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Communicable Diseases Intelligence

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Contents:

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VIRUSES, CHLAMYDIAS, COXIELLAS, RICKETTSIAS AND MYCOPLASMAS REPORTING SCHEME: A total of 1,562 reports were processed during this period.

Seventeen cases of Q fever (13 males, 1 female, 3 sex not stated) were reported during this period. Ages ranged from 17 to 47 years, including a 19-year-old jackaroo.

Eighty-five reports of influenza A (subtype not stated), 23 reports of influenza A(H3N2) and eighty reports of influenza B were received during this period.

Parainfluenza virus (typing pending) was isolated from the cerebrospinal fluid of a 13-month-old female who presented with fever (39°C), lethargy, anorexia and a small amount of vomiting. The fever continued for a further 5 days before resolving. A secondary respiratory tract infection responded to treatment with penicillin. No subsequent sequelae have been observed.

Eight serologically-confirmed cases of dengue fever were reported; all cases were imported. Of the 29 reports of arbovirus group A received during this period, 9 were identified as Barmah Forest virus.

Gancyclovir resistant cytomegalovirus was isolated from the urine of a 1-year-old female bone marrow transplant patient on maintenance gancyclovir. The patient, who was CMV-positive prior to BMT has developed bilateral CMV retinitis.

- The Bulletin is compiled and distributed by the Communicable Diseases Section, Communicable Diseases and Social Health Branch, Telephone: (062) 89 1555, Department of Community Services and Health.
- Contributions are solicited, and do not preclude later publication elsewhere.

- Figures given may be subject to revision.

OVERSEAS BRIEFS:

1. PNEUMOCOCCAL MENINGITIS - UNITED REPUBLIC OF TANZANIA

An outbreak of pneumococcal meningitis has been reported in the Ngorongoro District of the Arusha Region of Tanzania. Up to 21 August 1989, 422 cases had been notified.

2. MENINGOCOCCAL MENINGITIS - MOZAMBIQUE

Fifteen adult cases of meningococcal meningitis, serogroup C, have been reported in Maputo, Mozambique. There have been 4 deaths. Vaccination of the local population is being undertaken.

3. CHOLERA

Outbreaks of cholera have been reported in the following areas:

- Niger (Maradi Department): A total of 57 cases and 8 deaths have been notified between 1 August and 20 August 1989.
- Nepal (Katmandu Valley): A total of 95 cases of cholera were reported in August 1989. During 1-7 September 1989, 26 cases with 2 deaths were reported.
- Angola: The number of cases reported during an outbreak of cholera in Angola is declining.
- Yugoslavia (Dimitrovgrad): Two cases of cholera were identified during the investigation of an outbreak of enterocolitis. No further cholera cases have been reported.

The current cholera vaccine has only limited effectiveness and its use is only recommended where this is an entry requirement. Regardless of cholera vaccination status travellers to these countries are reminded to take care when selecting food and water.

NON-AGGLUTINABLE *VIBRIO CHOLERAE*: A BRIEF REPORT

A 41-year-old woman was admitted to Fairfield Hospital, Infectious Diseases Hospital on 29 August 1989 with a four-day history of diarrhoea. The patient developed cramping and diarrhoea following a 16 hour stopover in Bangkok prior to returning to Australia. While in Bangkok she had eaten scallops, locally prepared rice and carrots. *Vibrio cholerae*, which was non-agglutinating in O1 antiserum, was isolated from the patient's stool. The patient was discharged well from hospital on 1 September 1989.

Isolates of *Vibrio cholerae* that do not agglutinate in O-group-1 sera have been called 'noncholera' or 'non-agglutinable' vibrios. While some strains have been incriminated as the causal agent of acute cholera-like diarrhoea, they are most frequently nonpathogenic environmental contaminants [1].

REFERENCE

1. A.S. Benenson. Cholera. In: Evans AS, Fellman HA, eds. Bacterial Infections of Humans. Epidemiology and Control. New York: Plenum Medical Book Company, 1982:187-206.

NOTIFICATION OF LEPTOSPIROSIS

(Contributed by Professor S. Faine, Department of Microbiology, Monash University)

A summary of notifiable diseases, as reported to State and Territory health authorities, was published in the Communicable Diseases Intelligence in August 1989 [1]. The total number of cases notified for the whole of Australia was 104; 25 cases were notified in Victoria.

The Department of Microbiology, Monash University, carries out diagnostic tests on sera of some patients suspected of having leptospirosis. It is not the only laboratory in Victoria doing these tests. An analysis of our results for 1988 shows that there were 81 new patients diagnosed in this laboratory alone (Table 1). All sera were submitted by medical practitioners for assistance in the diagnosis of clinically suspect cases. The criteria for 'positive' are the widely accepted single titre of ≥400 in a microscopic agglutination test, in a patient with appropriate clinical features, or a rising titre to ≥400 [2]. Sixty-seven of the patients reacted with serovar hardjo, ten with pomona and four with tarassovi. Most patients were rural and the illness was apparently occupational. The data exclude duplicate and repeat tests, that is, they refer to individual patients and not numbers of tests.

Table 1: Leptospirosis cases (presumptive serological diagnosis), Department of Microbiology, Monash University, 1988

Serovar	J	F	M	A	M	J	J	A	S	O	N	D	Total
Pomona	1	2	-	2	1	-	-	1	1	1	-	1	10
Tarassovi	1	-	1	-	1	1	-	-	-	-	-	-	4
Hardjo	4	5	5	2	2	3	-	4	13	16	10	3	67
	6	7	6	4	4	4	0	5	14	17	10	4	81

It is clear that notifications fail to give a true indication of the real incidence of leptospirosis. If the Victorian figures can be extrapolated to the whole country, then a conservative total would be 81/25 x 104 = 337. Since most of these are occupational in selected risk groups, the incidence of serodiagnosed acute leptospirosis is high enough in these groups to cause concern about the occupational safety of workers in the industries concerned (dairy farming and the meat industry).

CDI Editorial Comment

Disease notification in Australia currently relies on voluntary reporting by medical practitioners and diagnostic laboratories to State/Territory health authorities which in turn notify to the Commonwealth Department of Community Services and Health. The current reporting rate is estimated at approximately 15% of the total incidence of diseases. To improve the situation, a National Disease Surveillance Network has been proposed and

will shortly be considered by the National Health and Medical Research Council and the Australian Health Ministers' Advisory Council.

REFERENCES

1. Faine S. Guidelines for the control of leptospirosis. World Health Organization Offset Publication No 67, Geneva 1982.

MEASLES OUTBREAK - CHICAGO USA, 1989

(Based on MMWR 1989;34:591-2)

As of 23 August 1989, 1123 confirmed cases of measles have been reported to the Chicago Department of Health. Information is available for 1019 (91%) of these cases; 799 (78%) have occurred in preschool-aged children (less than 5 years old), including 340 (33%) children <16 months of age (ie, too young for routine immunisation). Blacks and Hispanics have accounted for 955 (94%) of the cases. Four measles-associated fatalities have been reported.

Outbreak-control activities have included intensified surveillance and lowering of the recommended age for measles vaccination to 6 months during the outbreak, with revaccination at age 15 months for children vaccinated before the first birthday. Single-antigen mealses vaccine is being used for children before the first birthday, and measles-mumps-rubella vaccine (MMR) is administered to older children. Seven new vaccination clinics have been established and have administered approximately 21,000 doses of vaccine; door-to-door vaccination teams in high-risk communities have administered an additional 2000 doses of vaccine. Hospital emergency department vaccination clinics have been set up in four locations.

MMWR Editorial Note:

This outbreak is similar to others among inner-city populations in the United States in that it involves primarily unvaccinated black and Hispanic preschool-aged children [1-3]. The Chicago Department of Health has implemented agressive outbreak strategies directed toward reaching the highest-risk group ie, unvaccinated preschool-aged children. Such children are also likely to be a reservoir for transmitting virus to other age groups. As part of the extensive outbreak-control efforts, children are being vaccinated in emergency departments. Provision of vaccine to inner-city children who use these facilities for their primary source of health care should help to increase vaccination levels in patients who receive sporadic health care and may reduce the transmission of measles in emergency department settings.

REFERENCES

1. CDC. Measles - Dade County, Florida. MMWR 1987;36:45-8.
2. CDC. Measles - Los Angeles County, California, 1988. MMWR 1989;38:49-52,57.
3. Markowitz LE, Preblud SR, Orenstein WA, et al. Patterns of transmission in measles outbreaks in the United States, 1985-1986. N Engl J Med 1989;320:75-85.

MEASLES EPIDEMIC IN QUEBEC, CANADA

(Based on Can Dis Wkly Rep 1989;15[29])

Quebec is experiencing the most important measles epidemic to occur in that province in over 10 years. From the beginning of the year to 13 July 1989, approximately 9,800 cases of measles have been notified. In contrast, only 1,284 and 3,672 cases were reported in the 2 epidemic years 1984 and 1980, respectively. The epidemic is already responsible for 4 deaths (3 from pneumonia and 1 from myocarditis), and is also associated with the death of a fifth case. Moreover, 4 cases of encephalitis and 1 case of meningitis have also been observed.

The epidemic began on the west island of Montreal in late December 1988 and gradually spread throughout the province over the next 6 months. The municipalities bordering the greater metropolitan region of Montreal have been the areas most affected. A trend towards decreasing incidence has been observed in most regions of the province since mid-June.

A preliminary analysis carried our on epidemiological data available for 2,709 cases residing outside metropolitan Montreal and 1,182 cases from Montreal indicated that the age distribution of cases is similar in the 2 areas.

The results indicate that the age groups primarily affected by this epidemic have been those 10 to 19 years of age. About 33% to 50% of these cases had no prior history of measles immunisation. It therefore appears that the presence of a substantial pool of non-immune subjects, especially among those 10 to 19 years of age, has been an important factor in this epidemic.

CDWR Editorial Comment:

A review of measles and measles immunisation policy in Canada for both routine and outbreak situations will be made by the Canadian National Advisory Committee on Immunization as soon as data from the current outbreaks in Quebec and Ontario have been analysed. Until this review and analysis have been completed, the Committee has recommended that no change in routine measles immunisation in Canada be made at this time. The current recommendation is a single dose of measles/mumps/rubella vaccine (MMR) for all children on, or as soon as practicable after, their first birthday.

NON-A, NON-B HEPATITIS - ILLINOIS, USA

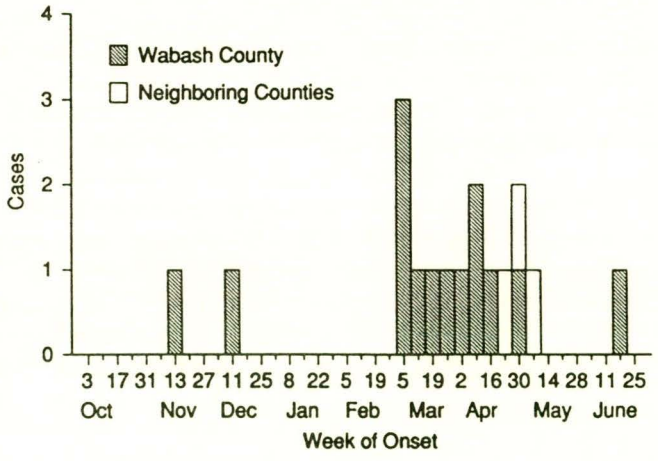
(Based on MMWR 1989;38:529-31)

From 15 November 1988 to 30 June 1989, 17 cases of non-A, non-B (NANB) hepatitis were reported to the Wabash County (Illinois) Health Department. In Wabash County, a small rural county in southern Illinois (estimated 1987 population: 13,800), only one other case of NANB hepatitis had been reported since 1982.

Of the 17 reported cases, 14 met a case definition for NANB hepatitis: an acute illness with symptoms and signs of hepatitis, elevated serum alanine aminotransferase (ALT) levels

>2.5 times the upper limit for normal, and negative serological markers for acute hepatitis A and B. Interviews with local physicians and review of the county hospital's medical records and emergency room log books detected no other cases among Wabash County residents since September 1988, but three cases were identified in neighbouring counties (Figure 1). Based on cases reported from January to June 1989, the annual rate of NANB hepatitis for Wabash County was 87.0 per 100,000 population, more than 100 times higher than the rate for all of Illinois during 1988 (0.7 per 100,000). Of the 17 cases in Wabash and neighbouring county residents: six (35%) were male; 16 (94%) were white, and one was American Indian; and the median age was 28 years (range: 14-36 years). Nine (53%) patients were clinically jaundiced, and nine (53%) required hospitalisation for their acute illness. Peak ALT values at onset of illness ranged from 201 to 3950 (median: 1493).

Figure 1: Non-A, non-B hepatitis cases, by week of onset - southern Illinois, October 1988 - June 1989



Patients were contacted to identify potential risk factors for acquiring NANB hepatitis. For 12 patients, information was obtained by interview, and for five, from medical chart review. Seven (41%) patients admitted to intravenous (IV)-drug use, and five (29%) were suspected IV-drug users. Of the seven who admitted IV-drug use, four used heroin and/or cocaine; one used heroin, cocaine, and methamphetamine; one used only methamphetamine; for one, the drug was unknown. Three of the 12 patients reported drinking >55 ounces of alcohol per week. None of the patients reported blood transfusion within 6 months before onset of illness; none reported employment in a health-care setting with frequent blood exposure; and none reported sexual contact within 6 months before onset of illness with a person known to have had NANB hepatitis.

Blood specimens were obtained in late May and in June from 12 of the patients and 28 of their household, sexual, and needle-sharing contacts. All contacts denied symptoms of hepatitis. However, four had abnormal ALT values: three with histories of IV-drug use (elevations of 57-91 units/litre [upper limits of normal range from 36 to 53]) and a 6-year-old boy (ALT of 430) whose mother was a case-patient. All contacts were negative for IgM antibody to hepatitis B core antigen; of

those with elevated ALT values, all were negative for IgM antibody to hepatitis A virus. Serological testing of patients and contacts using a new assay for a parenterally transmitted NANB hepatitis virus is pending [1]. Efforts will be made to obtain follow-up specimens to determine the extent of transmission to household and sexual contacts.

IV-drug use has existed in the county for many years; most drug users are thought to reside within the community and to have limited interaction with drug users from other areas. However, the apparent index patient was an IV-drug user who had lived intermittently in other states; he had recently returned to the area and became ill 1 week after arrival in November. Before his illness, he shared needles with another person who became ill 4 weeks later. Among the cases in March and April, two distinct clusters occurred that involved persons who were both friends and known or suspected IV-drug users. During the New Year holiday, some of these persons attended parties at which IV drugs were reportedly used. One IV-drug user reported that, because the area's needle supply had been scarce during the past year, needle-sharing had increased.

MMWR Editorial Note:

Parenterally transmitted NANB hepatitis accounts for 20%-40% of acute viral hepatitis in the United States. Although it has traditionally been considered a transfusion-associated disease, studies of community-acquired NANB hepatitis and data from the CDC national surveillance system have shown that 23%-42% of NANB hepatitis cases are associated with IV-drug use [2,3]; in addition, 8%-11% are attributed to blood transfusion and 4%-8% to health-care occupational exposure. However, for as many as 57%, no source of infection can be identified [3]. In this outbreak, the high proportion of ill persons who were confirmed or suspected IV-drug users and the lack of an identifiable common hepatoxic chemical or drug suggest that the etiological agent is parenterally transmitted NANB hepatitis virus.

Community-based outbreaks of parenterally transmitted NANB hepatitis have not been reported previously in the United States. Large outbreaks of NANB hepatitis occur in developing countries [4]; however, in these settings, the disease is transmitted enterically and is caused by an agent distinct from that causing parenterally transmitted NANB hepatitis [5]. This enterically transmitted form of disease is not believed to occur in the United States except for occasional imported cases [6].

The role of person-to-person contact in the transmission of NANB hepatitis in the United States has not been well defined. Transmission between spouses has been observed [7]. In addition, a recent case-control study of patients with acute NANB hepatitis showed that contact with multiple heterosexual partners and household or sexual contact with a person who had had hepatitis were associated with risk for disease [8].

A portion of the genome of a virus that is probably a major cause of parenterally transmitted NANB hepatitis was recently cloned and a candidate serological assay was developed [1,9]. The assay should assist with studies of the mechanisms and extent of transmission of NANB hepatitis outside the transfusion setting, such as transmission by household and

sexual contact. Previous studies of household and sexual transmission of NANB hepatitis using ALT testing have been limited by the lack of specificity of ALT values and the possibility of asymptomatic, biochemically silent transmission.

IV-drug use traditionally has been considered a problem of urban areas. The recognition of a high prevalence of drug use and an associated epidemic of a bloodborne disease in this rural community and the increased recognition of outbreaks of hepatitis A and B among drug users in rural settings [10-12] emphasise that IV drug use is not limited to urban areas. This recognition also underscores the need for prevention and treatment programs in many geographic areas.

REFERENCES

1. Kuo G, Choo Q-L, Alter HJ, et al. An assay for circulating antibodies to a major etiologic virus of human non-A, non-B hepatitis. Science 1989;244:362-4.
2. Alter MJ, Hadler SC, Francis DP, Maynard JE. The epidemiology of non-A, non-B hepatitis in the United States. In: Dodd RY, Barker LF, eds. Infection, immunity, and blood transfusion. New York: Alan R. Liss, 1985:71-9.
3. CDC. Hepatitis surveillance report no. 52. Atlanta: US Department of Health and Human Services, Public Health Service, 1989:25-7.
4. Ramalingaswami V, Purcell RH. Waterborne non-A, non-B hepatitis. Lancet 1988;1:571-3.
5. Krawczynski K, Bradley DW. Enterically transmitted non-A, non-B hepatitis: identification of virus-associated antigen in experimentally infected cynomolgus macaques. J Infect Dis 1989;159:1042-9.
6. De Cock KM, Bradley DW, Sandford NL, et al. Epidemic non-A, non-B hepatitis in patients from Pakistan. Ann Intern Med 1987;106:227-30.
7. Guyer B, Bradley DW, Bryan JA, Maynard JE. Non-A, non-B hepatitis among participants in a plasmapheresis stimulation program. J Infect Dis 1979;139:634-40.
8. Alter MJ, Coleman PJ, Alexander WJ, et al. Importance of heterosexual activity in the transmission of hepatitis B and non-A, non-B hepatitis. Jama 1989;262:1201-5.
9. Choo Q-L, Kuo G, Weiner AJ, Overby LR, Bradley DW, Houghton M. Isolation of a cDNA clone derived from a blood-borne non-A, non-B viral hepatitis genome. Science 1989;244:359-62.
10. CDC. Hepatitis B - New Bern, North Carolina. MMWR 1979;28:373-4.
11. CDC. Fulminant hepatitis B among parenteral drug abusers - Kentucky, California. MMWR 1984;33:70,76-7.
12. Harkess J, Gildon B, Istre GR. Outbreaks of hepatitis A among illicit drug users, Oklahoma, 1984-87. Am J Public Health 1989;79:463-6.

MELIOIDOSIS IN A TRAVELLER

(Based on Can Dis Wkly Rep 1989;15:109-10)

In July 1988, a 40-year-old male resident of Quebec who had worked as a cooperant in Bangkok, Thailand for 6 months returned to Canada. Three weeks prior to his return home, he began to experience fever and chills, and had a cough producing a coloured sputum. Following a cursory examination, a Thai physician had diagnosed probable malaria, even though the smear was negative, and prescribed therapeutic doses of chloroquine.

Immediately upon returning to Canada, the patient sought medical advice at a hospital. There was nothing unusual in his medical history and he had never had any tropical diseases. His work requires him to travel abroad regularly, and for the past 6 months he had been mainly in Bangkok, although he had spent some time in the outlying areas, particularly the north, and sometimes in the bush. He had also visited Hong Kong briefly.

Upon questioning, the patient said that he felt generally unwell; the fever persisted, but he was no longer experiencing

chills. He had lost 5kg. His cough was slightly productive but there was no significant dyspnoea. On examination, his temperature was 38.7°C; all other vital signs were normal. Pulmonary auscultation revealed discrete abnormalities in the right upper chest; the rest of the physical examination was normal. There was a slight leukocytosis (11.2 X 10^9/L with a normal differential). Sedimentation rate was elevated at 107 mm/h. Pulmonary X-ray showed a right upper lobe cavity surrounded by an alveolar infiltrate. Tomography sections confirmed the presence of a cavity 4 cm in diameter.

The patient was admitted to hospital with a diagnosis of pulmonary abscess, probably tuberculous. Bronchoscopic samples yielded no *Mycobacterium tuberculosis*; however, there were many gram-negative rods which had a bipolar appearance when stained.

On the first day of hospitalisation, cefoxitin was administered. The following day this was replaced by clindamycin and tobramycin. Because the fever still persisted on the third day, it was decided to add ceftazidime, 2 g IV every 6 h, to the antibiotic regimen. On the fifth day of hospitalisation, the laboratory confirmed the presence of *Pseudomonas pseudomallei* in the sputum. The clindamycin and tobramycin were replaced by trimethoprim/sulfamethoxazole (TMP/SMX) (TMP 250 mg or 4 mg/kg IV every 6 h). By the next day, the patient's temperature had dropped and the cough had decreased. After 10 days of treatment, the patient was totally asymptomatic and a follow-up X-ray showed virtually complete resolution of the lesion on the right upper lobe. The patient received IV treatment for 1 month. It was decided to consolidate this treatment with 2 months of oral antibiotic therapy with TMP/SMX (160 mg TMP every 12 h). At the September 1988 follow-up visit the patient was still well.

CDWR Editorial Comment:

Samples were taken for serolgy at the beginning of hospitalisation and sent to LCDC in Ottawa, and to CDC, Atlanta. They were found positive for *P. pseudomallei*, with a titre of 1:2048 by microhaemagglutination.

There have been 3 cases of melioidosis reported in the Canadian literature [1,2,3], all involving refugees from Southeast Asia. In this area, subclinical infection is common and serological studies have shown significant antibody titres in up to 20% of asymptomatic individuals. The organism can remain latent for months to many years. Concomitent diseases such as diabetes, burns and alcoholism have frequently been associated with its reactivation. Consequently, melioidosis may occur or recur years after an individual has left the endemic area.

The case presented here appears to be the first person of Canadian origin in whom melioidosis, apparently acute, has been described. The Quebec Public Health Laboratory reports that this is the first time such a case has occurred in this province.

The majority of cases of melioidosis occur in Southeast Asia or in persons who have stayed in that area. Sporadic cases have also been reported in India, Korea, the Philippines, Australia, Panama, Ecuador and Turkey.

P. pseudomallei is a waterborne bacterium transmitted to man directly from the environment. The asymptomatic form of the infection occurs the most frequently, followed by lung involvement which may be acute or chronic. It characteristically involves the upper lobes, sometimes with cavitation. Skin infection occurs most often after a wound becomes infected. In its severe form, the disease may involve septicemia and metastatic dissemination. In addition, chronic suppurative forms have been described in several organs.

In a patient returning from a stay in Southeast Asia, the infectious agents to be considered in a differential diagnosis of pulmonary involvement include tuberculosis, melioidosis and trematodes of the genus *Paragonimus*.

Treatment of melioidosis is often problematic because the bacterium is resistant to many agents. Ceftazidime has been used successfully against multiresistant strains [4] and laboratory sensitivity tests have recently confirmed its efficacy [5].

CDI Editorial Comment:

Symptomatic *P. pseudomallei* is usually associated with altered intercurrent illness, injury or stress. The patient had spent a large part of the last 6 months in Southeast Asia and may have been taking antimalarial prophylaxis, which in certain persons can change the haematological profile, mimicking an immunocompromised state.

REFERENCES

1. Gregor WM, Jones JR. Melioidosis in a Southeast Asian refugee - Ontario. CDWR 1981;7:253-4.
2. MacDonald NE, Huang P-P, Galey JP, Saginur R. Melioidosis of the distal femur - Ontario. CDWR 1984;18:33-4.
3. Chan CK, Hyland RH, Leers WD, et al. Pleuropulmonary melioidosis in a Cambodian refugee. Can Med Assoc J 1984;131:1365-7.
4. So SY, Chan PY, Leung YK, et al. Successful treatment of melioidosis caused by a multiresistant strain in an immunocompromised host with third generation cephalosporins. Am Rev Respir Dis 1983;127:650-4.
5. McEniry DW, Gillespie SH, Felminghan D. Susceptibility of Pseudomonas pseudomallei to new B-Lactam and aminoglycoside antibiotics. J Antimicrob Chemother 1988;21:171-5.

AUSTRALIA - COMMUNICABLE DISEASES INTELLIGENCE

VIRAL IDENTIFICATIONS FROM CONTRIBUTING LABORATORIES
BASED ON DATE OF REPORTING

PERIOD 14/9/89 TO 27/9/89

1. CODE 019 - FAIRFIELD(VIC)
2. CODE 065 - STATE LAB(WA) PMH(WA)
3. CODE 110 - IMVS(SA)
4. CODE 111 - RCH(VIC)
5. CODE 112 - ICPMR(NSW) WVH(ACT)
6. CODE 113 - PHH POW(NSW)
7. CODE 114 - RAHC(NSW)
8. CODE 115 - STATE LAB(QLD)

			019	065	110	111	112	113	114	115	TOTAL
		1	0	0	0	0	0	0	0	0	1
0100	ADENOVIRUS NOT TYPED	0	0	3	2	0	2	5	0	18	30
0101	ADENOVIRUS TYPE 1	0	1	0	0	0	3	0	0	0	4
0102	ADENOVIRUS TYPE 2	0	4	0	0	0	1	0	0	0	5
0103	ADENOVIRUS TYPE 3	0	2	0	3	0	0	0	1	0	6
0104	ADENOVIRUS TYPE 4	0	4	0	0	0	0	0	0	0	4
0105	ADENOVIRUS TYPE 5	0	1	0	0	0	1	0	0	0	2
0106	ADENOVIRUS TYPE 6	0	0	0	2	0	0	0	0	0	2
0108	ADENOVIRUS TYPE 8	0	0	0	2	0	0	0	0	0	2
0109	ADENOVIRUS TYPE 9	0	0	0	0	0	1	0	0	0	1
0112	ADENOVIRUS TYPE 12	0	0	0	0	0	1	0	0	0	1
0121	ADENOVIRUS TYPE 21	0	0	1	0	0	0	0	0	0	1
0130	ADENOVIRUS TYPE 30	0	0	0	0	0	2	0	0	0	2
0199	ADENOVIRUS TYPING PENDING	0	0	0	0	7	0	0	2	0	9
0201	INFLUENZA A VIRUS	0	9	14	23	22	9	4	2	2	85
0202	INFLUENZA A VIRUS SUBTYPE H3N2	0	6	0	2	4	2	0	0	9	23
0203	INFLUENZA B VIRUS	0	14	3	14	11	5	7	3	23	80
0299	INFLUENZA VIRUS - TYPING PENDI	0	0	0	0	1	0	0	0	0	1
0301	PARAINFLUENZA VIRUS TYPE 1	0	1	0	0	0	1	0	0	0	2
0302	PARAINFLUENZA VIRUS TYPE 2	0	0	0	0	0	2	1	0	1	4
0303	PARAINFLUENZA VIRUS TYPE 3	0	2	3	21	0	1	0	0	14	41
0399	PARAINFLUENZA VIRUS TYPING PEN	0	0	0	0	0	0	0	0	22	22
0400	RESPIRATORY SYNCYTIAL VIRUS (R	0	11	2	16	16	6	7	13	10	81
0500	RHINOVIRUS (ALL TYPES)	0	6	2	14	1	2	0	0	0	25
0600	MYCOPLASMA PNEUMONIAE	0	6	3	0	7	10	1	0	4	31
0700	ORNITHOSIS-PSITTACOSIS	0	7	0	1	0	0	1	0	0	9
0823	COXSACKIEVIRUS A23 = ECHOVIRUS	0	0	0	0	0	1	0	0	0	1
0903	COXSACKIEVIRUS B3	0	0	0	0	0	1	0	0	0	1
1003	ECHOVIRUS TYPE 3	0	0	1	0	0	0	0	0	0	1
1006	ECHOVIRUS TYPE 6	0	1	0	0	0	0	0	0	0	1
1009	ECHOVIRUS TYPE 9	0	0	0	0	0	1	0	0	0	1
1023	ECHOVIRUS TYPE 23	0	0	0	0	0	1	0	1	0	2
1025	ECHOVIRUS TYPE 25	0	0	0	0	0	1	0	0	0	1
1028	ECHOVIRUS TYPE 28 = RHINO VIRU	0	0	0	0	0	0	0	1	0	1
1030	ECHOVIRUS TYPE 30	0	0	2	0	0	1	0	0	0	3
1100	POLIOVIRUS NOT TYPED	0	0	0	0	0	0	6	0	0	6
1101	POLIOVIRUS TYPE 1	0	1	0	2	0	1	0	0	0	4
1102	POLIOVIRUS TYPE 2	0	1	0	1	0	0	0	0	0	2
1103	POLIOVIRUS TYPE 3	0	2	0	0	0	2	0	0	0	4
1104	POLIOVIRUS - MIXED VACCINAL ST	0	2	1	0	0	0	0	0	0	3
1200	MUMPS VIRUS	0	1	0	0	0	7	1	0	0	9
1300	HERPES VIRUS GROUP - NOT TYPED	0	0	1	0	0	4	0	0	0	5
1301	HERPES SIMPLEX VIRUS - NOT TYP	0	0	3	1	0	56	0	0	58	118
1302	EPSTEIN-BARR VIRUS (EB VIRUS)	0	3	0	8	2	3	0	4	22	42
1303	VARICELLA-ZOSTER VIRUS	0	2	3	2	0	8	3	1	1	20
1306	HERPES SIMPLEX TYPE 1	0	37	14	18	0	6	4	0	0	79
1307	HERPES SIMPLEX TYPE 2	0	52	46	16	0	22	24	0	1	161
1399	HERPES VIRUS TYPING PENDING	0	0	0	0	1	0	0	0	0	1
1401	COXIELLA BURNETII	0	0	0	0	0	6	0	0	11	17
1502	PICORNIA VIRUS - NOT TYPED = E	0	0	0	0	0	0	5	1	7	13
1521	MEASLES VIRUS	0	3	0	0	0	1	0	0	0	4
1522	RUBELLA VIRUS	0	14	0	6	0	0	0	0	16	36
1532	HEPATITIS B ANTIGEN	0	14	26	5	1	37	10	1	46	140
1535	HEPATITIS A ANTIBODY	0	0	2	2	0	0	1	0	0	5
1541	CHLAMYDIA A - C. TRACHOMATIS	0	0	28	16	0	33	0	0	2	79
1556	CMV - CYTOMEGALOVIRUS	0	39	2	4	2	4	9	1	28	89
1563	CORONAVIRUS	0	0	0	0	0	2	0	0	0	2
1564	ROTAVIRUS	0	4	12	58	0	25	40	7	0	146
1565	CALICI VIRUS	0	0	0	0	0	2	0	0	0	2
1599	ENTEROVIRUS TYPING PENDING	0	0	0	0	5	0	14	0	0	19
9901	ARBOVIRUS GROUP A.(UNSPECIFIED	0	0	0	0	0	0	0	0	29	29
9992	ROSS RIVER VIRUS	0	2	3	0	0	0	0	0	16	21
9994	SMALL VIRUS (LIKE) PARTICLE	0	1	2	0	0	1	0	2	0	6
9995	DENGUE	0	0	0	0	0	0	0	0	8	8
9997	KUNJIN VIRUS	0	0	0	0	0	0	0	0	1	1
9998	ARBOVIRUS GROUP B.(UNSPECIFIED	0	0	0	0	0	0	0	0	5	5
TOTAL		1	253	177	239	80	275	143	40	354	1562

AUSTRALIA - COMMUNICABLE DISEASES INTELLIGENCE

VIRAL IDENTIFICATIONS BY CLINICAL INFORMATION TABLE 1

PERIOD 14/9/89 TO 27/9/89

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	6	7	8	9	10	11	TOTAL
0100	ADENOVIRUS NOT TYPED	1	19	0	0	0	8	0	0	0	0	28
0101	ADENOVIRUS TYPE 1	0	2	0	0	0	2	0	0	0	0	4
0102	ADENOVIRUS TYPE 2	0	4	0	0	0	1	0	0	0	0	5
0103	ADENOVIRUS TYPE 3	0	3	0	0	0	0	0	0	0	0	3
0104	ADENOVIRUS TYPE 4	0	1	0	0	0	0	0	0	0	0	1
0105	ADENOVIRUS TYPE 5	0	1	0	0	0	1	0	0	0	0	2
0106	ADENOVIRUS TYPE 6	0	2	0	0	0	0	0	0	0	0	2
0109	ADENOVIRUS TYPE 9	1	0	0	0	0	0	0	0	0	0	1
0112	ADENOVIRUS TYPE 12	0	0	0	0	0	1	0	0	0	0	1
0121	ADENOVIRUS TYPE 21	1	0	0	0	0	0	0	0	0	0	1
0130	ADENOVIRUS TYPE 30	1	0	0	0	0	0	0	0	0	0	1
0199	ADENOVIRUS TYPING PENDING	0	6	0	0	0	0	0	0	0	0	6
0201	INFLUENZA A VIRUS	5	68	1	0	1	1	0	1	0	0	77
0202	INFLUENZA A VIRUS SUBTYPE H3N2	0	20	0	0	1	0	0	0	0	0	21
0203	INFLUENZA B VIRUS	3	66	0	0	0	0	0	0	0	0	69
0299	INFLUENZA VIRUS - TYPING PENDI	0	1	0	0	0	0	0	0	0	0	1
0301	PARAINFLUENZA VIRUS TYPE 1	1	1	0	0	0	0	0	0	0	0	2
0302	PARAINFLUENZA VIRUS TYPE 2	1	2	0	0	0	0	0	0	0	1	4
0303	PARAINFLUENZA VIRUS TYPE 3	1	40	0	0	0	0	0	0	0	0	41
0399	PARAINFLUENZA VIRUS TYPING PEN	0	20	0	0	1	0	0	0	0	1	22
0400	RESPIRATORY SYNCYTIAL VIRUS (R	2	74	0	0	0	1	0	0	1	0	78
0500	RHINOVIRUS (ALL TYPES)	0	24	0	0	0	0	0	0	0	1	25
0600	MYCOPLASMA PNEUMONIAE	7	18	1	0	0	0	0	0	0	0	26
0700	ORNITHOSIS-PSITTACOSIS	2	4	1	0	0	0	0	0	0	0	7
0823	COXSACKIEVIRUS A23 = ECHOVIRUS	0	1	0	0	0	0	0	0	0	0	1
0903	COXSACKIEVIRUS B3	0	1	0	0	0	0	0	0	0	0	1
1006	ECHOVIRUS TYPE 6	0	1	0	0	0	0	0	0	0	0	1
1009	ECHOVIRUS TYPE 9	0	0	0	1	0	0	0	0	0	0	1
1025	ECHOVIRUS TYPE 25	0	0	0	0	0	1	0	0	0	0	1
1028	ECHOVIRUS TYPE 28 = RHINO VIRU	0	1	0	0	0	0	0	0	0	0	1
1030	ECHOVIRUS TYPE 30	0	0	0	3	0	0	0	0	0	0	3
1100	POLIOVIRUS NOT TYPED	0	0	0	0	0	6	0	0	0	0	6
1101	POLIOVIRUS TYPE 1	1	2	0	0	0	0	0	0	0	0	3
1102	POLIOVIRUS TYPE 2	0	2	0	0	0	0	0	0	0	0	2
1103	POLIOVIRUS TYPE 3	1	2	0	0	0	1	0	0	0	0	4
1104	POLIOVIRUS - MIXED VACCINAL ST	0	3	0	0	0	0	0	0	0	0	3
1200	MUMPS VIRUS	4	1	0	0	0	0	0	0	0	0	5
1300	HERPES VIRUS GROUP - NOT TYPED	0	0	0	0	0	0	0	0	0	5	5
1301	HERPES SIMPLEX VIRUS - NOT TYP	16	1	1	0	1	0	0	0	0	53	72
1302	EPSTEIN-BARR VIRUS (EB VIRUS)	2	8	1	1	0	1	0	1	0	1	15
1303	VARICELLA-ZOSTER VIRUS	6	1	1	0	0	0	0	0	0	10	18
1306	HERPES SIMPLEX TYPE 1	6	6	0	0	0	0	0	0	1	38	51
1307	HERPES SIMPLEX TYPE 2	11	0	0	0	0	0	0	0	1	47	59
1399	HERPES VIRUS TYPING PENDING	0	0	0	0	0	0	0	0	0	1	1
1401	COXIELLA BURNETII	3	1	0	0	0	2	0	0	0	0	6
1502	PICORNIA VIRUS - NOT TYPED = E	0	3	0	0	2	8	0	0	0	0	13
1521	MEASLES VIRUS	2	0	0	0	0	0	0	0	0	2	4
1522	RUBELLA VIRUS	4	1	0	0	0	0	0	0	0	8	13
1532	HEPATITIS B ANTIGEN	53	0	0	0	0	0	82	0	0	0	135
1535	HEPATITIS A ANTIBODY	0	0	0	0	0	0	5	0	0	0	5
1541	CHLAMYDIA A - C. TRACHOMATIS	17	0	0	0	0	0	1	0	0	0	18
1556	CMV - CYTOMEGALOVIRUS	7	22	0	0	1	0	1	2	6	0	39
1563	CORONAVIRUS	1	1	0	0	0	0	0	0	0	0	2
1564	ROTAVIRUS	7	0	0	0	0	137	0	0	0	0	144
1565	CALICI VIRUS	1	0	0	0	0	1	0	0	0	0	2
1599	ENTEROVIRUS TYPING PENDING	0	2	0	0	0	11	0	0	0	1	14
9901	ARBOVIRUS GROUP A.(UNSPECIFIED	5	2	0	0	0	2	0	0	0	1	10
9992	ROSS RIVER VIRUS	2	0	0	0	0	0	0	0	0	5	7
9994	SMALL VIRUS (LIKE) PARTICLE	0	0	0	0	0	5	0	0	0	0	5
9995	DENGUE	3	1	0	0	0	0	0	0	0	0	4
9998	ARBOVIRUS GROUP B.(UNSPECIFIED	0	0	1	0	0	0	0	0	0	0	1
TOTAL		178	439	7	5	7	190	89	4	9	175	1103

AUSTRALIA - COMMUNICABLE DISEASES INTELLIGENCE

VIRAL IDENTIFICATIONS BY CLINICAL INFORMATION TABLE 2

PERIOD 14/9/89 TO 27/9/89

12. CODE 10 - EYE
13. CODE 59 - GENITAL
14. CODE 39 - ENDOCRINE/SALIVARY GL.
15. CODE 38 - RETICULO-ENDOTHELIAL
16. CODE 29 - MUSCLE/JOINT
17. CODE 69 - CONGENTIAL
18. CODE P8 - PUO
19. CODE G8 - FEVER/MALAISE
20. CODE 09 - OTHER
21. CODE A1 - SIDS

		12	13	14	15	16	17	18	19	20	21	TOTAL
0100	ADENOVIRUS NOT TYPED	1	0	0	0	0	0	0	1	0	0	2
0103	ADENOVIRUS TYPE 3	0	1	0	0	0	0	0	1	1	0	3
0104	ADENOVIRUS TYPE 4	3	0	0	0	0	0	0	0	0	0	3
0108	ADENOVIRUS TYPE 8	2	0	0	0	0	0	0	0	0	0	2
0130	ADENOVIRUS TYPE 30	0	0	0	0	0	0	0	0	1	0	1
0199	ADENOVIRUS TYPING PENDING	0	0	0	0	0	0	0	3	0	0	3
0201	INFLUENZA A VIRUS	0	0	0	0	2	0	0	4	2	0	8
0202	INFLUENZA A VIRUS SUBTYPE H3N2	0	0	0	0	0	0	0	2	0	0	2
0203	INFLUENZA B VIRUS	0	0	0	0	0	0	1	9	1	0	11
0400	RESPIRATORY SYNCYTIAL VIRUS (R	0	0	0	0	0	0	0	2	1	0	3
0600	MYCOPLASMA PNEUMONIAE	0	0	0	0	1	0	0	1	3	0	5
0700	ORNITHOSIS-PSITTACOSIS	0	0	0	0	0	0	1	1	0	0	2
1003	ECHOVIRUS TYPE 3	0	0	0	0	0	0	0	1	0	0	1
1023	ECHOVIRUS TYPE 23	0	0	0	0	0	2	0	0	0	0	2
1101	POLIOVIRUS TYPE 1	0	0	0	0	0	0	0	1	0	0	1
1200	MUMPS VIRUS	0	0	1	0	1	0	0	2	0	0	4
1301	HERPES SIMPLEX VIRUS - NOT TYP	0	45	0	0	0	0	0	0	1	0	46
1302	EPSTEIN-BARR VIRUS (EB VIRUS)	0	0	8	11	1	0	0	5	2	0	27
1303	VARICELLA-ZOSTER VIRUS	0	1	0	0	0	0	0	0	1	0	2
1306	HERPES SIMPLEX TYPE 1	4	18	0	0	0	0	1	1	4	0	28
1307	HERPES SIMPLEX TYPE 2	0	99	0	0	0	0	0	0	3	0	102
1401	COXIELLA BURNETII	0	0	0	0	2	0	1	7	1	0	11
1522	RUBELLA VIRUS	0	0	2	0	3	1	1	1	15	0	23
1532	HEPATITIS B ANTIGEN	0	0	0	0	0	0	0	0	5	0	5
1541	CHLAMYDIA A - C. TRACHOMATIS	1	59	0	0	0	0	0	1	0	0	61
1556	CMV - CYTOMEGALOVIRUS	3	6	0	1	0	4	1	10	25	0	50
1564	ROTAVIRUS	0	0	0	0	0	0	1	0	1	0	2
1599	ENTEROVIRUS TYPING PENDING	0	0	0	0	1	0	0	0	1	3	5
9901	ARBOVIRUS GROUP A.(UNSPECIFIED	0	0	0	0	15	0	0	0	4	0	19
9992	ROSS RIVER VIRUS	0	0	0	0	4	0	0	4	6	0	14
9994	SMALL VIRUS (LIKE) PARTICLE	0	0	0	0	0	0	0	0	1	0	1
9995	DENGUE	0	0	0	0	1	0	0	2	1	0	4
9997	KUNJIN VIRUS	0	0	0	0	1	0	0	0	0	0	1
9998	ARBOVIRUS GROUP B.(UNSPECIFIED	0	0	0	0	3	0	0	1	0	0	4
TOTAL		14	229	11	12	35	7	7	60	80	3	458