0	0	1	7	2	7	4	3	24
0101	ADENOVIRUS TYPE 1	0	2	0	0	3	2	1	0	8
0102	ADENOVIRUS TYPE 2	0	4	0	0	0	8	3	0	15
0103	ADENOVIRUS TYPE 3	0	4	1	0	2	3	1	0	11
0104	ADENOVIRUS TYPE 4	0	1	0	0	0	0	0	0	1
0107	ADENOVIRUS TYPE 7	0	1	0	0	0	0	0	0	1
0108	ADENOVIRUS TYPE 8	0	3	0	0	1	0	0	0	4
0109	ADENOVIRUS TYPE 9	0	1	0	0	0	0	0	0	1
0110	ADENOVIRUS TYPE 10	0	0	0	0	1	0	0	0	1
0111	ADENOVIRUS TYPE 11	0	3	0	0	1	0	4	0	8
0113	ADENOVIRUS TYPE 13	0	0	0	0	1	0	0	0	1
0116	ADENOVIRUS TYPE 16	0	0	0	0	1	0	0	0	1
0122	ADENOVIRUS TYPE 22	0	1	0	0	0	0	0	0	1
0126	ADENOVIRUS TYPE 26	0	2	0	0	0	0	0	0	2
0137	ADENOVIRUS TYPE 37	0	0	0	0	2	0	0	0	2
0199	ADENOVIRUS TYPING PENDING	0	0	0	0	0	4	0	0	4
0201	INFLUENZA A VIRUS	0	0	0	0	1	0	0	0	1
0203	INFLUENZA B VIRUS	0	0	0	0	4	0	0	0	4
0301	PARAINFLUENZA VIRUS TYPE 1	0	0	0	3	2	0	0	0	5
0302	PARAINFLUENZA VIRUS TYPE 2	0	0	0	2	6	0	0	0	8
0303	PARAINFLUENZA VIRUS TYPE 3	0	2	0	3	14	3	1	1	24
0400	RESPIRATORY SYNCYTIAL VIRUS (R	0	0	0	0	4	1	1	0	6
0500	RHINOVIRUS (ALL TYPES)	0	8	0	0	6	3	0	0	17
0600	MYCOPLASMA PNEUMONIAE	0	3	1	0	4	0	6	0	14
0700	ORNITHOSIS-PSITTACOSIS	0	1	0	0	2	0	0	0	3
0809	COXSACKIEVIRUS A9	0	0	0	0	0	0	4	1	5
0902	COXSACKIEVIRUS B2	0	1	0	0	2	0	0	0	3
0903	COXSACKIEVIRUS B3	0	0	0	0	0	0	1	1	2
0904	COXSACKIEVIRUS B4	0	3	0	0	0	4	0	3	10
0905	COXSACKIEVIRUS B5	0	0	0	0	0	0	2	0	2
1001	ECHOVIRUS TYPE 1	0	0	0	0	0	0	1	0	1
1006	ECHOVIRUS TYPE 6	0	0	0	0	1	0	0	0	1
1014	ECHOVIRUS TYPE 14	0	1	0	0	0	0	1	0	2
1021	ECHOVIRUS TYPE 21	0	1	0	0	0	0	0	0	1
1022	ECHOVIRUS TYPE 22	0	0	0	0	0	0	1	0	1
1100	POLIOVIRUS NOT TYPED	0	0	0	0	0	3	0	2	5
1101	POLIOVIRUS TYPE 1	0	0	0	0	1	0	1	0	2
1102	POLIOVIRUS TYPE 2	0	1	0	0	0	0	1	0	2
1300	HERPES VIRUS GROUP - NOT TYPED	0	1	0	0	0	0	0	0	1
1301	HERPES SIMPLEX VIRUS - NOT TYP	0	0	5	12	1	0	22	0	40
1302	EPSTEIN-BARR VIRUS (EB VIRUS)	0	4	9	0	47	5	6	0	71
1303	VARICELLA-ZOSTER VIRUS	0	11	7	0	2	0	3	0	23
1306	HERPES SIMPLEX TYPE 1	0	86	43	0	50	2	6	13	200
1307	HERPES SIMPLEX TYPE 2	3	68	50	0	34	0	16	22	193
1399	HERPES VIRUS TYPING PENDING	0	4	0	0	0	2	0	0	6
1401	COXIELLA BURNETII	0	8	0	0	1	0	1	0	10
1502	PICORNIA VIRUS - NOT TYPED = E	0	0	3	0	0	0	1	9	13
1521	MEASLES VIRUS	0	11	2	0	0	1	0	0	14
1522	RUBELLA VIRUS	0	3	0	0	1	0	1	0	5
1532	HEPATITIS B ANTIGEN	0	27	13	0	16	0	23	15	94
1535	HEPATITIS A ANTIBODY	0	3	6	0	9	0	5	0	23
1536	HEPATITIS C VIRUS	0	0	13	0	0	0	0	0	13
1541	CHLAMYDIA TRACHOMATIS - UNSPEC	0	0	32	0	8	0	19	0	59
1551	NDV - NEWCASTLE DISEASE VIRUS	0	1	0	0	0	0	0	0	1
1556	CMV - CYTOMEGALOVIRUS	0	92	3	5	11	3	9	3	126
1562	REOVIRUS (ALL TYPES)	0	0	0	0	0	0	1	0	1
1563	CORONAVIRUS	0	1	0	0	0	0	1	0	2
1564	ROTAVIRUS	0	1	2	6	0	1	0	2	12
1565	CALICI VIRUS	0	0	0	0	0	0	1	0	1
1571	ENTEROVIRUS TYPE 71 (BCR)	0	1	0	0	0	0	0	0	1
1599	ENTEROVIRUS TYPING PENDING	0	0	0	0	0	4	0	26	30
9902	POXVIRUS GROUP NOT TYPED	0	1	0	0	0	0	0	0	1
9906	BARMAH FOREST VIRUS	0	0	0	0	0	0	0	1	1
9981	DENGUE TYPE 1	0	0	1	0	0	0	0	0	1
9992	ROSS RIVER VIRUS	0	78	17	0	22	0	1	7	125
9993	ASTROVIRUS	0	0	0	0	0	0	1	0	1
9994	SMALL VIRUS (LIKE) PARTICLE	0	0	0	0	0	0	1	0	1
TOTAL		3	444	209	38	263	56	151	109	1273

AUSTRALIA - COMMUNICABLE DISEASES INTELLIGENCE

VIRAL IDENTIFICATIONS FROM CONTRIBUTING LABORATORIES - PART 2
BASED ON DATE OF REPORTING

PERIOD 13/02/91 TO 26/02/91

CODE 018 - MICROBIOLOGICAL DIAGNOSTIC UNIT, UNIVERSITY OF MELBOURNE (VIC)
CODE 019 - FAIRFIELD HOSPITAL, MELBOURNE (VIC)
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CODE DSH - DIAGNOSTIC SERVICES LTD, HOBART (TAS)
CODE RHH - ROYAL HOBART HOSPITAL (TAS)
CODE TPL - TOOWOOMBA PATHOLOGY LABORATORY (QLD)

		114	115	116	400	DSH	RHH	TPL	TOTAL
0100	ADENOVIRUS NOT TYPED	0	6	0	0	0	0	0	6
0101	ADENOVIRUS TYPE 1	0	0	0	0	0	1	0	1
0102	ADENOVIRUS TYPE 2	0	0	0	0	0	2	0	2
0103	ADENOVIRUS TYPE 3	0	0	1	0	0	1	0	2
0199	ADENOVIRUS TYPING PENDING	2	0	0	0	0	0	0	2
0201	INFLUENZA A VIRUS	0	3	0	0	0	0	0	3
0302	PARAINFLUENZA VIRUS TYPE 2	0	1	0	0	0	0	0	1
0303	PARAINFLUENZA VIRUS TYPE 3	1	3	0	0	0	0	0	4
0400	RESPIRATORY SYNCYTIAL VIRUS (R	0	0	0	0	0	1	0	1
0500	RHINOVIRUS (ALL TYPES)	0	1	0	0	0	0	0	1
0600	MYCOPLASMA PNEUMONIAE	0	1	0	0	0	0	0	1
0700	ORNITHOSIS-PSITTACOSIS	0	0	1	0	0	0	0	1
0809	COXSACKIEVIRUS A9	1	0	6	0	0	0	0	7
0902	COXSACKIEVIRUS B2	1	0	0	0	0	0	0	1
0904	COXSACKIEVIRUS B4	1	0	0	0	0	0	0	1
0905	COXSACKIEVIRUS B5	1	0	0	0	0	0	0	1
1002	ECHOVIRUS TYPE 2	1	0	0	0	0	0	0	1
1022	ECHOVIRUS TYPE 22	2	0	0	0	0	0	0	2
1301	HERPES SIMPLEX VIRUS - NOT TYP	7	3	11	2	0	0	0	23
1302	EPSTEIN-BARR VIRUS (EB VIRUS)	0	24	0	54	0	0	0	78
1303	VARICELLA-ZOSTER VIRUS	0	7	3	0	0	1	0	11
1306	HERPES SIMPLEX TYPE 1	0	39	0	0	0	0	0	39
1307	HERPES SIMPLEX TYPE 2	0	35	0	0	0	1	1	37
1401	COXIELLA BURNETII	0	10	0	1	0	0	0	11
1502	PICORNIA VIRUS - NOT TYPED = E	0	5	0	0	0	0	0	5
1514	MOLLUSCUM CONTAGIOSUM	0	0	0	8	0	0	0	8
1522	RUBELLA VIRUS	0	2	0	2	0	0	0	4
1532	HEPATITIS B ANTIGEN	1	29	5	3	0	2	0	40
1535	HEPATITIS A ANTIBODY	0	1	0	0	0	0	0	1
1541	CHLAMYDIA TRACHOMATIS - UNSPEC	0	17	5	0	0	4	14	40
1542	CHLAMYDIA TRACHOMATIS - A-K	1	0	0	1	0	0	1	3
1543	CHLAMYDIA L1-L3 - (LGV TYPE)	0	0	0	2	0	0	0	2
1556	CMV - CYTOMEGALOVIRUS	1	13	0	24	0	1	0	39
1564	ROTAVIRUS	2	0	0	45	2	1	1	51
1599	ENTEROVIRUS TYPING PENDING	1	0	0	0	0	0	0	1
9906	BARMAH FOREST VIRUS	0	3	0	0	0	0	0	3
9981	DENGUE TYPE 1	0	1	0	0	0	0	0	1
9992	ROSS RIVER VIRUS	0	13	0	6	0	0	0	19
9994	SMALL VIRUS (LIKE) PARTICLE	1	0	0	0	0	0	0	1
9998	ARBOVIRUS GROUP B.(UNSPECIFIED	0	2	0	0	0	0	0	2
TOTAL		24	219	32	148	2	15	17	457

AUSTRALIA - COMMUNICABLE DISEASES INTELLIGENCE

VIRAL IDENTIFICATIONS FROM CONTRIBUTING LABORATORIES BY STATE OF CONTRIBUTING LABORATORY

PERIOD 13/02/91 TO 26/02/91

NSW: ICPMR; PHH/POW; RACH; ST GEORGE HOSP, KOGARAH; ROYAL NEWCASTLE HOSP.
VIC: FAIRFIELD; RCH; MDU, UNI MELB.
QLD: STATE LAB, BRIS; TOOWOOMBA PATH LAB; ROYAL BRIS HOSP; DR TB LYNCH, PATHOLOGIST, ROCKHAMPTON.
WA: STATE LAB, PERTH; PMH.
SA: IIMVS.
TAS: ROYAL HOBART HOSP; DIAGNOSTIC SERVICES, LAUNCESTON; LAUNCESTON GEN HOSP; DIAGNOSTIC SERVICES, HOBART; HOBART PATH; MERSEY GEN HOSP, LATROBE.
ACT: WVH.

		NSW	VIC	QLD	WA	SA	TAS	ACT	TOTAL
0100	ADENOVIRUS NOT TYPED	7	7	6	8	2	0	0	30
0101	ADENOVIRUS TYPE 1	1	4	0	0	3	1	0	9
0102	ADENOVIRUS TYPE 2	3	12	0	0	0	2	0	17
0103	ADENOVIRUS TYPE 3	1	7	0	1	2	1	1	13
0104	ADENOVIRUS TYPE 4	0	1	0	0	0	0	0	1
0107	ADENOVIRUS TYPE 7	0	1	0	0	0	0	0	1
0108	ADENOVIRUS TYPE 8	0	3	0	0	1	0	0	4
0109	ADENOVIRUS TYPE 9	0	1	0	0	0	0	0	1
0110	ADENOVIRUS TYPE 10	0	0	0	0	1	0	0	1
0111	ADENOVIRUS TYPE 11	4	3	0	0	1	0	0	8
0113	ADENOVIRUS TYPE 13	0	0	0	0	1	0	0	1
0116	ADENOVIRUS TYPE 16	0	0	0	0	1	0	0	1
0122	ADENOVIRUS TYPE 22	0	1	0	0	0	0	0	1
0126	ADENOVIRUS TYPE 26	0	2	0	0	0	0	0	2
0137	ADENOVIRUS TYPE 37	0	0	0	0	2	0	0	2
0199	ADENOVIRUS TYPING PENDING	2	4	0	0	0	0	0	6
0201	INFLUENZA A VIRUS	0	0	3	0	1	0	0	4
0203	INFLUENZA B VIRUS	0	0	0	0	4	0	0	4
0301	PARAINFLUENZA VIRUS TYPE 1	0	0	0	3	2	0	0	5
0302	PARAINFLUENZA VIRUS TYPE 2	0	0	1	2	6	0	0	9
0303	PARAINFLUENZA VIRUS TYPE 3	3	5	3	3	14	0	0	28
0400	RESPIRATORY SYNCYTIAL VIRUS (R	1	1	0	0	4	1	0	7
0500	RHINOVIRUS (ALL TYPES)	0	11	1	0	6	0	0	18
0600	MYCOPLASMA PNEUMONIAE	6	3	1	1	4	0	0	15
0700	ORNITHOSIS-PSITTACOSIS	0	1	0	0	2	0	1	4
0809	COXSACKIEVIRUS A9	6	0	0	0	0	0	6	12
0902	COXSACKIEVIRUS B2	1	1	0	0	2	0	0	4
0903	COXSACKIEVIRUS B3	2	0	0	0	0	0	0	2
0904	COXSACKIEVIRUS B4	4	7	0	0	0	0	0	11
0905	COXSACKIEVIRUS B5	3	0	0	0	0	0	0	3
1001	ECHOVIRUS TYPE 1	1	0	0	0	0	0	0	1
1002	ECHOVIRUS TYPE 2	1	0	0	0	0	0	0	1
1006	ECHOVIRUS TYPE 6	0	0	0	0	1	0	0	1
1014	ECHOVIRUS TYPE 14	1	1	0	0	0	0	0	2
1021	ECHOVIRUS TYPE 21	0	1	0	0	0	0	0	1
1022	ECHOVIRUS TYPE 22	3	0	0	0	0	0	0	3
1100	POLIOVIRUS NOT TYPED	2	3	0	0	0	0	0	5
1101	POLIOVIRUS TYPE 1	1	0	0	0	1	0	0	2
1102	POLIOVIRUS TYPE 2	1	1	0	0	0	0	0	2
1300	HERPES VIRUS GROUP - NOT TYPED	0	1	0	0	0	0	0	1
1301	HERPES SIMPLEX VIRUS - NOT TYP	29	0	5	17	1	0	11	63
1302	EPSTEIN-BARR VIRUS (EB VIRUS)	6	9	78	9	47	0	0	149
1303	VARICELLA-ZOSTER VIRUS	3	11	7	7	2	1	3	34
1306	HERPES SIMPLEX TYPE 1	19	88	39	43	50	0	0	239
1307	HERPES SIMPLEX TYPE 2	38	71	36	50	34	1	0	230
1399	HERPES VIRUS TYPING PENDING	0	6	0	0	0	0	0	6
1401	COXIELLA BURNETII	1	8	11	0	1	0	0	21
1502	PICORNIA VIRUS - NOT TYPED = E	10	0	5	3	0	0	0	18
1514	MOLLUSCUM CONTAGIOSUM	0	0	8	0	0	0	0	8
1521	MEASLES VIRUS	0	12	0	2	0	0	0	14
1522	RUBELLA VIRUS	1	3	4	0	1	0	0	9
1532	HEPATITIS B ANTIGEN	39	27	32	13	16	2	5	134
1535	HEPATITIS A ANTIBODY	5	3	1	6	9	0	0	24
1536	HEPATITIS C VIRUS	0	0	0	13	0	0	0	13
1541	CHLAMYDIA TRACHOMATIS - UNSPEC	19	0	31	32	8	4	5	99
1542	CHLAMYDIA TRACHOMATIS - A-K	1	0	2	0	0	0	0	3
1543	CHLAMYDIA L1-L3 - (LGV TYPE)	0	0	2	0	0	0	0	2
1551	NDV - NEWCASTLE DISEASE VIRUS	0	1	0	0	0	0	0	1
1556	CMV - CYTOMEGALOVIRUS	13	95	37	8	11	1	0	165
1562	REOVIRUS (ALL TYPES)	1	0	0	0	0	0	0	1
1563	CORONAVIRUS	1	1	0	0	0	0	0	2
1564	ROTAVIRUS	4	2	46	8	0	3	0	63
1565	CALICI VIRUS	1	0	0	0	0	0	0	1
1571	ENTEROVIRUS TYPE 71 (BCR)	0	1	0	0	0	0	0	1
1599	ENTEROVIRUS TYPING PENDING	27	4	0	0	0	0	0	31
9902	POXVIRUS GROUP NOT TYPED	0	1	0	0	0	0	0	1
9906	BARMAH FOREST VIRUS	1	0	3	0	0	0	0	4
9981	DENGUE TYPE 1	0	0	1	1	0	0	0	2
9992	ROSS RIVER VIRUS	8	78	19	17	22	0	0	144
9993	ASTROVIRUS	1	0	0	0	0	0	0	1
9994	SMALL VIRUS (LIKE) PARTICLE	2	0	0	0	0	0	0	2
9998	ARBOVIRUS GROUP B.(UNSPECIFIED	0	0	2	0	0	0	0	2
TOTAL		284	503	384	247	263	17	32	1730

NOTE: DIRECT COMPARISION BETWEEN STATES IS NOT POSSIBLE SINCE:
- SOME STATES HAVE MORE THAN ONE CONTRIBUTING LABORATORY; AND
- INTERSTATE REFERRRALS OCCUR REGULARLY.

AUSTRALIA - COMMUNICABLE DISEASES INTELLIGENCE

VIRAL IDENTIFICATIONS BY CLINICAL INFORMATION TABLE 1

PERIOD 13/02/91 TO 26/02/91

1. CODE 00, 99 ..... - NO ILL OR DATA
2. CODE 01, 02, 11, 12 - RESPIRATORY
3. CODE E3 ..... - ENCEPHALITIS
4. CODE M3 ..... - MENINGITIS
5. CODE 04 ..... - PARALYSIS
6. CODE 05, 13 ..... - CNS OTHER UNSPEC
7. CODE 07, 49 - GASTRO INTESTINAL
8. CODE 17, 47 - HEPATIC
9. CODE 19 ... - CVS
10. CODE 89 ... - URINARY TRACCT
11. CODE 06 ... - SKIN MUCOUS

		1	2	3	4	5	6	7	8	9	10	11	TOTAL
0100	ADENOVIRUS NOT TYPED	0	8	0	0	0	0	18	0	0	0	0	26
0101	ADENOVIRUS TYPE 1	1	5	0	0	0	0	2	0	0	0	0	8
0102	ADENOVIRUS TYPE 2	1	8	0	1	1	0	4	0	0	0	1	16
0103	ADENOVIRUS TYPE 3	0	4	0	0	0	0	1	0	0	0	0	5
0104	ADENOVIRUS TYPE 4	0	0	0	0	0	0	1	0	0	0	0	1
0109	ADENOVIRUS TYPE 9	0	0	0	0	0	0	1	0	0	0	0	1
0110	ADENOVIRUS TYPE 10	0	0	0	0	0	0	1	0	0	0	0	1
0111	ADENOVIRUS TYPE 11	0	0	0	0	0	0	2	0	0	0	0	2
0113	ADENOVIRUS TYPE 13	0	0	0	0	0	0	1	0	0	0	0	1
0116	ADENOVIRUS TYPE 16	0	0	0	0	0	0	1	0	0	0	0	1
0122	ADENOVIRUS TYPE 22	0	0	0	0	0	0	1	0	0	0	0	1
0126	ADENOVIRUS TYPE 26	0	0	0	0	0	0	2	0	0	0	0	2
0199	ADENOVIRUS TYPING PENDING	0	3	0	0	0	0	1	0	0	0	0	4
0201	INFLUENZA A VIRUS	0	2	1	1	0	0	0	0	0	0	0	4
0203	INFLUENZA B VIRUS	0	3	0	0	0	0	0	0	0	0	0	3
0301	PARAINFLUENZA VIRUS TYPE 1	0	5	0	0	0	0	0	0	0	0	0	5
0302	PARAINFLUENZA VIRUS TYPE 2	0	7	0	0	0	0	0	0	0	0	0	7
0303	PARAINFLUENZA VIRUS TYPE 3	3	21	1	0	0	0	0	0	0	0	0	25
0400	RESPIRATORY SYNCYTIAL VIRUS (R	0	7	0	0	0	0	0	0	0	0	0	7
0500	RHINOVIRUS (ALL TYPES)	0	10	0	0	0	0	2	0	0	0	0	12
0600	MYCOPLASMA PNEUMONIAE	2	11	1	0	0	0	0	0	0	0	0	14
0700	ORNITHOSIS-PSITTACOSIS	0	3	0	0	0	0	0	0	0	0	0	3
0809	COXSACKIEVIRUS A9	1	4	0	1	0	0	3	0	0	0	1	10
0902	COXSACKIEVIRUS B2	0	1	1	1	0	0	0	0	0	0	0	3
0903	COXSACKIEVIRUS B3	0	0	0	0	0	0	1	0	0	0	0	1
0904	COXSACKIEVIRUS B4	0	4	0	3	0	0	0	0	0	0	1	8
0905	COXSACKIEVIRUS B5	1	0	0	0	0	0	2	0	0	0	0	3
1001	ECHOVIRUS TYPE 1	1	0	0	0	0	0	0	0	0	0	0	1
1006	ECHOVIRUS TYPE 6	0	0	0	0	0	0	1	0	0	0	0	1
1014	ECHOVIRUS TYPE 14	0	0	0	1	0	0	1	0	0	0	0	2
1021	ECHOVIRUS TYPE 21	0	0	0	1	0	0	0	0	0	0	0	1
1022	ECHOVIPUS TYPE 22	2	0	0	0	0	0	1	0	0	0	0	3
1100	POLIOVIRUS NOT TYPED	0	2	0	0	1	0	1	0	0	0	0	4
1101	POLIOVIRUS TYPE 1	0	0	0	0	0	1	0	0	0	0	0	1
1102	POLIOVIRUS TYPE 2	1	0	0	0	0	0	0	0	0	0	0	1
1300	HERPES VIRUS GROUP - NOT TYPED	0	0	0	0	0	0	0	0	0	0	1	1
1301	HERPES SIMPLEX VIRUS - NOT TYP	5	3	0	1	0	0	0	0	0	0	32	41
1302	EPSTEIN-BARR VIRUS (EB VIRUS)	37	27	0	0	0	0	2	6	0	0	0	72
1303	VARICELLA-ZOSTER VIRUS	5	0	0	0	0	1	1	0	0	0	24	31
1306	HERPES SIMPLEX TYPE 1	7	11	0	0	0	1	2	0	0	0	158	179
1307	HERPES SIMPLEX TYPE 2	2	1	1	0	0	0	0	1	0	0	80	85
1399	HERPES VIRUS TYPING PENDING	0	0	0	0	0	0	1	0	0	0	2	3
1401	COXIELLA BURNETII	4	1	0	0	0	0	0	1	1	0	0	7
1502	PICORNIA VIRUS - NOT TYPED = E	1	4	0	2	0	1	6	0	0	0	3	17
1514	MOLLUSCUM CONTAGIOSUM	2	0	0	0	0	0	0	0	0	0	2	4
1521	MEASLES VIRUS	2	0	0	0	0	0	0	0	0	0	11	13
1522	RUBELLA VIRUS	3	1	0	0	0	0	0	0	0	0	1	5
1532	HEPATITIS B ANTIGEN	70	0	0	0	0	0	0	55	0	0	0	125
1535	HEPATITIS A ANTIBODY	7	0	0	0	0	0	0	13	2	0	0	22
1536	HEPATITIS C VIRUS	4	0	0	0	0	0	0	9	0	0	0	13
1541	CHLAMYDIA TRACHOMATIS - UNSPEC	10	1	0	0	0	0	0	0	0	0	1	12
1542	CHLAMYDIA TRACHOMATIS - A-K	0	1	0	0	0	0	0	0	0	1	0	2
1551	NDV - NEWCASTLE DISEASE VIRUS	0	1	0	0	0	0	0	0	0	0	0	1
1556	CMV - CYTOMEGALOVIRUS	23	15	0	0	0	5	2	9	8	6	0	68
1562	REOVIRUS (ALL TYPES)	0	0	0	0	0	0	1	0	0	0	0	1
1563	CORONAVIRUS	0	0	0	0	0	0	2	0	0	0	0	2
1564	ROTAVIRUS	15	0	0	0	0	0	47	0	0	0	0	62
1565	CALICI VIRUS	0	0	0	0	0	0	1	0	0	0	0	1
1571	ENTEROVIRUS TYPE 71 (BCR)	0	0	0	1	0	0	0	0	0	0	0	1
1599	ENTEROVIRUS TYPING PENDING	0	0	1	1	0	0	25	0	0	0	0	27
9902	POXVIRUS GROUP NOT TYPED	0	0	0	0	0	0	0	0	0	0	1	1
9906	BARMAH FOREST VIRUS	2	0	0	0	0	0	0	0	0	0	0	2
9981	DENGUE TYPE 1	1	0	0	0	0	0	0	0	0	0	0	1
9992	ROSS RIVER VIRUS	38	0	0	0	0	0	0	0	0	0	23	61
9993	ASTROVIRUS	1	0	0	0	0	0	0	0	0	0	0	1
9994	SMALL VIRUS (LIKE) PARTICLE	0	0	0	0	0	0	2	0	0	0	0	2
TOTAL		252	174	6	14	2	9	140	94	11	7	342	1051